

**United States Court of Appeals for the Second Circuit
Thurgood Marshall U.S. Courthouse
40 Foley Square
New York, NY 10007**

ROBERT A. KATZMANN
CHIEF JUDGE

Date: August 18, 2020
Docket #: 20-2729
Short Title: State of New York v. Wheeler

CATHERINE O'HAGAN WOLFE
CLERK OF COURT

Agency #: EPA-HQ-OPPT-2019-
0437; FRL-10011-16
Agency: Environmental
Protection Agency

SERVICE FOR PETITION FOR REVIEW

Pursuant to FRAP 15(c) you are hereby served with the enclosed petition for review of the United States Environmental Protection Agency order in the above-entitled case that was filed in this court on August 17, 2020.

A copy of the petition is enclosed herewith.

Inquiries regarding this case may be directed to 212-857-8529.

**IN THE UNITED STATES COURT OF APPEALS
FOR THE SECOND CIRCUIT**

State of New York, District of Columbia,
State of Hawai'i, State of Illinois, State of
Maine, State of Maryland, Commonwealth
of Massachusetts, State of Minnesota, State
of New Jersey, State of Oregon, State of
Rhode Island, State of Vermont, and the
City of New York,

Petitioners,

v.

Andrew Wheeler, as Administrator of the
Environmental Protection Agency, and the
Environmental Protection Agency,

Respondents.

Case No. 20-_____

PETITION FOR REVIEW

Pursuant to Federal Rule of Appellate Procedure 15, sections 6(i)(1) and 19(a) of the Toxic Substances Control Act, 15 U.S.C. §§ 2605(i)(1), 2618(a), and section 10 of the Administrative Procedure Act, 5 U.S.C. §§ 701-706, the State of New York, District of Columbia, State of Hawai'i, State of Illinois, State of Maine, State of Maryland, Commonwealth of Massachusetts, State of Minnesota, State of New Jersey, State of Oregon, State of Rhode Island, State of Vermont, and the City of New York (collectively, the "State and Municipal Petitioners"), petition this Court to review the Environmental Protection Agency's ("EPA") final agency

action whereby EPA issued an order determining that methylene chloride “does not present an unreasonable risk of injury to health or the environment,” *see Risk Evaluation for Methylene Chloride (Dichloromethane, DCM)*, Subsection 5.4.1; *Methylene Chloride (MC); Final Toxic Substances Control Act (TSCA) Risk Evaluation; Notice of Availability*, 85 Fed. Reg. 37,942 (June 24, 2020).

A copy of the *Risk Evaluation for Methylene Chloride (Dichloromethane, DCM)* is attached hereto as Attachment A.¹ A copy of the *Methylene Chloride (MC); Final Toxic Substances Control Act (TSCA) Risk Evaluation; Notice of Availability* is attached hereto as Attachment B.²

State and Municipal Petitioners seek a determination by this Court pursuant to section 19(c) of the Toxic Substances Control Act, 15 U.S.C. § 2618(c), that the order is unlawful and therefore must be set aside.

¹ See also <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0107>.

² See also <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0437-0081>.

August 17, 2020

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ATTACHMENT A

Risk Evaluation for Methylene Chloride (Dichloromethane, DCM)

CASRN: 75-09-2

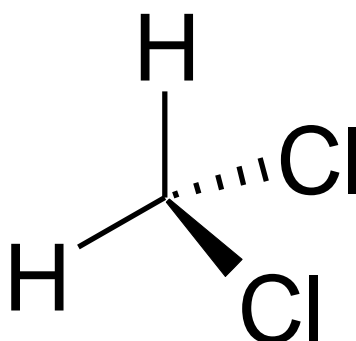


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Docket

Supporting information can be found in public docket: [EPA-HQ-OPPT-2016-0742](#).

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ABBREVIATIONS

°C	Degrees Celsius
ACGIH	American Conference of Government Industrial Hygienists
ACh	Acetylcholine
ACR	Acute-to-chronic Ratio
ADC	Average Daily Concentration
ADR	Acute Dose Rate
AEGL	Acute Exposure Guideline Level
AF	Assessment Factor
AhR	Aryl Hydrocarbon Receptor
AIC	Akaike information criterion
ALT	Alanine Transaminase
ANOVA	Analysis of Variance
APF	Assigned Protection Factor
ASD	Autism Spectrum Disorder
AST	Aspartate Amino Transferase
atm	Atmosphere(s)
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BCFBAF	EPI Suite™ model that estimates Bioconcentration and Bioaccumulation Factors
BIOWIN	EPI Suite™ model that estimates Biodegradation rates
BMD	Benchmark Dose
BMDL	Benchmark Dose Lower Confidence Limit
BMR	Benchmark Response
BMDS	Benchmark Dose Software
CAA	Clean Air Act
CADD	Chronic Average Daily Dose
CAR	Constitutive Androstane Receptor
CASRN	Chemical Abstracts Service Registry Number
CARB	California Air Resources Board
CBI	Confidential Business Information
CDR	Chemical Data Reporting
CEM	Consumer Exposure Model
CEPA	Canadian Environmental Protection Act
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
CFF	Critical Flicker Function
CFR	Code of Federal Regulations
CHIRP	Chemical Risk Information Platform
ChV	Chronic Value
CI	Confidence Interval
cm ³	Cubic Centimeter(s)
CNS	Central Nervous System
COC	Concentration of Concern
CoCAP	Cooperative Chemicals Assessment Program
COHb	Carboxyhemoglobin
COU	Conditions of Use
CPDat	Chemical and Products Database

CPSC	Consumer Product Safety Commission
CSCL	Chemical Substances Control Law
CWA	Clean Water Act
CYP450	Cytochrome P450
DCM	Dichloromethane (Methylene Chloride)
DF	Dilution Factor
DFq	Detection frequency
DMR	Discharge Monitoring Report
DNA	Deoxyribonucleic Acid
DoD	Department of Defense
EC ₅₀	Effect concentration at which 50% of test organisms exhibit an effect
ECHA	European Chemicals Agency
ECHO	Enforcement and Compliance History Online
ECOTOX	ECOTOXicology Knowledgebase System
EEG	Electroencephalogram
EF	Exposure Frequency
E-FAST	Exposure and Fate Assessment Screening Tool
ELCR	Excess Lifetime Cancer Risk
EPA	Environmental Protection Agency
EPCRA	Emergency Planning and Community Right-to-Know Act
EPI Suite™	Estimation Programs Interface suite of models
ER	Extra Risk
EU	European Union
EVOH	Ethylene Vinyl Alcohol
FACE	Fatality Assessment and Control Evaluation
FDA	Food and Drug Administration
FFDCA	Federal Food, Drug, and Cosmetic Act
FR	Federal Register
FRS ID	Facility Registry Service Identification
g	Gram(s)
GABA	Gamma-aminobutyric Acid
GC	Gas Chromatography
GD(s)	Gestational Day
GM	Geometric Mean
GSD	Geometric Standard Deviation
GSH	Glutathione
GST	Glutathione S-transferase
GSTT1	Theta 1 Isozyme
HAP	Hazardous Air Pollutant
HEC	Human Equivalent Concentration(s)
HED	Human Equivalent Dose(s)
HEDD	Human Equivalent Dermal Dose
HFC	Hydrofluorocarbon
HHE	Health Hazard Evaluation
HMTA	Hazardous Materials Transportation Act
Hr	Hour(s)
HR	Hazard Ratio
HSE	Health and Safety Executive
HSIA	Halogenated Solvents Industry Alliance

HUC	Hydrologic Unit Code
IARC	International Agency for Research on Cancer
ICIS	Integrated Compliance Information System
IDLH	Immediately Dangerous to Life or Health
IH	Industrial Hygiene
IMAP	Inventory Multi-Tiered Assessment and Prioritisation
IPCS	International Programme on Chemical Safety
IRIS	Integrated Risk Information System
IRR	Incidence rate ratios
ISHA	Industrial Safety and Health Act
IUR	Inhalation Unit Risk
K _{oc}	Soil Organic Carbon-Water Partitioning Coefficient
K _{ow}	Octanol/Water Partition Coefficient
kg	Kilogram(s)
L	Liter(s)
LADC	Lifetime Average Daily Concentration
lb	Pound(s)
LC ₅₀	Lethal Concentration at which 50% of test organisms die
LCL	Lower confidence limit
LOAEC	Lowest Observed Adverse Effect Concentration
LOAEL	Lowest Observed Adverse Effect Level
LOD	Limit of Detection
LOEC	Lowest Observable Effect Concentration
Log K _{oc}	Logarithmic Organic Carbon:Water Partition Coefficient
Log K _{ow}	Logarithmic Octanol:Water Partition Coefficient
m ³	Cubic Meter(s)
MACT	Maximum Achievable Control Technology
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MFO	Mixed Function Oxidase
mg	Milligram(s)
Min	Minute(s)
MLD	Millions of Liters per Day
mmHg	Millimeter(s) of Mercury
MOA	Mode of Action
MOE	Margin of Exposure
mPa·s	Millipascal(s)-Second
MSDS	Material Safety Data Sheet
MSW	Municipal Solid Waste
N/A	Not Applicable
NAC	National Advisory Committee
NAICS	North American Industry Classification System
NATA	National Air Toxics Assessment
NAWQA	National Water Quality Assessment Program
ND	Not Detected
NEI	National Emissions Inventory
NESHAP	National Emission Standards for Hazardous Air Pollutants
NHANES	National Health and Nutrition Examination Survey
NHL	Non-Hodgkin Lymphoma

NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NITE	National Institute of Technology and Evaluation
NMDA	N-Methyl-D-Aspartate
NMP	N-Methylpyrrolidone
NO	Nitric Oxide
NOAEL	No Observed Adverse Effect Level
NOEC	No Observed Effect Concentration
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulation
NPL	National Priority List
NRC	National Research Council
NT	Not tested
NTP	National Toxicology Program
NWIS	National Water Information System
OCSPP	Office of Chemical Safety and Pollution Prevention
OECD	Organisation for Economic Co-operation and Development
OEHHA	Office of Environmental Health Hazard Assessment
OEL	Occupational Exposure Limit
OES	Occupational Exposure Scenario
ONU	Occupational Non-User
OPPT	Office of Pollution Prevention and Toxics
OR	Odds Ratio
ORD	Office of Research and Development
OSHA	Occupational Safety and Health Administration
OTVD	Open-Top Vapor Degreaser
OW	Office of Water
PAH	Polycyclic Aromatic Hydrocarbons
PBMC	Peripheral Blood Mononuclear Cells
PBPK	Physiologically-Based Pharmacokinetic
PBPK/PD	Physiologically-Based Pharmacokinetic/Pharmacodynamic
PDM	Probabilistic Dilution Model
PE	Polyethylene
PECO	Population, Exposure, Comparator, and Outcome
PEL	Permissible Exposure Limit
PESS	Potentially Exposed or Susceptible Subpopulations
PF	Protection Factor
POD	Point of Departure
POTW	Publicly Owned Treatment Works
ppb	Part(s) per Billion
PPE	Personal Protective Equipment
ppm	Part(s) per Million
PVA	Polyvinyl Alcohol
PXR	Pregnane X Receptor
QC	Quality Control
QSAR	Quantitative Structure-Activity Relationships
RBC	Red blood cell
RCRA	Resource Conservation and Recovery Act

RD	Relative Deviation
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
REL	Reference Exposure Level for California EPA OEHHA
RfC	Reference Concentration
RfD	Reference Dose
RICE	Reciprocating Internal Combustion Engines
ROS	Reactive Oxygen Species
RQ	Risk Quotient
RTR	Risk and Technology Review
SAR	Supplied Air Respirator
SCBA	Self-Contained Breathing Apparatus
SD	Standard Deviation
SDH	Succinate Dehydrogenase
SDS	Safety Data Sheets
SDWA	Safe Drinking Water Act
SEMS	Superfund Enterprise Management System
SIC	Standard Industrial Classification
SIDS	Screening Information Data Set
SIR	Standard Incidence Rate
SMAC	Spacecraft Maximum Allowable Concentrations
SMR	Standardized Mortality Ratio
SNAP	Significant New Alternatives Policy
SpERC	Specific Environmental Release Categories
STEL	Short-Term Exposure Limit
STEWARDS	Sustaining the Earth's Watersheds – Agricultural Research Database System
STORET	STorage and RETrieval database
STPWIN	EPI Suite™ model of chemical removal in Sewage Treatment Plants
SVOC	Semivolatile Organic Compounds
SWC	Surface Water Concentration
TLV	Threshold Limit Value
TNO	The Netherlands Organisation for Applied Scientific Research
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TSDF	Treatment, Storage, and Disposal Facility
TTO	Total Toxic Organics
TWA	Time-Weighted Average
UCL	Upper confidence limit
UF	Uncertainty Factor
UF _A	Interspecies Uncertainty/Variability Factor
UF _H	Interspecies Uncertainty Factor
UF _L	LOAEL-to-NOAEL Uncertainty Factor
U.K.	United Kingdom
U.S.	United States
U.S.C.	United States Code
USGS	United States Geological Survey
VOC	Volatile Organic Compound
VER	Visual Evoked Response
WHO	World Health Organization
wk	Week

WQP	Water Quality Portal
WQX	Water Quality Exchange
WY	Exposed Working Years per Lifetime
Yr	Year(s)

EXECUTIVE SUMMARY

This risk evaluation for methylene chloride was performed in accordance with the Frank R. Lautenberg Chemical Safety for the 21st Century Act and is being issued following public comment and peer review. The Frank R. Lautenberg Chemical Safety for the 21st Century Act amended the Toxic Substances Control Act (TSCA), the Nation's primary chemicals management law, in June 2016. Under the amended statute, EPA is required, under TSCA § 6(b), to conduct risk evaluations to determine whether a chemical substance presents unreasonable risk of injury to health or the environment, under the conditions of use, without consideration of costs or other non-risk factors, including an unreasonable risk to potentially exposed or susceptible subpopulations, identified as relevant to the risk evaluation. Also, as required by TSCA § (6)(b), EPA established, by rule, a process to conduct these risk evaluations. [*Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* \(82 FR 33726\)](#). (Risk Evaluation Rule). This risk evaluation is in conformance with TSCA § 6(b), and the Risk Evaluation Rule, and is to be used to inform risk management decisions. In accordance with TSCA section 6(b), if EPA finds unreasonable risk from a chemical substance under its conditions of use in any final risk evaluation, the Agency will propose actions to address those risks within the timeframe required by TSCA. However, any proposed or final determination that a chemical substance presents unreasonable risk under TSCA section 6(b) is not the same as a finding that a chemical substance is "imminently hazardous" under TSCA section 7. The conclusions, findings, and determinations in this final risk evaluation are for the purpose of identifying whether the chemical substance presents unreasonable risk or no unreasonable risk under the conditions of use, in accordance with TSCA Section 6, and are not intended to represent any findings under TSCA Section 7.

TSCA § 26(h) and (i) require EPA, when conducting risk evaluations, to use scientific information, technical procedures, measures, methods, protocols, methodologies and models consistent with the best available science and to base its decisions on the weight of the scientific evidence.¹ To meet these TSCA § 26 science standards, EPA used the TSCA systematic review process described in the *Application of Systematic Review in TSCA Risk Evaluations* document ([U.S. EPA, 2018a](#)). The data collection, evaluation, and integration stages of the systematic review process are used to develop the exposure, fate, and hazard assessments for risk evaluations.

Methylene chloride has a wide range of uses, including as a solvent, propellant, processing aid, or functional fluid in the manufacturing of other chemicals. A variety of consumer and commercial products use methylene chloride as a solvent including sealants, automotive products, and paint and coating removers. Methylene chloride is subject to federal and state regulations and reporting requirements. Methylene chloride has been reportable to Toxics Release Inventory (TRI) chemical under Section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) since 1987. It is designated a Hazardous Air Pollutant (HAP) under the Clean Air Act (CAA), and is a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). It is subject to National Primary Drinking Water Regulations (NPDWR) under the Safe Drinking Water Act (SDWA) and designated as a toxic pollutant under the Clean Water Act (CWA) making it subject to effluent limitations. Under TSCA, EPA previously assessed the use of methylene

¹ Weight of the scientific evidence means a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a pre-established protocol to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance.

chloride in paint and coating removal ([U.S. EPA, 2014](#)). In March 2019 EPA issued a final rule, where the Agency made the determination that the use of methylene chloride in consumer paint and coating removal presents an unreasonable risk of injury to health due to acute human lethality (84 FR 1140). To address this unreasonable risk, the Agency prohibited the manufacture (including import), processing, and distribution in commerce of methylene chloride for paint and coating removal, including distribution to and by retailers; required manufacturers (including importers), processors, and distributors, except retailers, of methylene chloride for any use to provide downstream notification of these prohibitions; and required recordkeeping. The final rule took effect on May 28, 2019.

Methylene chloride is currently manufactured, processed, distributed, used, and disposed of as part of additional industrial, commercial, and consumer conditions of use. Leading applications for methylene chloride include as a solvent in the production of pharmaceuticals and polymers, metal cleaning, production of HFC-32, and as an ingredient in adhesives and paint removers. EPA evaluated the following categories of conditions of use: manufacturing; processing; distribution in commerce, industrial, commercial and consumer uses and disposal.² The total aggregate production volume ranged from 230 to 264 million pounds between 2012 and 2015 according to CDR (Section 1.2).

Approach

EPA used reasonably available information (defined in 40 CFR 702.33 in part as “*information that EPA possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines . . . for completing the evaluation . . .*”), in a fit-for-purpose approach, to develop a risk evaluation that relies on the best available science and is based on the weight of the scientific evidence. EPA used previous assessments, for example EPA’s IRIS assessment, as a starting point for identifying key and supporting studies to inform the exposure, fate, and hazard assessments. EPA also evaluated other studies published since the publication of previous analyses. EPA reviewed reasonably available the information and evaluated the quality of the methods and reporting of results of the individual studies using the evaluation strategies described in *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). To satisfy requirements in TSCA section 26(j)(4) and 40 CFR 702.51(e), EPA has provided a list of studies considered in carrying out the risk evaluation and the results of those studies in Appendix H, Appendix K, and several supplemental files ([EPA, 2019f](#)); ([EPA, 2019e](#)); ([EPA, 2019d](#)); ([EPA, 2019c](#)); ([EPA, 2019q](#)); ([EPA, 2019p](#)); ([EPA, 2019r](#)); ([EPA, 2019u](#)); ([EPA, 2019s](#)); ([EPA, 2019t](#)); ([EPA, 2019a](#)); ([EPA, 2019o](#)).

In the problem formulation, EPA identified the conditions of use within the scope of the risk evaluation and presented three conceptual models and an analysis plan for this risk evaluation ([U.S. EPA, 2018c](#)). These have been carried into the risk evaluation where EPA has quantitatively evaluated the risk to the environment and human health, using both monitoring data and modeling approaches, for the conditions of use (identified in Section 1.4.1 of this risk evaluation).³ EPA quantitatively evaluated the risk to aquatic species from exposure to surface water where, as a result of the manufacturing, processing, use, or disposal of methylene chloride. EPA evaluated the risk to workers, from inhalation

² Although EPA has identified both industrial and commercial uses here for purposes of distinguishing scenarios in this analysis, the Agency interprets the authority over “any manner or method of commercial use” under TSCA section 6(a)(5) to reach both.

³ EPA did not identify any “legacy uses” or “associated disposals” of methylene chloride, as those terms are described in EPA’s Risk Evaluation Rule, 82 FR 33726 (July 20, 2017). Therefore, no such uses or disposals were added to the scope of the risk evaluation for methylene chloride following the issuance of the opinion in *Safer Chemicals, Healthy Families v. EPA*, 943 F.3d 397 (9th Cir. 2019).

and dermal exposures, and occupational non-users (ONUs)⁴, from inhalation exposures, by comparing the estimated acute and chronic exposures to human health hazards (e.g., CNS effects, liver effects, and liver and lung tumors). EPA also evaluated the risk to consumers, from acute inhalation and dermal exposures, and bystanders, from inhalation exposures, by comparing the estimated exposures to acute human health hazards.

EPA used environmental fate parameters, physical-chemical properties, modelling, and monitoring data to assess ambient water exposure to aquatic organisms and sediment-dwelling organisms. While methylene chloride is present in various environmental media, such as groundwater, surface water, and air, EPA determined during problem formulation that no further analysis beyond what was presented in the problem formulation document would be done for environmental exposure pathways in this risk evaluation. While these exposure pathways remain in the scope of the risk evaluation, EPA found no further analysis was necessary in the risk evaluation for sediment, soil and land-applied biosolid pathways leading to exposure to terrestrial and aquatic organisms. Further analysis was not conducted for biosolid, soil and sediment pathways based on a qualitative assessment of the physical-chemical properties and fate of methylene chloride in the environment and a quantitative comparison of hazards and exposures for aquatic and terrestrial organisms. However, exposures to aquatic organisms from surface water, are assessed and presented in this risk evaluation and used to inform the risk determination. These analyses are described in Sections 2.1, 2.3, and 4.1.

EPA evaluated exposures to methylene chloride in occupational and consumer settings for the conditions of use included in the scope of the risk evaluation, listed in Section 1.4 (Scope of the Evaluation). In occupational settings, EPA evaluated acute and chronic inhalation exposures to workers and ONUs, and acute and chronic dermal exposures to workers. EPA used inhalation monitoring data from literature sources that met data evaluation criteria, where reasonably available. EPA also used modeling approaches, where reasonably available, to estimate potential inhalation exposures. Dermal doses for workers were estimated in occupational exposure scenarios since dermal monitoring data was not reasonably available. In consumer settings, EPA evaluated acute inhalation exposures to both consumers and bystanders, and acute dermal exposures to consumers. Inhalation exposures and dermal doses for consumers and bystanders in these scenarios were estimated since inhalation and dermal monitoring data were not reasonably available. These analyses are described in Section 2.4 of this risk evaluation.

EPA reviewed the environmental hazard data using the data quality review evaluation metrics and the rating criteria described in the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). EPA concluded that methylene chloride poses a hazard to environmental aquatic receptors with amphibians being the most sensitive taxa for both acute and chronic exposures. The results of the environmental hazard assessment are in Section 3.1.

EPA evaluated reasonably available information for human health hazards and identified hazard endpoints including acute and chronic toxicity for non-cancer effects and cancer. EPA used the *Framework for Human Health Risk Assessment to Inform Decision Making* ([EPA, 2014a](#)) to evaluate, extract, and integrate methylene chloride's human health hazard and dose-response information. EPA reviewed key and supporting information from previous hazard assessments [EPA OPPT Risk Assessment ([U.S. EPA, 2014](#)), EPA IRIS Toxicologic Review ([U.S. EPA, 2011](#)), an ATSDR Toxicological Profile ([ATSDR, 2000](#)) and ([ATSDR, 2010](#)) addendum, an Interim AEGL ([Nac/Aegl,](#)

⁴ ONUs are workers who do not directly handle methylene chloride but perform work in an area where methylene chloride is present.

[2008b](#)), Spacecraft Maximum Allowable Concentrations Assessment ([Nrc, 1996](#)), Report on Carcinogens, Twelfth Edition, Dichloromethane ([NIH, 2016](#)), Occupational Exposure to Methylene Chloride (OSHA) ([1997b](#)), Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride ([Oehha, 2008a](#)) and other international assessments listed in Table 1-3]. EPA also screened and evaluated new studies that were published since these reviews (i.e., from 2011 – 2018).

EPA developed a hazard and dose-response analysis using endpoints observed in inhalation and oral hazard studies, evaluated the weight of the scientific evidence considering EPA and National Research Council (NRC) risk assessment guidance, and selected the points of departure (POD) for acute and chronic non-cancer endpoints, and inhalation unit risk and cancer slope factors for cancer risk estimates. Potential health effects of methylene chloride exposure described in the literature include effects on the central nervous system (CNS), liver, immune system, as well as irritation/burns, and cancer. EPA identified acute PODs for inhalation and dermal exposures based on acute CNS effects observed in humans ([Putz et al., 1979](#)). The chronic POD for inhalation exposures are based on a study observing increased liver vacuolation in rats ([Nitschke et al., 1988a](#)). EPA used a probabilistic physiologically-based pharmacokinetic (PBPK) model for interspecies extrapolation from rats to humans and for toxicokinetic variability among humans. EPA searched for, but did not identify, toxicity studies by the dermal route that were adequate for dose-response assessment. Therefore, dermal candidate values were derived by route-to-route extrapolation from the inhalation PODs mentioned above. In accordance with U.S. EPA ([EPA, 2005a](#)) *Guidelines for Carcinogen Risk Assessment*, methylene chloride is considered “likely to be carcinogenic to humans” based on sufficient evidence in animals, limited supporting evidence in humans, and mechanistic data showing a mutagenic mode of action (MOA) relevant to humans. EPA calculated cancer risk with a linear model using cancer slope factors based on evidence of increased risk of cancer in mice exposed to methylene chloride through air ([Aiso et al., 2014a](#); [NTP, 1986](#)). The results of these analyses are described in Section 3.2.

Risk Characterization

Environmental Risk: For environmental risk, EPA utilized a risk quotient (RQ) to compare the environmental concentration to the effect level to characterize the risk to aquatic organisms. EPA included a quantitative assessment describing methylene chloride exposure from surface water and sediments. The results of the risk characterization are in Section 4.2, including a table that summarizes the RQs for acute and chronic risks.

EPA identified expected environmental exposures for aquatic species under the conditions of use in the scope of the risk evaluation. While the estimated releases from specific facilities result in modeled surface water concentrations that were equal to or exceed the aquatic benchmark ($RQ \geq 1$), all but two conditions of use (recycling and disposal) had $RQs < 1$, indicating that exposures resulting from environmental concentrations were less than the effect concentration, or the concentration of concern. Details of these estimates are in Section 4.2.2.

Human Health Risks: For human health risks to workers and consumers, EPA identified potential cancer and non-cancer human health risks. Risks from acute exposures include central nervous system risks such as central nervous system depression and a decrease in peripheral vision, each of which can lead to workplace accidents and which are precursors to more severe central nervous system effects such as incapacitation, loss of consciousness, and death. For chronic exposures, EPA identified risks of non-cancer liver effects as well as liver and lung tumors.

For workers and ONUs, EPA estimated potential cancer risk from chronic exposures to methylene chloride using inhalation unit risk or dermal cancer slope factor values multiplied by the chronic

exposure for each COU. For workers and ONUs, EPA also estimated potential non-cancer risks resulting from acute or chronic inhalation or dermal exposures and used a Margin of Exposure (MOE) approach. For workers, EPA estimated risks using several occupational exposure scenarios, which varied assumptions regarding the use of personal protective equipment (PPE) for respiratory and dermal exposures for workers directly handling methylene chloride. More information on respiratory and dermal protection, including EPA's approach regarding the occupational exposure scenarios for methylene chloride, is in Section 2.4.1.

For workers, acute and chronic non-cancer risks (i.e., central nervous system effects and non-cancer liver effects) were indicated for all conditions of use under high-end inhalation or dermal exposure scenarios if PPE was not used. For most industrial and commercial conditions of use, cancer risks were also identified for high-end inhalation or dermal occupational exposure scenarios if PPE was not used. With use of PPE during relevant conditions of use, worker exposures were estimated to be reduced. This resulted in fewer conditions of use with estimated acute, chronic non-cancer, or cancer inhalation or dermal risks. With use of respiratory protection, cancer risks from chronic inhalation risks were not indicated for most conditions of use. Similarly, with dermal protection, non-cancer risks from acute and chronic exposures, and cancer risks were not indicated for most conditions of use. However, some conditions of use continued to present non-cancer inhalation risks to workers under high end occupational exposure scenarios even with PPE (respirators APF 25 or 50, and gloves of various protection factors). Specifically, even with use of respirators (APF 25 or 50), acute and chronic non-cancer risks were indicated for processing methylene chloride as part of one condition of use and for most industrial and commercial uses of methylene chloride. EPA's estimates for worker risks for each occupational exposure scenario are presented in Section 4.3.2.1 and summarized in Table 4-106 in Section 4.1.2.

For ONUs, acute and chronic non-cancer risks (i.e., central nervous system effects and non-cancer liver effects) were indicated for high-end inhalation occupational exposure scenarios for processing methylene chloride as part of several conditions of use, and for most industrial and commercial uses of methylene chloride. Central tendency estimates of inhalation exposures showed that while fewer conditions of use indicated non-cancer risks to ONUs from acute or chronic exposures, under many conditions of use, inhalation risks remained. ONUs were not assumed to be using PPE to reduce exposures to methylene chloride used in their vicinity. ONUs are not assumed to be dermally exposed to methylene chloride; therefore, dermal risks to ONUs were not identified. EPA's estimates for ONU risks for each occupational exposure scenario are presented in Sections 4.3.2.1 and 4.3.2.2 and Table 4-2 in Section 4.1.2.

For consumers and bystanders for consumer use, EPA estimated non-cancer risks resulting from acute inhalation or dermal exposures that were modeled with a range of user intensities, described in detail in Section 2.4.2. EPA assumed that consumers or bystanders would not use PPE and that all exposures would be acute, rather than chronic. As explained in Section 4.3.2.3,

For consumers and bystanders, risks from acute exposure (of central nervous system effects) were indicated for most conditions of use for consumers for medium and high intensity acute inhalation and dermal consumer exposure scenarios. Conditions of use that indicated acute risks to consumer users (for inhalation and dermal exposure) also indicated risks to bystanders (for inhalation exposures only). As explained in Section 4.3.2.3, estimates of MOEs for consumers were calculated for consumers for acute inhalation and dermal exposures, because the exposure frequencies were not considered sufficient to cause the health effects (i.e., liver effects and liver and lung tumors) that were observed in chronic animal studies typically defined as at least 10% of the animal's lifetime

Uncertainties: Key assumptions and uncertainties in the environmental risk estimation include the uncertainty around modeled releases. For the human health risk estimation, key assumptions and uncertainties are related to the estimates for ONU inhalation exposures, because monitoring data were not reasonably available for many of the conditions of use evaluated. An additional source of uncertainty is the inhalation to dermal route-to-route extrapolations, which is a source of uncertainty in the dermal risk assessment for dermal cancer and non-cancer risk estimates. Similarly, for assessing cancer risks, although EPA chose to model the combination of liver and lung tumor results from a cancer bioassay using mice, there is uncertainty regarding the modeling of these tumor types for humans. These and other assumptions and key sources of uncertainty are detailed in Section 4.4.

EPA's assessments, risk estimations, and risk determinations account for uncertainties throughout the risk evaluation. EPA used reasonably available information, in a fit-for-purpose approach, to develop a risk evaluation that relies on the best available science and is based on the weight of the scientific evidence. For instance, systematic review was conducted to identify reasonably available information related to MC hazards and exposures. If no applicable monitoring data were identified, exposure scenarios were assessed using a modeling approach that requires the input of various chemical parameters and exposure factors. When possible, default model input parameters were modified based on chemical-specific inputs available in literature databases. The consideration of uncertainties support the Agency's risk determinations, each of which is supported by substantial evidence, as set forth in detail in later sections of this final risk evaluation.

Potentially Exposed Susceptible Subpopulations: TSCA § 6(b)(4) requires that EPA conduct risk evaluations to determine whether a chemical substance presents unreasonable risk under the conditions of use, including unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation. TSCA § 3(12) defines "*potentially exposed or susceptible subpopulation*" as a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly.

In developing the risk evaluation, EPA analyzed reasonably available information to ascertain whether some human receptor groups may have greater exposure or greater susceptibility than the general population to the hazard posed by methylene chloride. For consideration of the most highly exposed groups, EPA considered methylene chloride exposures to be higher among workers using methylene chloride and ONUs in the vicinity of methylene chloride use than the exposures experienced by the general population. Additionally, variability of susceptibility to methylene chloride may be correlated with genetic polymorphism in its metabolizing enzymes. Factors other than polymorphisms that regulate CYP2E1 may have greater influence on the formation of COHb, a metabolic product of methylene chloride exposure. The CYP2E1 enzyme is easily inducible by many substances, resulting in increased metabolism. For example, alcohol drinkers may have increased CO and COHb ([Nac/Aegl, 2008b](#)). Additionally, the COHb generated from methylene chloride is expected to be additive to COHb from other sources. Populations of particular concern are smokers who maintain significant constant levels of COHb, persons with existing cardiovascular disease ([ATSDR, 2000](#)), as well as fetuses and infants. Hemoglobin in the fetus has a higher affinity for CO than does adult hemoglobin. Thus, the neurotoxic and cardiovascular effects may be exacerbated in fetuses and infants with higher residual levels of fetal hemoglobin when exposed to high concentrations of methylene chloride ([OEHHA, 2008b](#)).

Aggregate and Sentinel Exposures Section 2605(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk evaluation, describe whether aggregate or sentinel exposures under the conditions of use were considered and the basis for their consideration. The EPA has defined aggregate exposure as “*the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways* (40 CFR § 702.33).” Exposures to methylene chloride were evaluated by inhalation and dermal routes separately. Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers. EPA chose not to employ simple additivity of exposure pathways at this time within a condition of use, because it would result in an overestimate of risk.

EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures* (40 CFR § 702.33).” In this risk evaluation, EPA considered sentinel exposure the highest exposure given the details of the conditions of use and the potential exposure scenarios. In terms of this risk evaluation, EPA considered sentinel exposure the highest exposure given the details of the conditions of use and the potential exposure scenarios. Sentinel exposures for workers are the high-end no PPE within each OES. In cases where sentinel exposures result in MOEs greater than the benchmark or cancer risk lower than the benchmark, EPA did no further analysis because sentinel exposures represent the worst-case scenario.

Unreasonable Risk Determination

In each risk evaluation under TSCA section 6(b), EPA determines whether a chemical substance presents an unreasonable risk of injury to health or the environment, under the conditions of use. The determination does not consider costs or other non-risk factors. In making this determination, EPA considers relevant risk-related factors, including, but not limited to: the effects of the chemical substance on health and human exposure to such substance under the conditions of use (including cancer and non-cancer risks); the effects of the chemical substance on the environment and environmental exposure under the conditions of use; the population exposed (including any potentially exposed or susceptible subpopulations, as determined by EPA); the severity of hazard (including the nature of the hazard, the irreversibility of the hazard); and uncertainties. EPA also takes into consideration the Agency’s confidence in the data used in the risk estimate. This includes an evaluation of the strengths, limitations, and uncertainties associated with the information used to inform the risk estimate and the risk characterization. The rationale for the unreasonable risk determination is in section 5.2. The Agency’s risk determinations are supported by substantial evidence, as set forth in detail in later sections of this final risk evaluation.

While use of methylene chloride as a functional fluid in a closed system during pharmaceutical manufacturing was included in the problem formulation and draft risk evaluation, upon further analysis of the details of this process, EPA has determined that this use falls outside TSCA’s definition of “chemical substance.” Under TSCA § 3(2)(B)(vi), the definition of “chemical substance” does not include any food, food additive, drug, cosmetic, or device (as such terms are defined in section 201 of the Federal Food, Drug, and Cosmetic Act) when manufactured, processed, or distributed in commerce for use as a food, food additive, drug, cosmetic, or device. EPA has found that methylene chloride use as a functional fluid in a closed system during pharmaceutical manufacturing entails use as an extraction solvent in the purification of pharmaceutical products, and has concluded that this use falls within the aforementioned definitional exclusion and is not a “chemical substance” under TSCA.

Unreasonable Risk of Injury to the Environment: Based on its physical-chemical properties, methylene chloride does not partition to or accumulate in soil. Therefore, EPA determined that there is no unreasonable risk to terrestrial organisms from all conditions of use. To characterize the exposures to

methylene chloride by aquatic organisms EPA considered modeled data to represent surface water concentrations near facilities actively releasing methylene chloride to surface water, as well as monitored concentrations to represent ambient water concentrations of methylene chloride. EPA considered the biological relevance of the species to determine the concentrations of concern, as well as frequency and duration of the exposures, and uncertainties of the limited number of data points above the RQ. EPA determined that the evaluation does not support an unreasonable risk determination to aquatic organisms. Similarly, EPA determined that the evaluation does not support an unreasonable risk determination to sediment dwelling organisms, since methylene chloride is most likely present in the pore waters and the concentrations in sediment pore water are assumed to be similar or less to the concentrations in the overlying water.

Unreasonable Risks of Injury to Health: EPA's determination of unreasonable risk for specific conditions of use of methylene chloride listed below are based on health risks to workers, ONUs, consumers, or bystanders from consumer use. As described below, EPA did not evaluate unreasonable risk to the general population in this risk evaluation. For acute exposures, EPA evaluated unreasonable risk to the central nervous system, such as central nervous system depression and a decrease in peripheral vision, each of which can lead to workplace accidents and which are precursors to more severe central nervous system effects such as incapacitation, loss of consciousness, and death. For chronic exposures, EPA evaluated unreasonable risk of non-cancer liver effects (including vacuolization, necrosis, hemosiderosis and hepatocellular degeneration) as well as cancer (liver and lung tumors).

Unreasonable Risk of Injury to Health of the General Population: As part of the problem formulation for methylene chloride, EPA found that exposures to the general population may occur from the conditions of use due to releases to air, water or land. The exposures to the general population via surface water, drinking water, ambient air and sediment pathways falls under the jurisdiction of other environmental statutes administered by EPA, i.e., CAA, SDWA, CWA, and RCRA. As explained in more detail in section 1.4.2, EPA believes it is both reasonable and prudent to tailor TSCA risk evaluations when other EPA offices have expertise and experience to address specific environmental media, rather than attempt to evaluate and regulate potential exposures and risks from those media under TSCA. EPA believes that coordinated action on exposure pathways and risks addressed by other EPA-administered statutes and regulatory programs is consistent with statutory text and legislative history, particularly as they pertain to TSCA's function as a "gap-filling" statute, and also furthers EPA aims to efficiently use Agency resources, avoid duplicating efforts taken pursuant to other Agency programs, and meet the statutory deadline for completing risk evaluations. EPA has therefore tailored the scope of the risk evaluation for methylene chloride using authorities in TSCA sections 6(b) and 9(b)(1). EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and as such the unreasonable risk determinations for relevant conditions of use do not account for exposures to the general population ([U.S. EPA, 2018c](#)).

Unreasonable Risk of Injury to Health of Workers: EPA evaluated non-cancer effects from acute and chronic inhalation and dermal occupational exposures and cancer from chronic inhalation and dermal occupational exposures to determine if there was unreasonable risk to workers' health. The drivers for EPA's determination of unreasonable risk of injury for workers are central nervous system effects resulting from acute inhalation exposure, adverse effects to the liver due to chronic inhalation exposure, and cancer from chronic inhalation.

EPA evaluated unreasonable risk to workers from dermal occupational exposure and determined unreasonable risk to workers from dermal exposure from one condition of use: the industrial and

commercial use of methylene chloride in laundry and dishwashing, where EPA is not assuming use of gloves in dry cleaning facilities.

EPA generally assumes compliance with OSHA requirements for protection of workers. In support of this assumption, EPA used reasonably available information, including public comments, indicating that some employers, particularly in the industrial setting, are providing appropriate engineering or administrative controls or PPE to their employees consistent with OSHA requirements. While EPA does not have similar information to support this assumption for each condition of use, EPA does not believe that the Agency must presume, in the absence of such information, a lack of compliance with existing regulatory programs and practices. Rather, EPA assumes there is compliance with worker protection standards unless case-specific facts indicate otherwise, and therefore existing OSHA regulations for worker protection and hazard communication will result in use of appropriate PPE in a manner that achieves the stated APF or PF. EPA's decisions for unreasonable risk to workers are based on high-end exposure estimates, in order to account for the uncertainties related to whether or not workers are using PPE. EPA believes this is a reasonable and appropriate approach that reflects real-world scenarios, accounts for reasonably available information related to worker protection practices, and addresses uncertainties regarding availability and use of PPE.

For each condition of use of methylene chloride with an identified risk for workers, EPA assumes, as a baseline, the use of a respirator with an APF of 25 or 50. Similarly, EPA assumes the use of gloves with PF of 5 and 10 in commercial settings and gloves with PF of 5 and 20 in industrial settings. However, EPA assumes that for some conditions of use, the use of appropriate respirators is not a standard industry practice, based on best professional judgement given the burden associated with the use of supplied-air respirators, including the expense of the equipment and the necessity of fit-testing and training for proper use. Similarly, EPA does not assume that as a standard industry practice that workers in dry cleaning facilities use gloves for spot cleaning.

The unreasonable risk determinations reflect the severity of the effects associated with the occupational exposures to methylene chloride and incorporate consideration of the PPE that EPA assumes (respirator of APF 25 or 50 and gloves with PF 5, 10, or 20). A full description of EPA's unreasonable risk determination for each condition of use is in section 5.2.

Unreasonable Risk of Injury to Health of Occupational Non-Users (ONUs): EPA evaluated non-cancer effects to ONUs from acute and chronic inhalation occupational exposures and cancer from chronic inhalation occupational exposures to determine if there was unreasonable risk of injury to ONUs' health. The unreasonable risk determinations reflect the severity of the effects associated with the occupational exposures to methylene chloride and the assumed absence of PPE for ONUs, since ONUs do not directly handle the chemical and are instead doing other tasks in the vicinity of methylene chloride use. Non-cancer effects and cancer from dermal occupational exposures to ONUs were not evaluated because ONUs are not dermally exposed to methylene chloride. For inhalation exposures, EPA, where possible, estimated ONUs' exposures and described the risks separately from workers directly exposed. When the difference between ONUs' exposures and workers' exposures cannot be quantified, EPA assumed that ONU inhalation exposures are lower than inhalation exposures for workers directly handling the chemical substance, and EPA considered the central tendency risk estimate when determining ONU risk. A full description of EPA's unreasonable risk determination for each condition of use is in section 5.2.

Unreasonable Risk of Injury to Health of Consumers: EPA evaluated non-cancer effects to consumers from acute inhalation and dermal exposures to determine if there was unreasonable risk to consumers' health. A consumer condition of use sometimes was evaluated using multiple Consumer Exposure

Scenarios. In the Draft Risk Evaluation, EPA used the results from each Consumer Exposure Scenario to draft separate preliminary unreasonable risk determinations, which resulted in multiple preliminary unreasonable risk determinations for a single condition of use (e.g., consumer use in metal degreasers had three unreasonable risk determinations). In this Final Risk Evaluation, EPA consolidated risk estimates for multiple exposure scenarios in order to present clearer unreasonable risk determinations and the unreasonable risk determinations adhere to the conditions of use as they were presented in the Problem Formulation; as a result, in some cases a single determination may be informed by multiple risk estimates from multiple Consumer Exposure Scenarios. Therefore, whereas the draft Risk Evaluation presented 29 consumer risk determinations on 12 conditions of use, the Final Evaluation shows only the 12. Overall, the Draft Risk Evaluation had 71 unreasonable risk determinations, whereas the Final Risk Evaluation determination has 53 unreasonable risk determinations. The exposure scenarios supporting the unreasonable risk determinations for the conditions of use are listed in the detailed description of each consumer use and listed in Table 5-2.

Unreasonable Risk of Injury to Health of Bystanders (from Consumer Uses): EPA evaluated non-cancer effects to bystanders from acute inhalation exposures to determine if there was unreasonable risk of injury to bystanders' health. EPA did not evaluate non-cancer effects from dermal exposures to bystanders because bystanders are not dermally exposed to methylene chloride. A full description of EPA's unreasonable risk determination for each condition of use is in section 5.2.

Summary of Unreasonable Risk Determinations:

In conducting risk evaluations, "EPA will determine whether the chemical substance presents an unreasonable risk of injury to health or the environment under each condition of use within the scope of the risk evaluation..." 40 CFR 702.47. Pursuant to TSCA section 6(i)(1), a determination of "no unreasonable risk" shall be issued by order and considered to be final agency action. This subsection of the final risk evaluation therefore constitutes the order required under TSCA section 6(i)(1), and the "no unreasonable risk" determinations in this subsection are considered to be final agency action effective on the date of issuance of this order.

EPA has determined that the following conditions of use of methylene chloride do not present an unreasonable risk of injury to health or the environment. These determinations are considered final agency action and are being issued by order pursuant to TSCA section 6(i)(1). The details of these determinations are in section 5.2, and the TSCA section 6(i)(1) order is contained in Section 5.4.1 of this final risk evaluation.

Conditions of Use that Do Not Present an Unreasonable Risk
<ul style="list-style-type: none"> • Manufacturing (Domestic Manufacture) • Processing: as a reactant • Processing: recycling • Distribution in commerce • Industrial and commercial use as laboratory chemical • Disposal

EPA has determined that the following conditions of use of methylene chloride present an unreasonable risk of injury to health. EPA will initiate TSCA section 6(a) risk management actions on these conditions of use as required under TSCA section 6(c)(1). Pursuant to TSCA section 6(i)(2), the unreasonable risk determinations for these conditions of use are not considered final agency action. The details of these determinations are in section 5.2.

Manufacturing that Presents an Unreasonable Risk

- Import

Processing that Present an Unreasonable Risk

- Processing: incorporation into a formulation, mixture, or reaction products
- Processing: repackaging

Industrial and Commercial Uses that Present an Unreasonable Risk

- Industrial and commercial use as solvent for batch vapor degreasing
- Industrial and commercial use as solvent for in-line vapor degreasing
- Industrial and commercial use as solvent for cold cleaning
- Industrial and commercial use as solvent for aerosol spray degreaser/cleaner
- Industrial and commercial use in adhesives, sealants and caulks
- Industrial and commercial use in paints and coatings
- Industrial and commercial use in paint and coating removers
- Industrial and commercial use in adhesive and caulk removers
- Industrial and commercial use in metal aerosol degreasers
- Industrial and commercial use in metal non-aerosol degreasers
- Industrial and commercial use in finishing products for fabric, textiles and leather
- Industrial and commercial use in automotive care products (functional fluids for air conditioners)
- Industrial and commercial use in automotive care products (interior car care)
- Industrial and commercial use in automotive care products (degreasers)
- Industrial and commercial use in apparel and footwear care products
- Industrial and commercial use in spot removers for apparel and textiles
- Industrial and commercial use in liquid lubricants and greases

Industrial and Commercial Uses that Present an Unreasonable Risk

- Industrial and commercial use in spray lubricants and greases
- Industrial and commercial use in aerosol degreasers and cleaners
- Industrial and commercial use in non-aerosol degreasers and cleaners
- Industrial and commercial use in cold pipe insulations
- Industrial and commercial use as solvent that becomes part of a formulation or mixture
- Industrial and commercial use as a processing aid
- Industrial and commercial use as propellant and blowing agent
- Industrial and commercial use for electrical equipment, appliance, and component manufacturing
- Industrial and commercial use for plastic and rubber products manufacturing
- Industrial and commercial use in cellulose triacetate film production
- Industrial and commercial use as anti-spatter welding aerosol
- Industrial and commercial use for oil and gas drilling, extraction, and support activities
- Industrial and commercial use in toys, playground and sporting equipment
- Industrial and commercial use in lithographic printing plate cleaner
- Industrial and commercial use in carbon remover, wood floor cleaner, and brush cleaner

Consumer Uses that Present an Unreasonable Risk

- Consumer use as solvent in aerosol degreasers/cleaners
- Consumer use in adhesives and sealants
- Consumer use in brush cleaners for paints and coatings
- Consumer use in adhesive and caulk removers
- Consumer use in metal degreasers
- Consumer use in automotive care products (functional fluids for air conditioners)
- Consumer use in automotive care products (degreasers)
- Consumer use in lubricants and greases
- Consumer use in cold pipe insulation
- Consumer use in arts, crafts, and hobby materials glue
- Consumer use in an anti-spatter welding aerosol

Consumer Uses that Present an Unreasonable Risk

- Consumer use in carbon removers and other brush cleaners

1 INTRODUCTION

This document represents the final risk evaluation for methylene chloride under the Frank R. Lautenberg Chemical Safety for the 21st Century Act. The Frank R. Lautenberg Chemical Safety for the 21st Century Act amended the Toxic Substances Control Act (TSCA), the Nation's primary chemicals management law, in June 2016.

The Environmental Protection Agency (EPA) published the Scope of the Risk Evaluation for methylene chloride in June 2017 ([U.S. EPA, 2017c](#)), and the problem formulation in June 2018 ([U.S. EPA, 2018c](#)), which represented the analytical phase of risk evaluation in which “the purpose for the assessment is articulated, the problem is defined, and a plan for analyzing and characterizing risk is determined,” as described in Section 2.2 of the [Framework for Human Health Risk Assessment to Inform Decision Making](#). The problem formulation identified conditions of use and presented three conceptual models and an analysis plan. Based on EPA's analysis of the conditions of use, physical-chemical and fate properties, environmental releases, and exposure pathways, the problem formulation preliminarily concluded that further analysis was necessary for exposure pathways to ecological receptors exposed via surface water, workers, and consumers. EPA subsequently published a draft risk evaluation for methylene chloride and has taken public and peer review comments. The conclusions, findings, and determinations in this final risk evaluation are for the purpose of identifying whether the chemical substance presents unreasonable risk or no unreasonable risk under the conditions of use, in accordance with TSCA Section 6, and are not intended to represent any findings under TSCA Section 7.

As per EPA's final rule, [Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act](#) (82 FR 33726 (July 20, 2017)), this risk evaluation was subject to both public comment and peer review, which are distinct but related processes. EPA provided 60 days for public comment on any and all aspects of this risk evaluation, including the submission of any additional information that might be relevant to the science underlying the risk evaluation and the outcome of the systematic review associated with methylene chloride. This satisfies TSCA (15 U.S.C. 2605(b)(4)(H)), which requires EPA to provide public notice and an opportunity for comment on a draft risk evaluation prior to publishing a final risk evaluation.

Peer review was conducted in accordance with EPA's regulatory procedures for chemical risk evaluations, including using the [EPA Peer Review Handbook](#) and other methods consistent with the science standards laid out in Section 26 of TSCA (*See* 40 CFR 702.45). As explained in the [Risk Evaluation Rule](#) (82 FR 33726 (July 20, 2017)), the purpose of peer review is for the independent review of the science underlying the risk assessment. As such, peer review addressed aspects of the underlying science as outlined in the charge to the peer review panel such as hazard assessment, assessment of dose-response, exposure assessment, and risk characterization.

As EPA explained in the [Risk Evaluation Rule](#) (82 FR 33726 (July 20, 2017)), it is important for peer reviewers to consider how the underlying risk evaluation analyses fit together to produce an integrated risk characterization, which forms the basis of an unreasonable risk determination. EPA believed peer reviewers were most effective in this role if they received the benefit of public comments on draft risk

evaluations prior to peer review. For this reason, and consistent with standard Agency practice, the public comment period preceded peer review. The final risk evaluation changed in response to public comments received on the draft risk evaluation and/or in response to peer review, which itself may be informed by public comments. EPA responded to public and peer review comments received on the draft risk evaluation and explained changes made in response to those comments in this final risk evaluation and the associated response to comments document.

In this final risk evaluation, Section 1.1 presents the basic physical-chemical characteristics of methylene chloride, as well as a background on regulatory history, conditions of use, and conceptual models, with particular emphasis on any changes since the publication of the draft risk evaluation. This section also includes a discussion of the systematic review process utilized in this final risk evaluation. Section 2 provides a discussion and analysis of the exposures, both health and environmental, that can be expected based on the conditions of use for methylene chloride. Section 3 discusses environmental and health hazards of methylene chloride. Section 4 presents the risk characterization, where EPA integrates and assesses reasonably available information on health and environmental hazards and exposures, as required by TSCA (15 U.S.C. 2605(b)(4)(F)). This section also includes a discussion of any uncertainties and how they impact the final risk evaluation. Section 5 presents EPA's determination of whether the chemical presents an unreasonable risk under the conditions of use, as required under TSCA (15 U.S.C. 2605(b)(4)).

EPA also solicited input on the first 10 chemicals as it developed use documents, scope documents, and problem formulations. At each step, EPA has received information and comments specific to individual chemicals and of a more general nature relating to various aspects of the risk evaluation process, technical issues, and the regulatory and statutory requirements. EPA has considered comments and information received at each step in the process and factored in the information and comments as the Agency deemed appropriate and relevant including comments on the published problem formulation of methylene chloride.

1.1 Physical and Chemical Properties

Physical-chemical properties influence the environmental behavior and the toxic properties of a chemical, thereby informing the potential conditions of use, exposure pathways and routes and hazards that EPA is evaluating. For scope development, EPA considered the measured or estimated physical-chemical properties set forth in Table 1-1. EPA found no additional information during the process of drafting the risk evaluation, nor did it hear of any information from the peer review or public commenters that would change these values for the final risk evaluation.

Table 1-1. Physical and Chemical Properties of Methylene Chloride

Property	Measured Values	References	Data Quality Rating
Molecular formula	CH ₂ Cl ₂		
Molecular weight	84.93 g/mol		
Physical form	Colorless liquid; sweet, pleasant odor resembling chloroform	U.S. Coast Guard (1984)	High
Melting point	-95°C	O'Neil (2013)	High
Boiling point	39.7°C	O'Neil (2013)	High

Property	Measured Values	References	Data Quality Rating
Density	1.33 g/cm ³ at 20°C	O'Neil (2013)	High
Vapor pressure	435 mmHg at 25°C	Boublik et al. (1984)	High
Vapor density	2.93 (relative to air)	Holbrook (2003)	High
Water solubility	13 g/L at 25°C	Horvath (1982)	High
Octanol/water partition coefficient (log K _{ow})	1.25	Hansch et al. (1995)	High
Octanol/air partition coefficient (log K _{OA})	2.27	U.S. EPA (2012)	High
Henry's Law constant	0.00291 atm·m ³ /mole (equivalent to concentration/concentration dimensionless 0.119)	Leighton and Calo (1981)	High
Flash point	Not readily available		
Autoflammability	Not readily available		
Viscosity	0.437 mPa·s at 20°C	Rossberg et al. (2011)	High
Refractive index	1.4244 at 20°C	O'Neil (2013)	High
Dielectric constant	9.02 at 20°C	Laurence et al. (1994)	High

1.2 Uses and Production Volume

Methylene chloride has a wide-range of uses, including in sealants, automotive products, and paint and coating removers. EPA assessed paint removers containing methylene chloride in a previous risk assessment but only previously finalized an unreasonable risk determination for the consumer paint and coating remover condition of use ([U.S. EPA, 2014](#)). The use of paint and coating removers containing methylene chloride in industrial or commercial sectors are included in this risk evaluation; the resultant analysis is described in Appendix L. Methylene chloride is also used by federal agencies in a variety of uses, including those deemed mission critical.

Methylene chloride has known applications as a process solvent in paint removers and the manufacture of pharmaceuticals and film coatings. It is used as an agent in urethane foam blowing and in the manufacture of hydrofluorocarbon (HFC) refrigerants, such as HFC-32. It can also be found in aerosol propellants and in solvents for electronics manufacturing, metal cleaning and degreasing, and furniture finishing. Additionally, it has been used for agricultural and food processing purposes such as an extraction solvent for spice oleoresins, hops, and for the removal of caffeine from coffee, a degreening agent for citrus fruits, and a postharvest fumigant for grains and strawberries ([Processing Magazine, 2015](#); [U.S. EPA, 2000](#)). However methylene chloride is no longer contained in any registered pesticide products and was removed from the list of pesticide product inert ingredients (63 FR 34384, June 24, 1998) and tolerance exemptions for methylene chloride in foods were revoked (67 FR 16027, April 4, 2002) (see Appendix A for more information).

In 2005, the use percentages of methylene chloride by sector were as follows: paint stripping and removal (30%), adhesives (22%), pharmaceuticals (11%), metal cleaning (8%), aerosols (8%), chemical processing (8%), flexible polyurethane foam (5%), and miscellaneous (8%) ([ICIS, 2005](#)).

As of 2016, the leading applications for methylene chloride are as a solvent in the production of pharmaceuticals and polymers and paint removers, although recent regulations are expected to decrease the chemical's use in the paint remover sector (40 CFR Part 751, Part B). An estimated 35 percent of consumption is attributable to pharmaceuticals and chemical processing, with pharmaceutical production accounting for roughly 30 percent of methylene chloride's use. Other applications include metal cleaning, production of HFC-32, and as an ingredient in adhesives and paint removers. Foam blowing is a minor use of methylene chloride ([IHS Markit, 2016](#)).

The Chemical Data Reporting (CDR) Rule under TSCA requires U.S. manufacturers (including importers) to provide EPA with information on the chemicals they manufacture or import into the U.S. For the 2016 CDR cycle, data collected per chemical include the company name, volume of each chemical manufactured/imported, the number of workers at each site, and information on whether the chemical is used in the Commercial, Industrial, and/or Consumer sector. However, only companies that manufactured or imported 25,000 pounds or more of methylene chloride at each of their sites during the 2015 calendar year were required to report information under the CDR rule ([U.S. EPA, 2016](#)).

The 2016 CDR reporting data for methylene chloride are provided in Table 1-2. from EPA's CDR database.

Table 1-2. Production Volume of Methylene Chloride in CDR Reporting Period (2012 to 2015)^a

Reporting Year	2012	2013	2014	2015
Total Aggregate Production Volume (lbs)	230,896,388	230,498,027	248,241,495	263,971,494

^a The CDR data for the 2016 reporting period is available via ChemView (<https://java.epa.gov/chemview>) ([U.S. EPA, 2016](#)). Because of an ongoing Confidential Business Information (CBI) substantiation process required by amended TSCA, the CDR data available in the risk evaluation is more specific than currently in ChemView.

1.3 Regulatory and Assessment History

EPA conducted a search of existing domestic and international laws, regulations and assessments pertaining to methylene chloride. EPA compiled this summary from available federal, state, international and other government data sources, as cited in Appendix A.

Federal Laws and Regulations

Methylene chloride is subject to other federal statutes and regulations that are implemented by other offices within EPA and/or other federal agencies/departments. A summary of federal laws, regulations and implementing authorities is provided in Appendix A.1.

State Laws and Regulations

Methylene chloride is subject to state statutes and regulations implemented by state agencies or departments. A summary of state laws, regulations and implementing authorities is provided in Appendix A.2.

Laws and Regulations in Other Countries and International Treaties or Agreements

Methylene chloride is subject to statutes and regulations in countries other than the U.S. and/or international treaties and/or agreements. A summary of these laws, regulations, treaties and/or agreements is provided in Appendix A.3.

Assessment History

EPA identified assessments conducted by other EPA Programs and other organizations (see Table 1-3). Depending on the source, these assessments may include information on conditions of use, hazards, exposures and potentially exposed or susceptible subpopulations (PESS). EPA found no additional assessments beyond those listed in the Problem Formulation document (see Table 1-1 in Methylene Chloride Problem Formulation document).

Table 1-3. Assessment History of Methylene Chloride

Authoring Organization	Assessment
EPA Assessments	
U.S. EPA, Office of Pollution Prevention and Toxics (OPPT)	TSCA Work Plan Chemical Risk Assessment Methylene Chloride: Paint Stripping Use CASRN: 75-09-2 U.S. EPA (2014)
U.S. EPA, Integrated Risk Information System (IRIS)	Toxicological Review of Dichloromethane (Methylene Chloride) (CAS No. 75-09-2) U.S. EPA (2011)
U.S. EPA, Office of Water (OW)	Ambient Water Quality Criteria for the Protection of Human Health U.S. EPA (2015)
Other U.S.-Based Organizations	
Agency for Toxic Substances and Disease Registry (ATSDR)	Toxicological Profile for Methylene Chloride ATSDR (2000) and ATSDR (2010) addendum
National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee)	Interim Acute Exposure Guideline Levels (AEGL) for Methylene Chloride Nac/Aegl (2008b)
U.S. National Academies, National Research Council (NRC)	Spacecraft Maximum Allowable Concentrations (SMAC) for Selected Airborne Contaminants: Methylene chloride (Volume 2) Nrc (1996)
National Toxicology Program (NTP), National Institutes of Health (NIH)	Report on Carcinogens, Twelfth Edition, Dichloromethane NIH (2016)
Occupational Safety and Health Administration (OSHA)	Occupational Exposure to Methylene Chloride OSHA (1997b)
California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (OEHHA)	Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride Oehha (2008a)
	Public Health Goal for Methylene Chloride in Drinking Water Oehha (2000)

Authoring Organization	Assessment
International	
Organisation for Economic Co-operation and Development (OECD), Cooperative Chemicals Assessment Program (CoCAP)	Dichloromethane: SIDS Initial Assessment Profile OECD (2011)
International Agency for Research on Cancer (IARC)	IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 110 IARC (2016)
World Health Organization (WHO)	Air Quality Guidelines for Europe WHO (2000)
WHO International Programme on Chemical Safety (IPCS)	Environmental Health Criteria 164 Methylene Chloride WHO (1996b)
Government of Canada, Environment Canada, Health Canada	Dichloromethane. Priority substances list assessment report. Health Canada (1993)
National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Australian Government	Human Health Tier II Assessment for Methane, dichloro- CAS Number: 75-09-2 NICNAS (2016)

1.4 Scope of the Evaluation

1.4.1 Conditions of Use Included in the Risk Evaluation

TSCA § 3(4) defines the conditions of use as “the circumstances, as determined by the Administrator, under which a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of.” Following the publication of the problem formulation, EPA finalized a rule that prohibits the manufacture (including import), processing and distribution of methylene chloride in all paint and coating removers for consumer use (40 CFR Part 751, Part B). EPA did not finalize any unreasonable risk determination for or regulate methylene chloride in commercial paint and coating removal as part of that rule; thus, this risk evaluation now includes commercial paint and coating remover uses (see Appendix L). This change is identified in Table 1-4, which identifies the conditions of use being evaluated, including those presented in the use document ([EPA-HQ-OPPT-2016-0742](#)), the life cycle diagram as presented in the problem formulation ([U.S. EPA, 2018c](#)), or received through public comment. The Problem Formulation also included uses such as metal products not covered elsewhere, apparel and footwear care products, and laundry and dishwashing products without distinguishing between industrial, commercial, and consumer uses. After additional review, no applicable consumer products were found for these uses. EPA has determined that there is no known, intended, or reasonably foreseen consumer use of these products. There are only industrial and commercial uses of methylene chloride for these conditions of use, and these conditions of use are assessed.

EPA has not exercised its authority in TSCA section 6(b)(4)(D) to exclude any methylene chloride conditions of use from the scope of the methylene chloride risk evaluation.

The life cycle diagram is presented below in Figure 1-1.

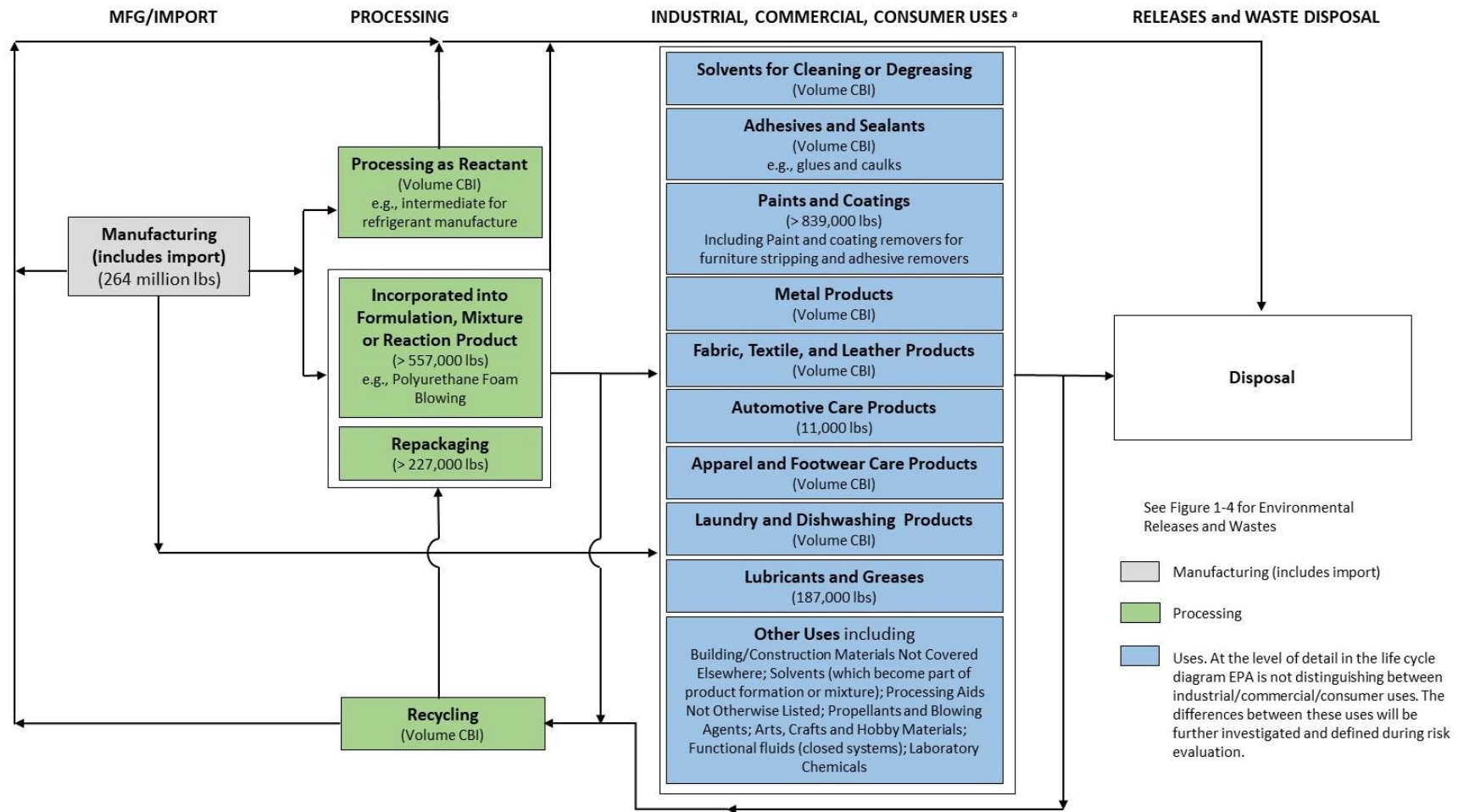


Figure 1-1. Methylene Chloride Life Cycle Diagram

The life cycle diagram depicts the conditions of use that are within the scope of the risk evaluation during various life cycle stages including manufacturing, processing, use (industrial, commercial, consumer), distribution and disposal. The production volumes shown are for reporting year 2015 from the 2016 CDR reporting period ([U.S. EPA, 2016](#)). Activities related to distribution (e.g., loading and unloading) are evaluated throughout the methylene chloride life cycle, rather than using a single distribution scenario.

^a See Table 1-4 for additional uses not mentioned specifically in this diagram.

Table 1-4. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	References
Manufacturing	Domestic manufacturing	Manufacturing	U.S. EPA (2016)
	Import	Import	U.S. EPA (2016)
Processing	Processing as a reactant	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants)	U.S. EPA (2016) ; U.S. EPA (2014) Market profile EPA-HQ-OPPT-2016-0742 Public Comments EPA-HQ-OPPT-2016-0742-0016 , EPA-HQ-OPPT-2016-0742-0017 , EPA-HQ-OPPT-2016-0742-0019
		Intermediate for pesticide, fertilizer, and other agricultural chemical manufacturing	U.S. EPA (2016)
		Petrochemical manufacturing*	U.S. EPA (2016)
		Intermediate for other chemicals	Public Comment EPA-HQ-OPPT-2016-0742-0008
	Incorporated into formulation, mixture, or reaction product	Solvents (for cleaning or degreasing), including manufacturing of: <ul style="list-style-type: none"> • All other basic organic chemical • Soap, cleaning compound and toilet preparation 	U.S. EPA (2016)
		Solvents (which become part of product formulation or mixture), including manufacturing of: <ul style="list-style-type: none"> • All other chemical product and preparation • Paints and coatings 	U.S. EPA (2016)

Life Cycle Stage	Category ^a	Subcategory ^b	References
		Propellants and blowing agents for all other chemical product and preparation manufacturing;	U.S. EPA (2016)
		Propellants and blowing agents for plastics product manufacturing	Use document EPA-HQ-OPPT-2016-0742-0003 , Market profile EPA-HQ-OPPT-2016-0742
		Paint additives and coating additives not described by other codes for CBI industrial sector*	U.S. EPA (2016)
		Laboratory chemicals for all other chemical product and preparation manufacturing	U.S. EPA (2016) , EPA-HQ-OPPT-2016-0742-0005 , EPA-HQ-OPPT-2016-0742-0014
		Laboratory chemicals for other industrial sectors*	U.S. EPA (2016)
		Processing aid, not otherwise listed for petrochemical manufacturing	U.S. EPA (2016)
		Adhesive and sealant chemicals in adhesive manufacturing	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016)
		oil and gas drilling, extraction, and support activities*	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016)
	Repackaging	Solvents (which become part of product formulation or mixture) for all other chemical product and preparation manufacturing	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016)
		all other chemical product and preparation manufacturing*	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016)
	Recycling	Recycling	U.S. EPA (2017e)

Life Cycle Stage	Category ^a	Subcategory ^b	References
Distribution in commerce	Distribution	Distribution	Use document EPA-HQ-OPPT-2016-0742-0003 U.S. EPA (2016)
Industrial, commercial and consumer uses	Solvents (for cleaning or degreasing) ^c	Batch vapor degreaser (e.g., open-top, closed-loop)	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016) ; Public comment EPA-HQ-OPPT-2016-0742-0017
		In-line vapor degreaser (e.g., conveyORIZED, web cleaner)	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016) ; Public comment EPA-HQ-OPPT-2016-0742-0017
		Cold cleaner	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016, 2014)
		Aerosol spray degreaser/cleaner	U.S. EPA (2016b, 2014b) EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742
	Adhesives and sealants	Single component glues and adhesives and sealants and caulks	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016) ; Public comments EPA-HQ-OPPT-2016-0742-0005 , EPA-HQ-OPPT-2016-0742-0013 , EPA-HQ-OPPT-2016-0742-0014 , EPA-HQ-OPPT-2016-0742-0017 , EPA-HQ-OPPT-2016-0742-0021 , EPA-HQ-OPPT-2016-0742-0033

Life Cycle Stage	Category ^a	Subcategory ^b	References
	Paints and coatings including commercial paint and coating removers ^c	Paints and coatings use and commercial paints and coating removers	U.S. EPA (2016b, 2014b) ; Market profile EPA-HQ-OPPT-2016-0742 Public Comments EPA-HQ-OPPT-2016-0742-0005 , EPA-HQ-OPPT-2016-0742-0009 , EPA-HQ-OPPT-2016-0742-0014 , EPA-HQ-OPPT-2016-0742-0017 , EPA-HQ-OPPT-2016-0742-0021 , EPA-HQ-OPPT-2016-0742-0025
		Adhesive/caulk removers	Use document EPA-HQ-OPPT-2016-0742-0003 , Market profile EPA-HQ-OPPT-2016-0742
	Metal products not covered elsewhere	Degreasers – aerosol and non-aerosol degreasers and cleaners (e.g., coil cleaners)	Market profile EPA-HQ-OPPT-2016-0742 U.S. EPA (2016)
	Fabric, textile and leather products not covered elsewhere	Textile finishing and impregnating/surface treatment products (e.g., water repellent)	Market profile EPA-HQ-OPPT-2016-0742
	Automotive care products	Function fluids for air conditioners: refrigerant, treatment, leak sealer	Use document EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 , U.S. EPA (2016)
		Interior car care – spot remover	Use document EPA-HQ-OPPT-2016-0742-0003
		Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Use document EPA-HQ-OPPT-2016-0742-0003 , Market profile EPA-HQ-OPPT-2016-0742 , U.S. EPA (2016)

Life Cycle Stage	Category ^a	Subcategory ^b	References
	Apparel and footwear care products	Post-market waxes and polishes applied to footwear (e.g., shoe polish)	Market profile EPA-HQ-OPPT-2016-0742
	Laundry and dishwashing products	Spot remover for apparel and textiles	Use document EPA-HQ-OPPT-2016-0742-0003
	Lubricants and greases	Liquid and spray lubricants and greases	U.S. EPA (2016) ; EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; Public Comment EPA-HQ-OPPT-2016-0742-0021
		Degreasers – aerosol and non-aerosol degreasers and cleaners	U.S. EPA (2016) ; EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; Public Comments EPA-HQ-OPPT-2016-0742-0005 , EPA-HQ-OPPT-2016-0742-0014
	Building/ construction materials not covered elsewhere	Cold pipe insulation	Use document EPA-HQ-OPPT-2016-0742-0003
	Solvents (which become part of product formulation or mixture)	All other chemical product and preparation manufacturing	U.S. EPA (2016)
	Processing aid not otherwise listed	In multiple manufacturing sectors ^d	Use document EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; U.S. EPA (2016)
	Propellants and blowing agents	Flexible polyurethane foam manufacturing	Market profile EPA-HQ-OPPT-2016-0742

Life Cycle Stage	Category ^a	Subcategory ^b	References
	Arts, crafts and hobby materials	Crafting glue and cement/concrete	Use document EPA-HQ-OPPT-2016-0742-0003
	Other Uses	Laboratory chemicals - all other chemical product and preparation manufacturing	Use document EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; Public Comment: EPA-HQ-OPPT-2016-0742-0066
		Electrical equipment, appliance, and component manufacturing	U.S. EPA (2016) , Public Comment EPA-HQ-OPPT-2016-0742-0017
		Plastic and rubber products	U.S. EPA (2016)
		Anti-adhesive agent - anti-spatter welding aerosol	Use document EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; Public Comment EPA-HQ-OPPT-2016-0742-0005
		Oil and gas drilling, extraction, and support activities	Use document EPA-HQ-OPPT-2016-0742-0003 ; U.S. EPA (2016)
		Toys, playground, and sporting equipment - including novelty articles (toys, gifts, etc.)	Use document EPA-HQ-OPPT-2016-0742-0003 ; EPA-HQ-OPPT-2016-0742-0069 ;
		Carbon remover, lithographic printing cleaner, brush cleaner, use in taxidermy, and wood floor cleaner	Use document EPA-HQ-OPPT-2016-0742-0003 ; Market profile EPA-HQ-OPPT-2016-0742 ; U.S. EPA (2016)
Disposal	Disposal	Industrial pre-treatment	U.S. EPA (2017e)
		Industrial wastewater treatment	
		Publicly owned treatment works (POTW)	
		Underground injection	

Life Cycle Stage	Category ^a	Subcategory ^b	References
		Municipal landfill	
		Hazardous landfill	
		Other land disposal	
		Municipal waste incinerator	
		Hazardous waste incinerator	
		Off-site waste transfer	
<p>Note that methylene chloride is used by federal agencies in a variety of uses, including some deemed mission critical.</p> <p>^a These categories of conditions of use appear in the initial life cycle diagram, reflect CDR codes and broadly represent conditions of use for methylene chloride in industrial and/or commercial settings.</p> <p>^b These subcategories reflect more specific uses of methylene chloride.</p> <p>^c Reported for the following sectors in the 2016 CDR for manufacturing of: plastic materials and resins, plastics products, miscellaneous, all other chemical product and preparation (U.S. EPA, 2016).</p> <p>^d Reported for the following sectors in the 2016 CDR for manufacturing of: petrochemicals, plastic materials and resins, plastics products, miscellaneous and all other chemical products * (U.S. EPA, 2016) also including as a chemical processor for polycarbonate resins and cellulose triacetate (photographic film).</p> <p>^e Consumer paint and coating remover uses are already addressed through rulemaking (see 40 CFR Part 751, Subpart B) and are outside the scope of this risk evaluation.</p> <p>* Conditions of use with CBI or unknown function were evaluated and considered for the methylene chloride risk evaluation; however, the non-CBI elements of the category, subcategory, function and industrial sector were used in the analysis as these data were higher quality. This applies to: CBI function for petrochemical manufacturing, paint additives and coating additives not described by other codes for CBI industrial sector, laboratory chemicals for CBI industrial sectors, manufacturing of CBI and oil and gas drilling, extraction, and support activities.</p> <p>** Although EPA has identified both industrial and commercial uses here for purposes of distinguishing scenarios in this document, the Agency interprets the authority over “any manner or method of commercial use” under TSCA section 6(a)(5) to reach both.</p>			

1.4.2 Exposure Pathways and Risks Addressed by Other EPA-Administered Statutes⁵

In its TSCA section 6(b) risk evaluations, EPA is coordinating action on certain exposure pathways and risks falling under the jurisdiction of other EPA-administered statutes or regulatory programs. More specifically, EPA is exercising its TSCA authorities to tailor the scope of its risk evaluations, rather than focusing on environmental exposure pathways addressed under other EPA-administered statutes or regulatory programs or risks that could be eliminated or reduced to a sufficient extent by actions taken under other EPA-administered laws. EPA considers this approach to be a reasonable exercise of the Agency's TSCA authorities, which include:

- TSCA section 6(b)(4)(D): “The Administrator shall, not later than 6 months after the initiation of a risk evaluation, publish the scope of the risk evaluation to be conducted, including the hazards, exposures, conditions of use, and the potentially exposed or susceptible subpopulations the Administrator expects to consider...”
- TSCA section 9(b)(1): “The Administrator shall coordinate actions taken under this chapter with actions taken under other Federal laws administered in whole or in part by the Administrator. If the Administrator determines that a risk to health or the environment associated with a chemical substance or mixture could be eliminated or reduced to a sufficient extent by actions taken under the authorities contained in such other Federal laws, the Administrator shall use such authorities to protect against such risk unless the Administrator determines, in the Administrator's discretion, that it is in the public interest to protect against such risk by actions taken under this chapter.”
- TSCA section 9(e): “...[I]f the Administrator obtains information related to exposures or releases of a chemical substance or mixture that may be prevented or reduced under another Federal law, including a law not administered by the Administrator, the Administrator shall make such information available to the relevant Federal agency or office of the Environmental Protection Agency.”
- TSCA section 2(c): “It is the intent of Congress that the Administrator shall carry out this chapter in a reasonable and prudent manner, and that the Administrator shall consider the environmental, economic, and social impact of any action the Administrator takes or proposes as provided under this chapter.”
- TSCA section 18(d)(1): “Nothing in this chapter, nor any amendment made by the Frank R. Lautenberg Chemical Safety for the 21st Century Act, nor any rule, standard of performance, risk evaluation, or scientific assessment implemented pursuant to this chapter, shall affect the right of a State or a political subdivision of a State to adopt or enforce any rule, standard of performance, risk evaluation, scientific assessment, or any other protection for public health or the environment that— (i) is adopted or authorized under the authority of any other Federal law or adopted to satisfy or obtain authorization or approval under any other Federal law...”

TSCA authorities supporting tailored risk evaluations and intra-agency referrals

⁵ The statutory interpretations and approach described in this subsection will apply to all TSCA risk evaluations and are not limited in application to this final risk evaluation for methylene chloride.

TSCA section 6(b)(4)(D)

TSCA section 6(b)(4)(D) requires EPA, in developing the scope of a risk evaluation, to identify the hazards, exposures, conditions of use, and potentially exposed or susceptible subpopulations the Agency “expects to consider” in a risk evaluation. This language suggests that EPA is not required to consider all conditions of use, hazards, or exposure pathways in risk evaluations. As EPA explained in the “Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act” (“Risk Evaluation Rule”), “EPA may, on a case-by-case basis, exclude certain activities that EPA has determined to be conditions of use in order to focus its analytical efforts on those exposures that are likely to present the greatest concern, and consequently merit an unreasonable risk determination.” 82 FR 33726, 33729 (July 20, 2017).

In the problem formulation documents for many of the first 10 chemicals undergoing risk evaluation, EPA applied the same authority and rationale to certain exposure pathways, explaining that “EPA is planning to exercise its discretion under TSCA 6(b)(4)(D) to focus its analytical efforts on exposures that are likely to present the greatest concern and consequently merit a risk evaluation under TSCA, by excluding, on a case-by-case basis, certain exposure pathways that fall under the jurisdiction of other EPA-administered statutes.” The approach discussed in the Risk Evaluation Rule and applied in the problem formulation documents is informed by the legislative history of the amended TSCA, which supports the Agency’s exercise of discretion to focus the risk evaluation on areas that raise the greatest potential for risk. See June 7, 2016 Cong. Rec., S3519-S3520. Consistent with the approach articulated in the problem formulation documents, and as described in more detail below, EPA is exercising its authority under TSCA to tailor the scope of exposures evaluated in TSCA risk evaluations, rather than focusing on environmental exposure pathways addressed under other EPA-administered, media-specific statutes and regulatory programs.

TSCA section 9(b)(1)

In addition to TSCA section 6(b)(4)(D), the Agency also has discretionary authority under the first sentence of TSCA section 9(b)(1) to “coordinate actions taken under [TSCA] with actions taken under other Federal laws administered in whole or in part by the Administrator.” This broad, freestanding authority provides for intra-agency coordination and cooperation on a range of “actions.” In EPA’s view, the phrase “actions taken under [TSCA]” in the first sentence of section 9(b)(1) is reasonably read to encompass more than just risk management actions, and to include actions taken during risk evaluation as well. More specifically, the authority to coordinate intra-agency actions exists regardless of whether the Administrator has first made a definitive finding of risk, formally determined that such risk could be eliminated or reduced to a sufficient extent by actions taken under authorities in other EPA-administered Federal laws, and/or made any associated finding as to whether it is in the public interest to protect against such risk by actions taken under TSCA. TSCA section 9(b)(1) therefore provides EPA authority to coordinate actions with other EPA offices without ever making a risk finding, or following an identification of risk. This includes coordination on tailoring the scope of TSCA risk evaluations to focus on areas of greatest concern rather than exposure pathways addressed by other EPA-

administered statutes and regulatory programs, which does not involve a risk determination or public interest finding under TSCA section 9(b)(2).

In a narrower application of the broad authority provided by the first sentence of TSCA section 9(b)(1), the remaining provisions of section 9(b)(1) provide EPA authority to identify risks and refer certain of those risks for action by other EPA offices. Under the second sentence of section 9(b)(1), “[i]f the Administrator determines that a risk to health or the environment associated with a chemical substance or mixture could be eliminated or reduced to a sufficient extent by actions taken under the authorities contained in such other Federal laws, the Administrator shall use such authorities to protect against such risk unless the Administrator determines, in the Administrator’s discretion, that it is in the public interest to protect against such risk by actions taken under [TSCA].” Coordination of intra-agency action on risks under TSCA section 9(b)(1) therefore entails both an identification of risk, and a referral of any risk that could be eliminated or reduced to a sufficient extent under other EPA-administered laws to the EPA office(s) responsible for implementing those laws (absent a finding that it is in the public interest to protect against the risk by actions taken under TSCA).

Risk may be identified by OPPT or another EPA office, and the form of the identification may vary. For instance, OPPT may find that one or more conditions of use for a chemical substance present(s) a risk to human or ecological receptors through specific exposure routes and/or pathways. This could involve a quantitative or qualitative assessment of risk based on reasonably available information (which might include, e.g., findings or statements by other EPA offices or other federal agencies). Alternatively, risk could be identified by another EPA office. For example, another EPA office administering non-TSCA authorities may have sufficient monitoring or modeling data to indicate that a particular condition of use presents risk to certain human or ecological receptors, based on expected hazards and exposures. This risk finding could be informed by information made available to the relevant office under TSCA section 9(e), which supports cooperative actions through coordinated information-sharing.

Following an identification of risk, EPA would determine if that risk could be eliminated or reduced to a sufficient extent by actions taken under authorities in other EPA-administered laws. If so, TSCA requires EPA to “use such authorities to protect against such risk,” unless EPA determines that it is in the public interest to protect against that risk by actions taken under TSCA. In some instances, EPA may find that a risk could be sufficiently reduced or eliminated by future action taken under non-TSCA authority. This might include, e.g., action taken under the authority of the Safe Drinking Water Act to address risk to the general population from a chemical substance in drinking water, particularly if the Office of Water has taken preliminary steps such as listing the subject chemical substance on the Contaminant Candidate List. This sort of risk finding and referral could occur during the risk evaluation process, thereby enabling EPA to use more a relevant and appropriate authority administered by another EPA office to protect against hazards or exposures to affected receptors.

Legislative history on TSCA section 9(b)(1) supports both broad coordination on current intra-agency actions, and narrower coordination when risk is identified and referred to another EPA office for action. A Conference Report from the time of TSCA’s passage explained that section 9 is intended “to assure that overlapping or duplicative regulation is avoided while attempting to

provide for the greatest possible measure of protection to health and the environment.” S. Rep. No. 94-1302 at 84. See also H. Rep. No. 114-176 at 28 (stating that the 2016 TSCA amendments “reinforce TSCA’s original purpose of filling gaps in Federal law,” and citing new language in section 9(b)(2) intended “to focus the Administrator’s exercise of discretion regarding which statute to apply and to encourage decisions that avoid confusion, complication, and duplication”). Exercising TSCA section 9(b)(1) authority to coordinate on tailoring TSCA risk evaluations is consistent with this expression of Congressional intent.

Legislative history also supports a reading of section 9(b)(1) under which EPA coordinates intra-agency action, including information-sharing under TSCA section 9(e), and the appropriately-positioned EPA office is responsible for the identification of risk and actions to protect against such risks. See, e.g., Senate Report 114-67, 2016 Cong. Rec. S3522 (under TSCA section 9, “if the Administrator finds that disposal of a chemical substance may pose risks that could be prevented or reduced under the Solid Waste Disposal Act, the Administrator should ensure that the relevant office of the EPA receives that information”); H. Rep. No. 114-176 at 28, 2016 Cong. Rec. S3522 (under section 9, “if the Administrator determines that a risk to health or the environment associated with disposal of a chemical substance could be eliminated or reduced to a sufficient extent under the Solid Waste Disposal Act, the Administrator should use those authorities to protect against the risk”). Legislative history on section 9(b)(1) therefore supports coordination with and referral of action to other EPA offices, especially when statutes and associated regulatory programs administered by those offices could address exposure pathways or risks associated with conditions of use, hazards, and/or exposure pathways that may otherwise be within the scope of TSCA risk evaluations.

TSCA sections 2(c) & 18(d)(1)

Finally, TSCA sections 2(c) and 18(d) support coordinated action on exposure pathways and risks addressed by other EPA-administered statutes and regulatory programs. Section 2(c) directs EPA to carry out TSCA in a “reasonable and prudent manner” and to consider “the environmental, economic, and social impact” of its actions under TSCA. Legislative history from around the time of TSCA’s passage indicates that Congress intended EPA to consider the context and take into account the impacts of each action under TSCA. S. Rep. No. 94-698 at 14 (“the intent of Congress as stated in this subsection should guide each action the Administrator takes under other sections of the bill”).

Section 18(d)(1) specifies that state actions adopted or authorized under any Federal law are not preempted by an order of no unreasonable risk issued pursuant to TSCA section 6(i)(1) or a rule to address unreasonable risk issued under TSCA section 6(a). Thus, even if a risk evaluation were to address exposures or risks that are otherwise addressed by other federal laws and, for example, implemented by states, the state laws implementing those federal requirements would not be preempted. In such a case, both the other federal and state laws, as well as any TSCA section 6(i)(1) order or TSCA section 6(a) rule, would apply to the same issue area. See also TSCA section 18(d)(1)(A)(iii). In legislative history on amended TSCA pertaining to section 18(d), Congress opined that “[t]his approach is appropriate for the considerable body of law regulating chemical releases to the environment, such as air and water quality, where the states

have traditionally had a significant regulatory role and often have a uniquely local concern.”
Sen. Rep. 114-67 at 26.

EPA’s careful consideration of whether other EPA-administered authorities are available and more appropriate for addressing certain exposures and risks is consistent with Congress’ intent to maintain existing federal requirements and the state actions adopted to locally and more specifically implement those federal requirements, and to carry out TSCA in a reasonable and prudent manner. EPA believes it is both reasonable and prudent to tailor TSCA risk evaluations in a manner reflective of expertise and experience exercised by other EPA and State offices to address specific environmental media, rather than attempt to evaluate and regulate potential exposures and risks from those media under TSCA. This approach furthers Congressional direction and EPA aims to efficiently use Agency resources, avoid duplicating efforts taken pursuant to other Agency and State programs, and meet the statutory deadline for completing risk evaluations.

EPA-administered statutes and regulatory programs that address specific exposure pathways and/or risks

During the course of the risk evaluation process for methylene chloride, OPPT worked closely with the offices within EPA that administer and implement regulatory programs under the Clean Air Act (CAA), the Safe Drinking Water Act (SDWA), the Clean Water Act (CWA) and the Resource Conservation and Recovery Act (RCRA). Through intra-agency coordination, EPA determined that specific exposure pathways are well-regulated by the EPA statutes and regulations described in the following paragraphs.

The CAA contains a list of hazardous air pollutants (HAP) and provides EPA with the authority to add to that list pollutants that present, or may present, a threat of adverse human health effects or adverse environmental effects. For stationary source categories emitting HAP, the CAA requires issuance of technology-based standards and, if necessary, additions or revisions to address developments in practices, processes, and control technologies, and to ensure the standards adequately protect public health and the environment. The CAA thereby provides EPA with comprehensive authority to regulate emissions to ambient air of any hazardous air pollutant.

Methylene Chloride is a HAP. See 42 U.S.C. 7412. EPA has issued a number of technology-based standards for source categories that emit methylene chloride to ambient air and, as appropriate, has reviewed, or is in the process of reviewing remaining risks. See 40 CFR part 63; Appendix A. Because stationary source releases of methylene chloride to ambient air are addressed under the CAA, EPA is not evaluating emissions to ambient air from commercial and industrial stationary sources or associated inhalation exposure of the general population or terrestrial species in this TSCA risk evaluation.

EPA has regular analytical processes to identify and evaluate drinking water contaminants of potential regulatory concern for public water systems under the Safe Drinking Water Act (SDWA). Under SDWA, EPA must also review and revise “as appropriate” existing drinking water regulations every 6 years.

EPA has promulgated National Primary Drinking Water Regulations (NPDWRs) for methylene chloride under SDWA. See 40 CFR part 151; Appendix A. EPA has set an enforceable Maximum Contaminant Level (MCL) as close as feasible to a health based, non-enforceable Maximum Contaminant Level Goal (MCLG). Feasibility refers to both the ability to treat water to meet the MCL and the ability to monitor water quality at the MCL, SDWA Section 1412(b)(4)(D), and public water systems are required to monitor for the regulated chemical based on a standardized monitoring schedule to ensure compliance with the maximum contaminant level (MCL).

Hence, because the drinking water exposure pathway for methylene chloride is currently addressed in the SDWA regulatory analytical process for public water systems, EPA is not evaluating exposures to the general population from the drinking water exposure pathway in the risk evaluation for methylene chloride under TSCA.

EPA develops recommended water quality criteria under section 304(a) of the CWA for pollutants in surface water that are protective of aquatic life or human health designated uses. EPA develops and publishes water quality criteria based on priorities of states and others that reflect the latest scientific knowledge. A subset of these chemicals are identified as “priority pollutants” (103 human health and 27 aquatic life). The CWA requires states adopt numeric criteria for priority pollutants for which EPA has published recommended criteria under section 304(a), the discharge or presence of which in the affected waters could reasonably be expected to interfere with designated uses adopted by the state. When states adopt criteria that EPA approves as part of state’s regulatory water quality standards, exposure is considered when state permit writers determine if permit limits are needed and at what level for a specific discharger of a pollutant to ensure protection of the designated uses of the receiving water. Once states adopt criteria as water quality standards, the CWA requires that National Pollutant Discharge Elimination System (NPDES) discharge permits include effluent limits as stringent as necessary to meet standards. CWA section 301(b)(1)(C). This is the process used under the CWA to address risk to human health and aquatic life from exposure to a pollutant in ambient waters.

EPA has identified methylene chloride as a priority pollutant and has developed recommended water quality criteria for protection of human health for methylene chloride which are available for adoption into state water quality standards for the protection of human health and are available for use by NPDES permitting authorities in deriving effluent limits to meet state criteria.⁶ See, e.g., 40 CFR part 423, Appendix A; 40 CFR 131.11(b)(1); 40 CFR 122.44(d)(vi). As such, EPA is not evaluating exposures to the general population from the surface water exposure pathway in the risk evaluation under TSCA.

Methylene chloride is included on the list of hazardous wastes pursuant to RCRA section 3001 (40 CFR §§ 261.33) as a listed waste on the F001, F002, K009, K010, K156, K157, K158, and U080 lists. The general standard in RCRA section 3004(a) for the technical criteria that govern the management (treatment, storage, and disposal) of hazardous waste are those “necessary to protect human health and the environment,” RCRA 3004(a). The regulatory criteria for identifying “characteristic” hazardous wastes and for “listing” a waste as hazardous also relate solely to the potential risks to human health or the environment. 40 C.F.R. §§ 261.11, 261.21-

⁶ See <https://www.regulations.gov/document?D=EPA-HQ-OW-2014-0135-0200>.

261.24. RCRA statutory criteria for identifying hazardous wastes require EPA to “tak[e] into account toxicity, persistence, and degradability in nature, potential for accumulation in tissue, and other related factors such as flammability, corrosiveness, and other hazardous characteristics.” Subtitle C controls cover not only hazardous wastes that are landfilled, but also hazardous wastes that are incinerated (subject to joint control under RCRA Subtitle C and the CAA hazardous waste combustion MACT) or injected into UIC Class I hazardous waste wells (subject to joint control under Subtitle C and SDWA).

EPA is not evaluating emissions to ambient air from municipal and industrial waste incineration and energy recovery units or associated exposures to the general population or terrestrial species in the risk evaluation, as these emissions are regulated under section 129 of the Clean Air Act. CAA section 129 requires EPA to review and, if necessary, add provisions to ensure the standards adequately protect public health and the environment. Thus, combustion by-products from incineration treatment of methylene chloride wastes would be subject to these regulations, as would methylene chloride burned for energy recovery. See 40 CFR part 60.

EPA is not evaluating on-site releases to land that go to underground injection or associated exposures to the general population or terrestrial species in its risk evaluation. Environmental disposal of methylene chloride injected into Class I hazardous well types are covered under the jurisdiction of RCRA and SDWA and disposal of methylene chloride via underground injection is not likely to result in environmental and general population exposures. See 40 CFR part 144.

EPA is not evaluating on-site releases to land from RCRA Subtitle C hazardous waste landfills or exposures of the general population or terrestrial species from such releases in the TSCA evaluation. Design standards for Subtitle C landfills require double liner, double leachate collection and removal systems, leak detection system, run on, runoff, and wind dispersal controls, and a construction quality assurance program. They are also subject to closure and post-closure care requirements including installing and maintaining a final cover, continuing operation of the leachate collection and removal system until leachate is no longer detected, maintaining and monitoring the leak detection and groundwater monitoring system. Bulk liquids may not be disposed in Subtitle C landfills. Subtitle C landfill operators are required to implement an analysis and testing program to ensure adequate knowledge of waste being managed, and to train personnel on routine and emergency operations at the facility. Hazardous waste being disposed in Subtitle C landfills must also meet RCRA waste treatment standards before disposal. See 40 CFR part 264; Appendix A.

EPA is not evaluating on-site releases to land from RCRA Subtitle D municipal solid waste (MSW) landfills or exposures of the general population or terrestrial species from such releases in the TSCA evaluation. While permitted and managed by the individual states, municipal solid waste landfills are required by federal regulations to implement some of the same requirements as Subtitle C landfills. MSW landfills generally must have a liner system with leachate collection and conduct groundwater monitoring and corrective action when releases are detected. MSW landfills are also subject to closure and post-closure care requirements, and must have financial assurance for funding of any needed corrective actions. MSW landfills have also been designed to allow for the small amounts of hazardous waste generated by households and very small quantity waste generators (less than 220 lbs per month). Bulk liquids, such as free solvent, may not be disposed of at MSW landfills. See 40 CFR part 258.

EPA is not evaluating on-site releases to land from industrial non-hazardous waste and construction/demolition waste landfills or associated exposures to the general population or terrestrial species in the methylene chloride risk evaluation. Industrial non-hazardous and construction/demolition waste landfills are primarily regulated under authorized state regulatory programs. States must also implement limited federal regulatory requirements for siting, groundwater monitoring and corrective action and a prohibition on open dumping and disposal of bulk liquids. States may also establish additional requirements such as for liners, post-closure and financial assurance, but are not required to do so. See, e.g., RCRA section 3004(c), 4007; 40 CFR part 257.

1.4.3 Conceptual Models

The conceptual model in Figure 1-2 presents the exposure pathways, exposure routes and hazards to human receptors from industrial and commercial activities and uses of methylene chloride.

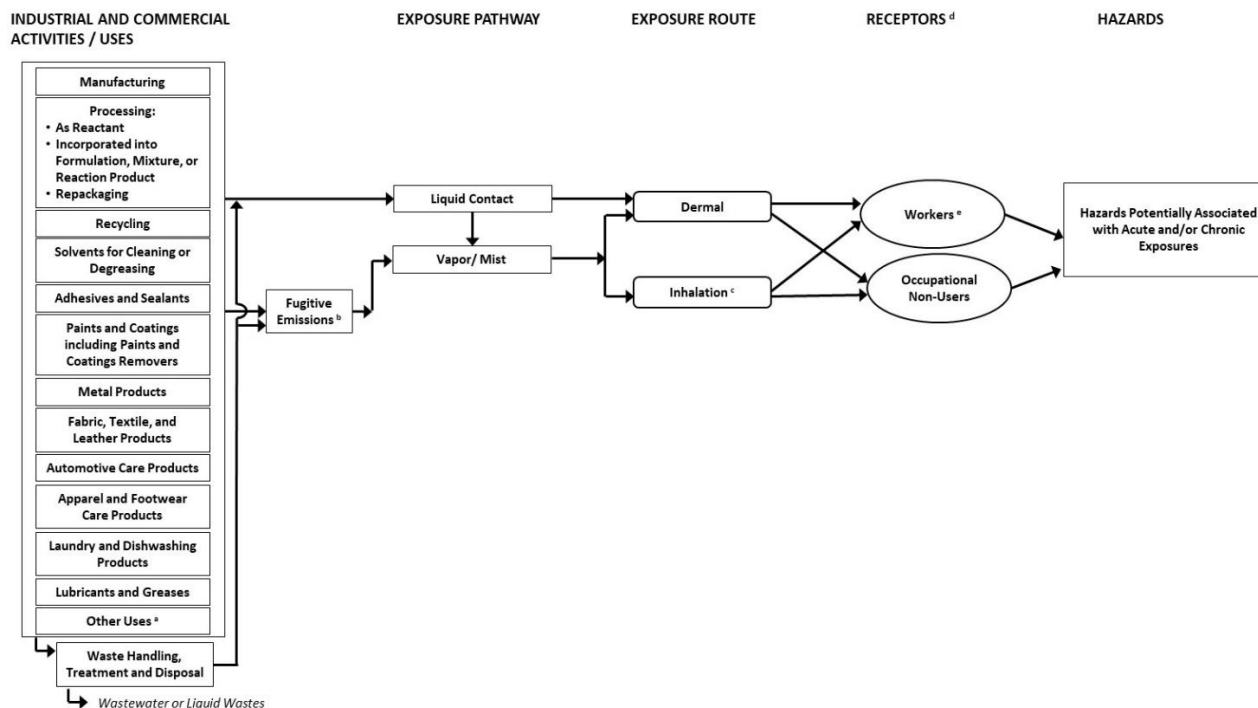


Figure 1-2. Methylene Chloride Conceptual Model for Industrial and Commercial Activities and Uses: Potential Exposure and Hazards

^a Some products are used in both commercial and consumer applications such as adhesives and sealants. Additional uses of methylene chloride are included in Table 1-4.

^b Fugitive air emissions are those that are not stack emissions and include fugitive equipment leaks from valves, pump seals, flanges, compressors, sampling connections and open-ended lines; evaporative losses from surface impoundment and spills; and releases from building ventilation systems.

^c Exposure may occur through mists that deposit in the upper respiratory tract. However, based on physical chemical properties, mists of methylene chloride will likely be rapidly absorbed in the respiratory tract or evaporate, and were evaluated as an inhalation exposure.

^d Receptors include PESS.

^e When data and information were available to support the analysis, EPA also considered the effect that engineering controls and/or personal protective equipment (PPE) have on occupational exposure levels.

The conceptual model in Figure 1-3 presents the exposure pathways, exposure routes and hazards to human receptors from consumer activities and uses of methylene chloride.

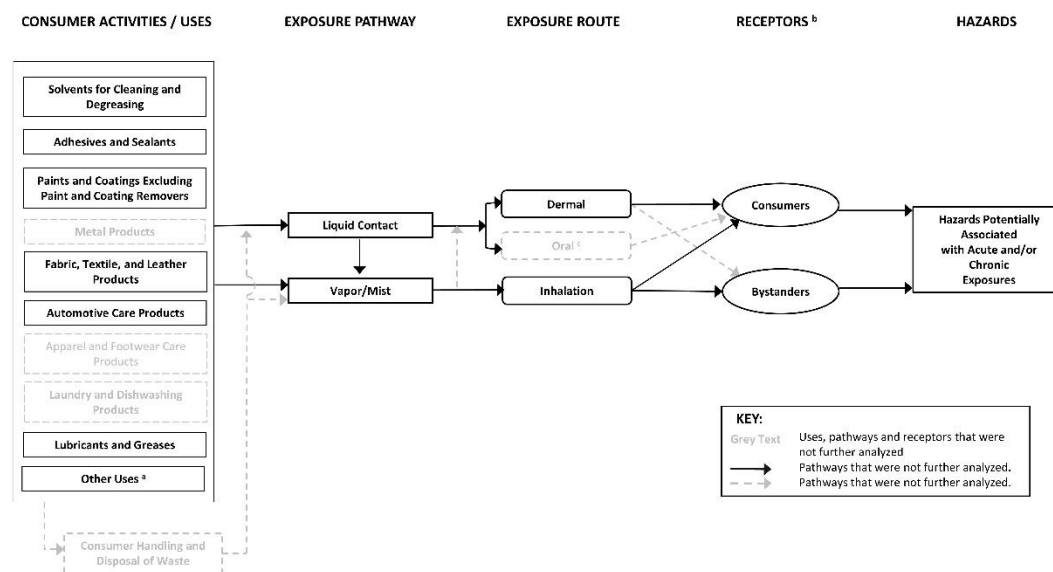


Figure 1-3. Methylene Chloride Conceptual Model for Consumer Activities and Uses: Potential Exposure and Hazards

^a Some products are used in both commercial and consumer applications. Additional uses of methylene chloride are included in Table 1-4.

^b Receptors include PESS.

^c Exposure may occur through mists that deposit in the upper respiratory tract or via transfer of methylene chloride from hand to mouth. However, this exposure pathway will be limited by a combination of rapid absorption and/or evaporation that will not result in oral exposure. Therefore, this pathway will not be further evaluated.

The conceptual model in Figure 1-4 presents the exposure pathways, exposure routes and hazards to human and environmental receptors from environmental releases and wastes of methylene chloride.

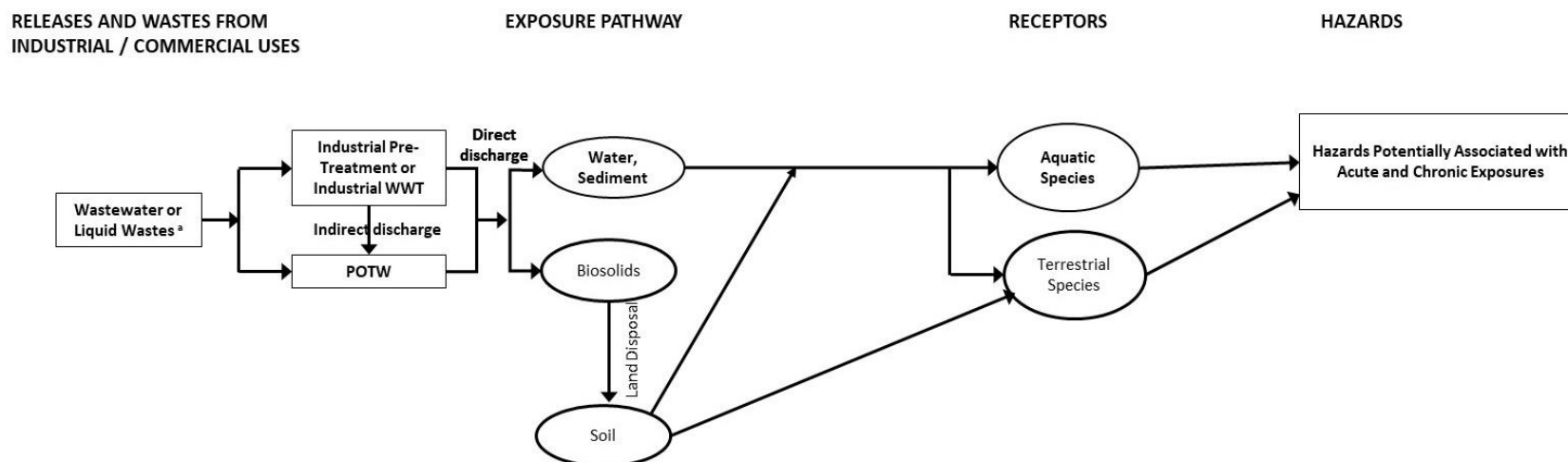


Figure 1-4. Methylene Chloride Conceptual Model for Environmental Releases and Wastes: Potential Exposures and Hazards

^a Industrial wastewater may be treated on-site and then released to surface water (direct discharge), or pre-treated and released to POTW (indirect discharge).

1.5 Systematic Review

TSCA requires EPA to use scientific information, technical procedures, measures, methods, protocols, methodologies and models consistent with the best available science when making science-based decisions under Section 6 and base decisions under Section 6 on the weight of scientific evidence. Within the TSCA risk evaluation context, the weight of the scientific evidence is defined as “*a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a pre-established protocol to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance*” (40 CFR 702.33).

To meet the TSCA § 26(h) science standards, EPA used the TSCA systematic review process described in the *Application of Systematic Review in TSCA Risk Evaluations* document ([U.S. EPA, 2018b](#)). The process complements the risk evaluation process in that the data collection, data evaluation and data integration stages of the systematic review process are used to develop the exposure and hazard assessments based on reasonably available information. EPA defines “reasonably available information” to mean information that EPA possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines for completing the evaluation (40 CFR 702.33).

EPA is implementing systematic review methods and approaches within the regulatory context of the amended TSCA. Although EPA adopted as many best practices as practicable from the systematic review community, EPA modified the process to ensure that the identification, screening, evaluation and integration of data and information can support timely regulatory decision making under the timelines of the statute.

1.5.1 Data and Information Collection

EPA planned and conducted a comprehensive literature search based on key words related to the different discipline-specific evidence supporting the risk evaluation (e.g., environmental fate and transport; environmental releases and occupational exposure; exposure to general population, consumers and environmental exposure; and environmental and human health hazard). EPA then developed and applied inclusion and exclusion criteria during the title/abstract screening to identify information potentially relevant for the risk evaluation process. The literature and screening strategy as specifically applied to methylene chloride is described in *Strategy for Conducting Literature Searches for Methylene Chloride (DCM): Supplemental File to the TSCA Scope Document* ([U.S. EPA, 2017d](#)) and the results of the title and abstract screening process were published in *Methylene Chloride (DCM) (CASRN: 75-09-2) Bibliography: Supplemental File for the TSCA Scope Document* ([U.S. EPA, 2017a](#)).

For studies determined to be on-topic (or relevant) after title and abstract screening, EPA conducted a full text screening to further exclude references that were not relevant to the risk evaluation. Screening decisions were made based on eligibility criteria documented in the form of the populations, exposures, comparators, and outcomes (PECO) framework or a modified

framework⁷. Data sources that met the criteria were carried forward to the data evaluation stage. The inclusion and exclusion criteria for full text screening for methylene chloride are available in Appendix F of *Problem Formulation of the Risk Evaluation for Methylene Chloride (Dichloromethane, DCM)* ([U.S. EPA, 2018c](#)).

In addition to the comprehensive search and screening process conducted as described above, EPA made the decision to leverage the literature published in previous assessments⁸ to identify key and supporting data⁹ and information for developing the methylene chloride risk evaluation. This is discussed in *Strategy for Conducting Literature Searches for Methylene Chloride (DCM): Supplemental File to the TSCA Scope Document* ([U.S. EPA, 2017d](#)). In general, many of the key and supporting data sources were identified in the comprehensive *Methylene Chloride (DCM) (CASRN: 75-09-2) Bibliography: Supplemental File for the TSCA Scope Document* ([U.S. EPA, 2017a](#)). However, there was an instance during the releases and occupational exposure data search for which EPA missed relevant references that were not captured in the initial categorization of the on-topic references. EPA found additional relevant data and information using backward reference searching, which was a technique that will be included in future search strategies. This issue is discussed in Section 4 of *Application of Systematic Review for TSCA Risk Evaluations* ([U.S. EPA, 2018b](#)). Other relevant key and supporting references were identified through targeted supplemental searches to support the analytical approaches and methods in the methylene chloride risk evaluation (e.g., to locate specific information for exposure modeling).

EPA used previous chemical assessments to quickly identify relevant key and supporting information as a pragmatic approach to expedite the quality evaluation of the data sources, but many of those data sources were already captured in the comprehensive literature as explained above. EPA also considered newer information not taken into account by previous chemical assessments as described in *Strategy for Conducting Literature Searches for Methylene Chloride (DCM): Supplemental File to the TSCA Scope Document* ([U.S. EPA, 2017d](#)). EPA then evaluated the confidence of the key and supporting data sources as well as newer information instead of evaluating the confidence of all the underlying evidence ever published on a chemical substance's fate and transport, environmental releases, environmental and human exposure and hazards. Such comprehensive evaluation of all of the data and information ever published for a chemical substance would be extremely labor intensive and could not be achieved under the TSCA statutory deadlines for most chemical substances especially those that have a data-rich database. Furthermore, EPA considered how evaluation of newer information in addition to the key and supporting data and information would change the conclusions presented in previous assessments.

⁷ A PESO statement was used during the full text screening of environmental fate and transport data sources. PESO stands for Pathways and Processes, Exposure, Setting or Scenario, and Outcomes. A RESO statement was used during the full text screening of the engineering and occupational exposure literature. RESO stands for Receptors, Exposure, Setting or Scenario, and Outcomes.

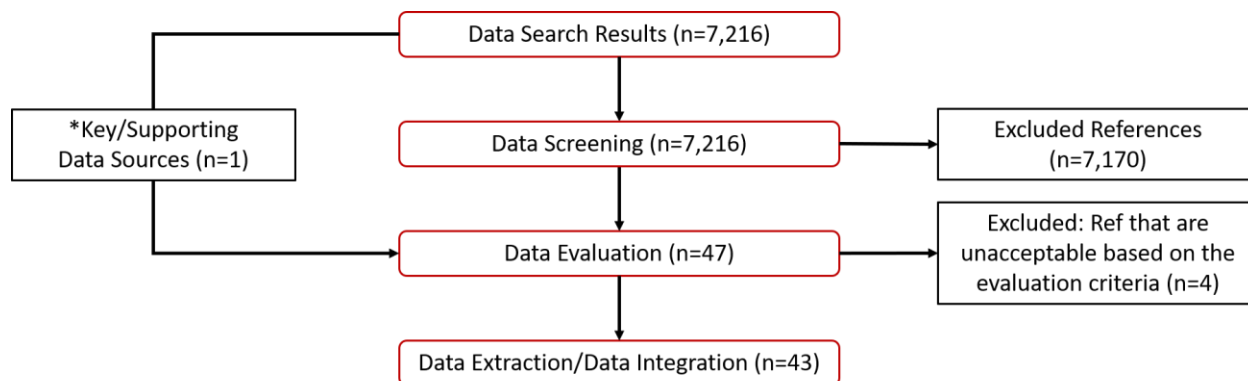
⁸ Examples of existing assessments are EPA's chemical assessments (e.g., previous work plan risk assessments, problem formulation documents), ATSDR's Toxicological Profiles and EPA's IRIS assessments. This is described in more detail in *Strategy for Conducting Literature Searches for Methylene Chloride (DCM): Supplemental File to the TSCA Scope Document* ([U.S. EPA, 2017d](#)).

⁹ Key and supporting data and information are those that support key analyses, arguments, and/or conclusions in the risk evaluation.

Figure 1-5 to Figure 1-9 depict literature flow diagrams illustrating the results of this process for each scientific discipline-specific evidence supporting the risk evaluation. Each diagram provides the total number of references at the start of each systematic review stage (i.e., data search, data screening, data evaluation, data extraction/data integration) and those excluded based on criteria guiding the screening and data quality evaluation decisions.

EPA made the decision to bypass the data screening step for data sources that were highly relevant to the risk evaluation as described above. These data sources are depicted as “key/supporting data sources” in the literature flow diagrams. Note that the number of “key/supporting data sources” were excluded from the total count during the data screening stage and added, for the most part, to the data evaluation stage depending on the discipline-specific evidence. The exception was the releases and occupational exposure data sources that were subject to a combined data extraction and evaluation step (Figure 1-6).

The number of publications considered in each step of the systematic review of methylene chloride for environmental fate and transport literature is summarized in Figure 1-5.

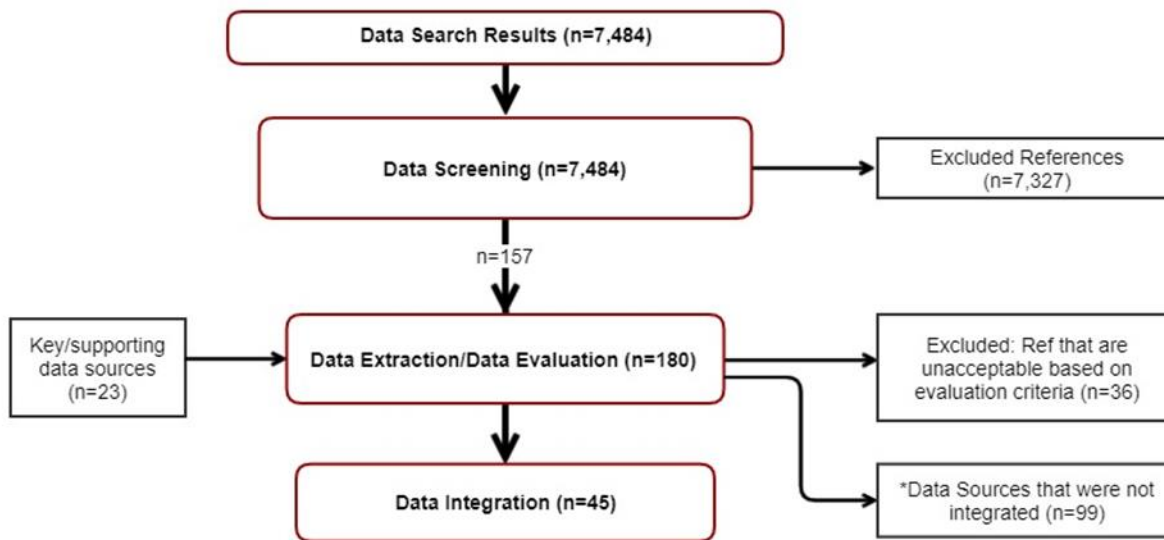


*This is a key and supporting source from existing assessments, the EPI Suite™ set of models, that was highly relevant for the TSCA risk evaluation. This source bypassed the data screening step and moved directly to the data evaluation step.

Figure 1-5. Literature Flow Diagram for Environmental Fate and Transport Data Sources

Note: Literature search results for the environmental fate and transport of methylene chloride yielded 7,216 studies. During problem formulation, following data screening, most environmental exposure pathways were removed from the conceptual models. As a result, 7,170 studies were deemed off-topic and excluded. One key source and the remaining 46 studies related to environmental exposure pathways retained in the conceptual models entered data evaluation, where 4 studies were deemed unacceptable and 43 moved into data extraction and integration.

The number of publications considered in each step of the systematic review of methylene chloride for releases and occupational exposure literature is summarized in Figure 1-6.

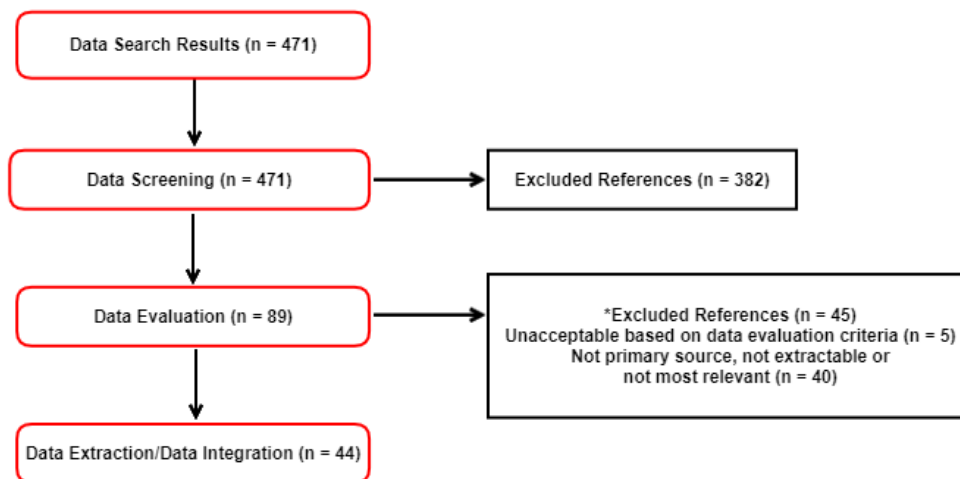


*The quality of data in these sources (n=99) were acceptable for risk evaluation purposes, but they were ultimately excluded from further consideration based on EPA's integration approach for environmental release and occupational exposure data/information. EPA's approach uses a hierarchy of preferences that guide decisions about what types of data/information are included for further analysis, synthesis and integration into the environmental release and occupational exposure assessments. EPA prefers using data with the highest rated quality among those in the higher level of the hierarchy of preferences (i.e., data > modeling > occupational exposure limits or release limits). If warranted, EPA may use data/information of lower rated quality as supportive evidence in the environmental release and occupational exposure assessments.

Figure 1-6. Releases and Occupational Exposures Literature Flow Diagram for Methylene Chloride

Note: Literature search results for environmental release and occupational exposure yielded 7,484 data sources. Of these data sources, initially 268 were determined to be relevant for the risk evaluation through the data screening process. Due to the scope changing the initial 268 data sources were reevaluated and it was determined 157 data sources to be relevant for the risk evaluation through the data screening process. These relevant data sources were entered into the data extraction/evaluation phase. After data extraction/evaluation, EPA identified several data gaps and performed a supplemental, targeted search to fill these gaps (e.g., to locate information needed for exposure modeling). The supplemental search yielded 23 relevant data sources that bypassed the data screening step and were evaluated and extracted in accordance with Appendix D of Data Quality Criteria for Occupational Exposure and Release Data of the Application of Systematic Review for TSCA Risk Evaluations document ([U.S. EPA, 2018b](#)). Of the 179 sources from which data were extracted and evaluated, 36 sources only contained data that were rated as unacceptable based on serious flaws detected during the evaluation. Of the 143 sources forwarded for data integration, data from 45 sources were integrated, and 99 sources contained data that were not integrated (e.g., lower quality data that were not needed due to the existence of higher quality data, data for release media that were removed from scope after data collection). The data integration strategy for releases and occupational exposure data is discussed in Appendix G of the document titled "Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment" ([EPA, 2019b](#)).

The number of publications considered in each step of the systematic review of methylene chloride for non-occupational exposure literature is summarized in Figure 1-7.



*The quality of data in these sources were acceptable for risk evaluation purposes and considered for integration. The sources; however, were not extracted for a variety of reasons, such as they contained only secondary source data, duplicate data, or non-extractable data (i.e., charts or figures). Additionally, some data sources were not as relevant to the PECO as other data sources which were extracted.

Figure 1-7. Literature Flow Diagram for General Population, Consumer and Environmental Exposure Data Sources

Note: EPA conducted a literature search to determine relevant data sources for assessing exposures for methylene chloride within the scope of the risk evaluation. This search identified 471 data sources including relevant supplemental documents. Of these, 382 were excluded during the screening of the title, abstract, and/or full text and 89 data sources were recommended for data evaluation across up to five major study types in accordance with *Appendix E: Data Quality Criteria for Studies on Consumer, General Population and Environmental Exposure of the Application of Systematic Review for TSCA Risk Evaluations* document. (U.S. EPA, 2018b). Following the evaluation process, 44 references were forwarded for further extraction and data integration.

The conceptual model for environmental exposures was modified during problem formulation, which changed 63 previously on-topic references to off-topic between data screening and data evaluation, leaving 79 publications in the data evaluation stage.

The number of publications considered in each step of the systematic review of methylene chloride for environmental hazard literature is summarized in Figure 1-8.

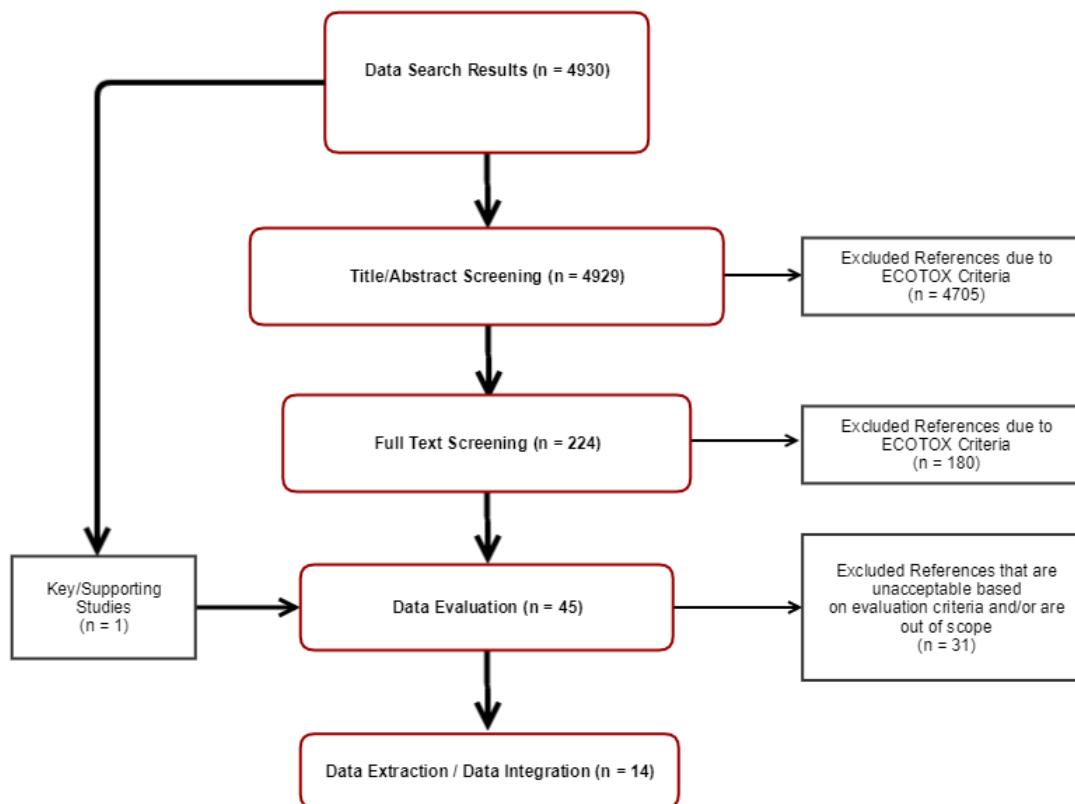


Figure 1-8. Literature Flow Diagram for Environmental Hazard Data Sources

Note: The environmental hazard data sources were identified through literature searches and screening strategies using the ECOTOXicology Knowledgebase System (ECOTOX) Standing Operating Procedures. For studies determined to be on-topic after title and abstract screening, EPA conducted a full text screening to further exclude references that were not relevant to the risk evaluation. Screening decisions were made based on eligibility criteria as documented in the ECOTOX User Guide ([EPA, 2018b](#)). Additional details can be found in the *Strategy for Conducting Literature Searches for Methylene Chloride Supplemental Document to the TSCA Scope Document* ([U.S. EPA, 2017d](#)).

The “Key/Supporting Studies” box represents data sources typically cited in existing assessments and considered highly relevant for the TSCA risk evaluation because they were used as key and supporting information by regulatory and non-regulatory organizations to support their chemical hazard and risk assessments. These citations were found independently from the ECOTOX process. These studies bypassed the data screening step and moved directly to the data evaluation step.

Studies could be considered “out of scope” after the screening steps, and therefore excluded from data evaluation, due to the elimination of pathways during scoping/problem formulation.

The number of publications considered in each step of the systematic review of methylene chloride for human health hazard literature is summarized in Figure 1-9.

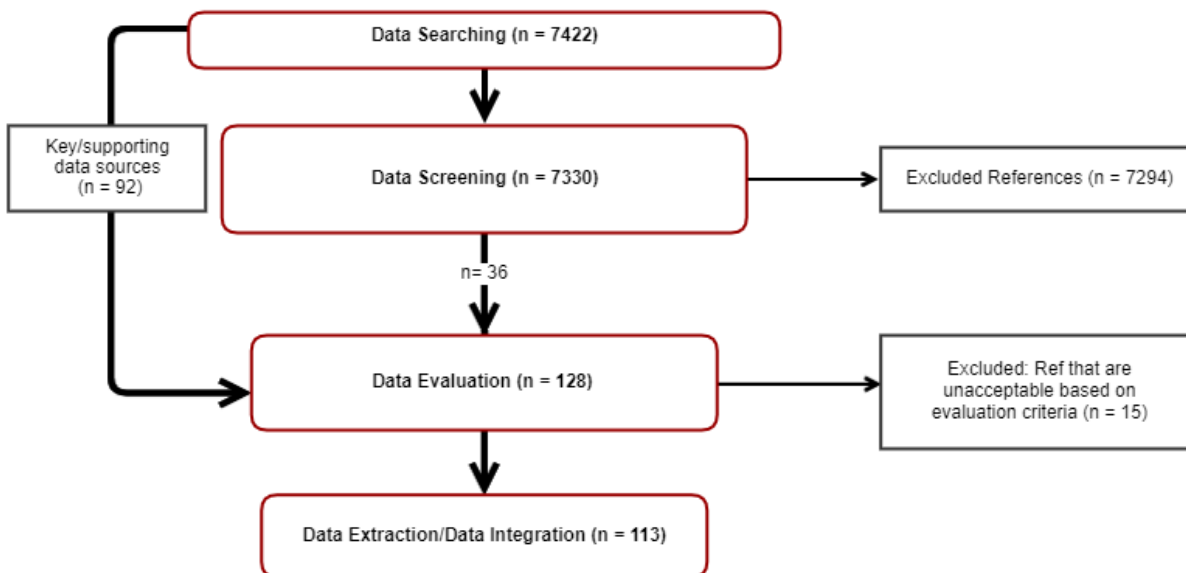


Figure 1-9. Literature Flow Diagram for Human Health Hazard Data Sources

Note: Literature search results for human health hazard of methylene chloride yielded 7,422 studies. This included 92 key and supporting studies identified from previous EPA assessments. Of the 7,330 new studies screened for relevance, 7,294 were excluded as off topic. The remaining 36 new studies and 92 key/supporting studies were evaluated for data quality. Fifteen studies were deemed unacceptable based on the evaluation criteria of human health hazard and the remaining 113 studies were carried forward to data extraction/data integration.

2 EXPOSURES

2.1 Fate and Transport

Environmental fate includes both environmental transport and transformation processes. Environmental transport is the movement of the chemical within and between environmental media. Transformation occurs through the degradation or reaction of the chemical in the environment. Hence, understanding the environmental fate of methylene chloride informs the determination of the specific exposure pathways, and potential human and environmental receptors which EPA considered in its risk evaluation.

2.1.1 Fate and Transport Approach and Methodology

EPA gathered and evaluated environmental fate information according to the process described in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018a). Reasonably available environmental fate data, including biotic and abiotic degradation rates, removal during wastewater treatment, volatilization from lakes and rivers, and an organic carbon:water partition coefficient (K_{OC}) were selected for use in the current evaluation. Sufficient numbers of high-confidence biodegradation studies were available, so it was not necessary to use lower-quality data for that endpoint; thus, in assessing the environmental fate and transport of methylene chloride, EPA considered the full range of results from sources that were rated high confidence. Complete data extraction tables are available in the supplemental file *Data Extraction Tables for Environmental Fate and Transport Studies* (EPA, 2019e) and complete data evaluation information is available in the supplemental file *Data Quality Evaluation of Environmental Fate and Transport Studies* (EPA, 2019f).

Other fate estimates were based on modeling results from EPI (Estimation Programs Interface) Suite™ (U.S. EPA, 2012), a predictive tool for physical/chemical and environmental fate properties (<https://www.epa.gov/tsca-screening-tools/epi-suite™-estimation-program-interface>). Information regarding the EPI Suite™ model inputs is available in Appendix C and model outputs are available in the supplemental file *Data Extraction Tables for Environmental Fate and Transport Studies* (EPA, 2019e). EPI Suite™ was reviewed by the EPA Science Advisory Board ([http://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/CCF982BA9F9CFCFA8525735200739805/\\$File/sab-07-011.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/CCF982BA9F9CFCFA8525735200739805/$File/sab-07-011.pdf)) and the individual models have been peer-reviewed in numerous articles published in technical journals. Citations for such articles are available in the EPI Suite™ help files.

Table 2-1 provides environmental fate data that EPA considered while assessing the fate of methylene chloride. The data in Table 2-1 were updated after problem formulation with information identified through systematic review.

Table 2-1. Environmental Fate Characteristics of Methylene Chloride

Property or Endpoint	Value ^a	References	Data Quality Rating
Indirect photodegradation half-life	79 days (atmospheric oxidation by reaction with hydroxyl radicals [\bullet OH]; estimated) ^b	U.S. EPA (2012)	High
	97 days (atmospheric oxidation by reaction with \bullet OH; estimated) ^c	(Mansouri et al., 2018)	High
Hydrolysis half-life	18 months	Dilling et al. (1975)	High
	4.3x10 ⁷ yrs (estimated) ^b	U.S. EPA (2012)	High
Aerobic Biodegradation	0% in 28 days (activated sludge)	Lapertot and Pulgarin (2006)	High
	100% in 7 days (activated sludge)	Tabak et al. (1981)	High
	90% in 6 days (marine water)	Krausova et al. (2006)	High
Anaerobic Biodegradation	58% in 30 hrs (pre-adapted culture)	Braus-Stromeyer et al. (1993)	High
	65-84% in 31 hrs (sediment)	Melin et al. (1996)	High
	Approx. 75% in 22 days (sediment)	Peijnenburg et al. (1998)	High
	100% in 10 days (digested sludge)	Gossett (1985)	High
Bioconcentration factor (BCF)	3.1 (estimated by linear regression from octanol-water partition coefficient) ^b	U.S. EPA (2012)	High
	2.6 (estimated by Arnot-Gobas quantitative structure-activity relationship [QSAR]) ^b		
Bioaccumulation factor (BAF)	<1 - 577 (measured in lentic ecosystem microcosm)	Thiébaud et al. (1994)	High
	2.6 (estimated by Arnot-Gobas QSAR) ^b	U.S. EPA (2012)	High
	15.1 (estimated) ^c	(Mansouri et al., 2018)	High
log K _{oc}	1.34 (estimated from molecular connectivity index) ^b	U.S. EPA (2012)	High
	1.08 (estimated from log K _{ow}) ^b		
	1.5 (estimated) ^c	(Mansouri et al., 2018)	High

^a Measured unless otherwise noted.
^b Information was estimated using EPI Suite™ ([U.S. EPA, 2012](#))
^c Information was estimated using OPERA ([Mansouri et al., 2018](#))

2.1.2 Summary of Fate and Transport

The EPI Suite™ ([U.S. EPA, 2012](#)) model that predicts removal in wastewater treatment (STPWIN; see Appendix C for information regarding inputs used for EPI Suite™) estimated that < 1% of methylene chloride in influent water will be removed via sorption to sludge. The organic water-carbon partition coefficient ($\log K_{OC}$) is estimated to be 1.4, which is associated with low sorption to sludge, soil, and sediment. Due to its Henry's Law constant (0.00325 atm·m³/mole), methylene chloride is expected to volatilize rapidly from water; STPWIN estimated that approximately 56% of methylene chloride in influent would be removed by volatilization to the air. Reported aerobic biodegradation rates are mixed, ranging from slow (e.g., negligible degradation in 28 days) to fast (e.g., complete degradation in 7 days) ([Krausova et al., 2006](#); [Lapertot and Pulgarin, 2006](#); [Tabak et al., 1981](#)), so overall removal of methylene chloride from wastewater treatment is expected to range from 57% (based on STPWIN estimates for volatilization to air and sorption to sludge, with negligible biodegradation) to complete (based on volatilization, sorption, and high biodegradation). The low end of this range is similar to the methylene chloride removal efficiency (54%) reported by the EPA Toxics Release Inventory (TRI) ([U.S. EPA, 2017f](#)).

Based on the results of the STPWIN model, in which removal of methylene chloride from wastewater is dominated by volatilization, in combination with possible biodegradation, concentrations of methylene chloride in land-applied biosolids are expected to be lower than concentrations in wastewater treatment plant effluents. Methylene chloride has been detected in biosolids [e.g., [EPA \(1996\)](#)] however land-applied biosolids are spread over a large area and diluted in runoff and surface water. Level III fugacity modeling as implemented in EPI Suite™ using 100% emission to soil as a proxy for land application of biosolids estimates that 58% of methylene chloride volatilizes to air, 38% remains in soil, and 3% is transported to water. However, the model assumes constant emissions rather than a pulse as land application of biosolids would be; thus, those model results likely overstate how much methylene chloride would remain in soil. Overall, based on p-chem and fate properties and the results of fugacity modeling, surface and drinking water exposures from land-applied biosolids are likely negligible.

Based on its low partitioning to organic matter and rapid biodegradation in anaerobic environments ([Peijnenburg et al., 1998](#); [Melin et al., 1996](#); [Braus-Stromeyer et al., 1993](#); [Gossett, 1985](#)), methylene chloride is expected to be present in sediments at concentrations similar to or lower than those of the overlying water. Although the $\log K_{OC}$ indicates that methylene chloride will partition to sediment organic carbon, organic matter typically comprises 25% or less of sediment composition (e.g., <https://pubs.usgs.gov/of/2006/1053/downloads/pdf/of-2006-1053.pdf>) of which approximately 40-60% is organic carbon ([Schwarzenbach et al., 2003](#)). Thus, the fraction of organic carbon (f_{OC}) in soil is typically 0.15 or less. Based on these values, the sediment-water K_d (where $K_d = K_{OC} * f_{OC}$) is expected to be equal to or less than 3.8, indicating that at equilibrium, concentrations in sediment would be expected to be less than four times higher than in porewater. However, methylene chloride concentrations in sediment are expected to be depressed by rapid biodegradation in anaerobic sediments and porewater interaction with overlying surface water. Thus, concentrations in sediment and pore water are expected to be similar to or less than concentrations in overlying water.

Due to its high Henry's Law constant and vapor pressure (435 mmHg at 25°C), methylene chloride is expected to volatilize from surface water and soil. The EPI Suite™ module that estimates volatilization from lakes and rivers (water volatilization model) was run using default settings to evaluate the volatilization half-life of methylene chloride in surface water and estimated that the half-life of methylene chloride in a model river will be 1.1 hours and the half-life in a model lake will be less than 4 days. In the atmosphere, methylene chloride will slowly react with hydroxyl radicals ($\bullet\text{OH}$), with an indirect photolysis half-life of 79 days. Due to its persistence, methylene chloride is expected to be subject to local and long-range atmospheric transport. Based on its vapor density (2.93 relative to air), volatilized methylene chloride is expected to remain near ground level in very calm conditions, but with mixing will readily disperse into the air.

Although methylene chloride released to the environment is likely to evaporate to the atmosphere, due to its low partitioning to organic matter it may migrate to groundwater. Indeed, detections of methylene chloride in groundwater have been reported (e.g., in the EPA's Water Quality portal, <http://www.waterqualitydata.us/portal.jsp>; reports of detection in groundwater did not go through data evaluation and extraction because groundwater pathways are outside the scope of this risk evaluation). In groundwater, methylene chloride may slowly hydrolyze.

The bioconcentration potential of methylene chloride is low; the EPI Suite™ BCFBAF model estimates bioconcentration factors of 2.6 to 3.1 and a bioaccumulation factor of 2.6 (U.S. EPA, 2012), and a study of bioaccumulation in a lentic microcosm reported radioactivity accumulation factors ranging from <1 to 577 (Thiébaud et al., 1994).

Overall, methylene chloride is expected to have limited accumulation potential in wastewater biosolids, soil, sediment, and biota. Methylene chloride released to surface water or soil is likely to volatilize to the atmosphere, where it will slowly photooxidize. Methylene chloride may migrate to groundwater, where it may be removed via anaerobic biodegradation or slowly hydrolyze. Figure 2-1 summarizes the overall environmental partitioning and degradation expected for methylene chloride.

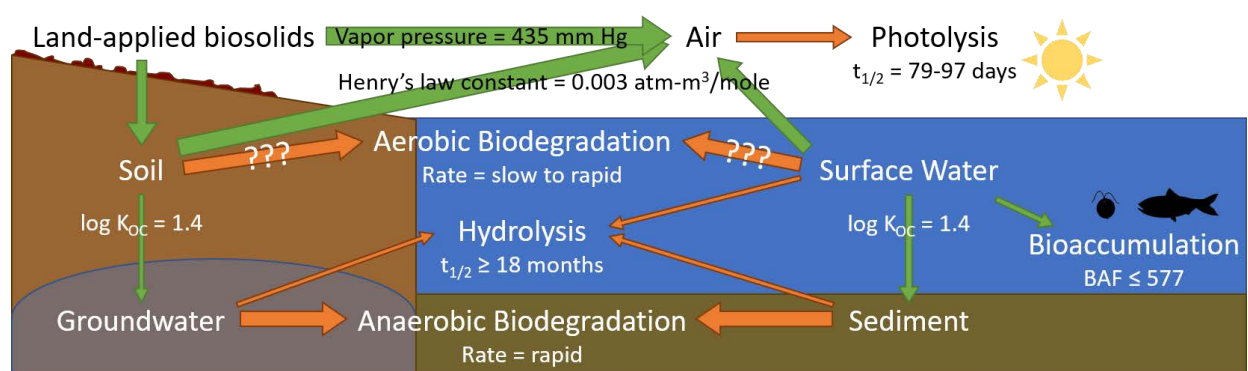


Figure 2-1 Environmental transport, partitioning, and degradation processes for methylene chloride.

In Figure 2-1, transport and partitioning are indicated by green arrows and degradation is indicated by orange arrows. The width of the arrow is a qualitative indication of the likelihood that the indicated partitioning will occur or the rate at which the indicated degradation will occur (i.e., wider arrows indicate more likely partitioning or more rapid degradation). The question marks over the aerobic biodegradation arrow indicate uncertainty regarding how quickly methylene chloride will biodegrade. Although transport and partitioning processes (green arrows) can occur in both directions, the image illustrates the primary direction of transport indicated by partition coefficients. Figure 2-1 considers only transport, partitioning, and degradation within and among environmental media; sources to the environment such as discharge and disposal are not illustrated.

2.1.3 Key Sources of Uncertainty in Fate and Transport Assessment

The experimentally determined methylene chloride biodegradation rates in aerobic environments ranged from slow to rapid (see Table 2-1). The fastest degradation was reported by Tabak et al. (1981), who measured 100% degradation in 7 days. Conversely, Lapertot and Pulgarin (2006) reported 0% degradation in 28 days with the explanation that methylene chloride was causing cell lysis. Cell lysis may not have been observed by Tabak et al. (1981) because methylene chloride was spiked into their test vessels at concentrations 5-10 times lower than those used by Lapertot and Pulgarin (2006) (5-10 mg/L versus 50 mg/L).

Methylene chloride biodegradation data reported to foreign governments demonstrate similar discrepancies. Data submitted to Japanese National Institute of Technology and Evaluation reported $\leq 13\%$ of methylene chloride degraded after 28 days from an initial concentration of 100 mg/L, whereas data submitted to the European Chemicals Agency showed that 68% of methylene chloride was removed in 28 days from an initial concentration of 5 mg/L.

For comparison, the EPI Suite™ module that predicts biodegradation rates (“BIOWIN” module) was run using default settings to estimate biodegradation rates of methylene chloride. The BIOWIN models for aerobic environments (BIOWIN 1-6) estimate that methylene chloride will not rapidly biodegrade in aerobic environments. In agreement with the experimental data for anaerobic biodegradation of methylene chloride, the BIOWIN model of anaerobic biodegradation (BIOWIN 7) predicts that methylene chloride will biodegrade rapidly under anaerobic conditions. Overall, methylene chloride biodegradation rates in aerobic environments may vary based on factors including microorganism consortia present and microorganisms’ previous exposure and adaptation to methylene chloride or other halogenated substances. This uncertainty in biodegradation rates was considered in the assessment of environmental persistence.

The uncertainty around aerobic biodegradation rates also impacts estimates of removal from wastewater. As described in Section 2.1.2, the STPWIN module of EPI Suite™ estimates that 57% of methylene chloride in influent wastewater will be removed via sorption to sludge or volatilization to air. Biodegradation rates in activated sludge and settled biosolids are dependent on factors such as the microbial consortia present, their previous adaptation to methylene chloride, and the biomass concentrations in activated sludge stage. Thus, biodegradation in WWTP may range from negligible to complete, resulting in overall removal estimates of 57% be abiotic processes alone to complete via abiotic and biotic removal processes.

2.2 Releases to the Environment

2.2.1 Water Release Assessment Approach and Methodology

EPA performed a literature search to identify process operations that could potentially result in direct or indirect discharges to water for each condition of use. Where available, EPA used 2016 Toxics Release Inventory (TRI) ([U.S. EPA, 2017f](#)) and 2016 Discharge Monitoring Report (DMR) ([EPA, 2016](#)) data to provide a basis for estimating releases. Facilities are only required to report to TRI if the facility has 10 or more full-time employees, is included in an applicable North American Industry Classification System (NAICS) code, and manufactures, processes, or uses the chemical in quantities greater than a certain threshold (25,000 pounds for manufacturers and processors of methylene chloride and 10,000 pounds for users of methylene chloride). Due to these limitations, some sites that manufacture, process, or use methylene chloride may not report to TRI and are therefore not included in these datasets.

For the 2016 DMR, EPA used the Water Pollutant Loading Tool within EPA's Enforcement and Compliance History Online (ECHO), <https://echo.epa.gov/trends/loading-tool/water-pollution-search/>, to query all methylene chloride point source water discharges in 2016. DMR data are submitted by National Pollutant Discharge Elimination System (NPDES) permit holders to states or directly to the EPA according to the monitoring requirements of the facility's permit. States are only required to load major discharger data into DMR and thus, may or may not load minor discharger data. The definition of major vs. minor discharger is set by each state and could be based on discharge volume or facility size. Due to these limitations, some sites that discharge methylene chloride may not be included in the DMR dataset.

Facilities reporting releases in TRI and DMR also report associated NAICS and Standard Industrial Classification (SIC) industry codes, respectively. Where possible, EPA reviewed the NAICS and SIC descriptions for each reported release and mapped each facility to a potential condition of use associated with occupational exposure scenarios (OES, see Table 2-22). For facilities that did not report a NAICS or SIC code, EPA performed a supplemental internet search of the specific facility to determine the mapping. Facilities that could not be mapped were grouped together into an "Other" category.

When possible for each OES covering conditions of use, EPA estimated annual releases, average daily releases, and number of release days/yr. Where TRI and/or DMR were available, EPA used the reported annual releases for each site and estimated the daily release by averaging the annual release over the estimated release days/yr. Where releases are expected but TRI and DMR data were not available, EPA included a qualitative discussion of potential release sources.

EPA did not locate data on number of release days/yr for facilities. The following guidelines were used to estimate the number of release days/yr:

- **Manufacturing:** For the manufacture of the solvents with large production volumes, EPA assumes 350 days/yr for release frequency. This frequency assumes that the facility operates 7 days/week and 50 weeks/yr (with two weeks down for turnaround) and that the facility is producing and releasing the chemical daily during operation.

- Processing as Reactant: Methylene chloride is used to manufacture other commodity chemicals, such as refrigerants or other chlorinated compounds, which will likely occur year-round. Therefore, EPA assumes 350 days/yr for release frequency based on the same assumptions for Manufacturing.
- Processing into Formulation Product: For these facilities, EPA does not expect that methylene chloride will be used year-round, even if the facility operates year-round. Therefore, EPA assumes 300 days/yr for release frequency, which is based on a European Union SpERC that uses a default of 300 days/yr for release frequency for the chemical industry ([Echa, 2013](#)).
- Wastewater Treatment Plants: For these facilities, EPA expects that they will be used year-round. Therefore, EPA assumes 365 days/yr for release frequency.
- All Other Scenarios: For all other scenarios, EPA does not expect that methylene chloride will be used year-round and assumes 250 days/yr for release frequency (5 days/week, 50 weeks/yr).

2.2.2 Water Release Estimates by Occupational Exposure Scenario

As noted in the previous section, EPA mapped each facility to a potential condition of use associated with occupational exposure scenarios (OES, see Table 2-22). Facilities that could not be mapped were grouped together into an “Other” category. The following sections show release estimates per facility for each OES. The supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)) provides background details on industries that may use methylene chloride, processes, and numbers of sites for each OES.

2.2.2.1 Manufacturing

EPA assumed that sites under NAICS 325199 (All Other Basic Organic Chemical Manufacturing) or SIC 2869 (Industrial Organic Chemicals, Not Elsewhere Classified) are potentially applicable to manufacturing of methylene chloride. These NAICS codes may be applicable to other conditions of use (processing as a reactant, processing—incorporation into formulation, mixture, or reaction product); however, insufficient information was reasonably available to make these determinations.

Table 2-2 lists all facilities under these NAICS and SIC codes that reported direct or indirect water releases in the 2016 TRI or 2016 DMR. Of the potential manufacturing sites listed in CDR, only one facility was present in Table 2-2, which reported 128 pounds (58 kg) of methylene chloride transferred off-site to wastewater treatment (Olin Blue Cube, Freeport, TX) ([U.S. EPA, 2017f](#)). Due to TRI and CDR reporting thresholds, some sites that reported manufacturing methylene chloride in CDR may not report to TRI, or vice versa. For the sites reporting for this scenario, the release estimates range from 0.01 to 76 kg/site-yr over 350 days/yr.

Table 2-2. Reported TRI Releases for Organic Chemical Manufacturing Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
COVESTRO LLC	BAYTOWN	TX	1	350	0.004	Surface Water	U.S. EPA (2017f)
EMERALD PERFORMANCE MATERIALS LLC	HENRY	IL	0.5	350	0.001	Surface Water	U.S. EPA (2017f)
FISHER SCIENTIFIC CO LLC	FAIR LAWN	NJ	2	350	0.01	POTW	U.S. EPA (2017f)
FISHER SCIENTIFIC CO LLC	BRIDGEWATER	NJ	2	350	0.01	POTW	U.S. EPA (2017f)
OLIN BLUE CUBE FREEPORT TX	FREEPORT	TX	58	350	0.2	Non-POTW WWT	U.S. EPA (2017f)
REGIS TECHNOLOGIES INC	MORTON GROVE	IL	2	350	0.01	POTW	U.S. EPA (2017f)
SIGMA-ALDRICH MANUFACTURING LLC	SAINT LOUIS	MO	2	350	0.01	POTW	U.S. EPA (2017f)
VANDERBILT CHEMICALS LLC-MURRAY DIV	MURRAY	KY	0.5	350	0.001	Non-POTW WWT	U.S. EPA (2017f)
E I DUPONT DE NEMOURS - CHAMBERS WORKS	DEEPWATER	NJ	76	350	0.2	Surface Water	EPA (2016)
BAYER MATERIALSCIENCE BAYTOWN	BAYTOWN	TX	10	350	0.03	Surface Water	EPA (2016)
INSTITUTE PLANT	INSTITUTE	WV	3	350	0.01	Surface Water	EPA (2016)
MPM SILICONES LLC	FRIENDLY	WV	2	350	0.005	Surface Water	EPA (2016)
BASF CORPORATION	WEST MEMPHIS	AR	1	350	0.003	Surface Water	EPA (2016)
ARKEMA INC	PIFFARD	NY	0.3	350	0.001	Surface Water	EPA (2016)
EAGLE US 2 LLC - LAKE CHARLES COMPLEX	LAKE CHARLES	LA	0.2	350	0.001	Surface Water	EPA (2016)
BAYER MATERIALSCIENCE	NEW MARTINSVILLE	WV	0.2	350	0.001	Surface Water	EPA (2016)
ICL-IP AMERICA INC	GALLIPOLIS FERRY	WV	0.1	350	0.0004	Surface Water	EPA (2016)
KEESHAN AND BOST CHEMICAL CO., INC.	MANVEL	TX	0.02	350	0.00005	Surface Water	EPA (2016)
INDORAMA VENTURES OLEFINS, LLC	SULPHUR	LA	0.01	350	0.00003	Surface Water	EPA (2016)
CHEMTURA NORTH AND SOUTH PLANTS	MORGANTOWN	WV	0.01	350	0.00002	Surface Water	EPA (2016)

2.2.2.2 Processing as a Reactant

EPA assumed that sites classified under NAICS 325320 (Pesticide and Other Agricultural Chemical Manufacturing) or SIC 2879 (Pesticides and Agricultural Chemicals, Not Elsewhere Classified) are potentially applicable to processing of methylene chloride as a reactant. Table 2-3 lists all facilities under these NAICS and SIC codes that reported direct or indirect water releases in the 2016 TRI or 2016 DMR. For the sites reporting for this scenario, the release estimates range from 0.1 to 213 kg/site-yr over 350 days/yr.

Table 2-3. Reported 2016 TRI and DMR Releases for Potential Processing as Reactant Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
AMVAC CHEMICAL CO	AXIS	AL	213	350	0.6	Non-POTW WWT	U.S. EPA (2017f)
THE DOW CHEMICAL CO	MIDLAND	MI	25	350	0.1	Surface Water	U.S. EPA (2017f)
FMC CORPORATION	MIDDLEPORT	NY	0.1	350	0.0003	Surface Water	EPA (2016)

2.2.2.3 Processing – Incorporation into Formulation, Mixture, or Reaction Product

EPA identified six NAICS and SIC codes, listed in Table 2-4, that reported water releases in the 2016 TRI and may be related to use as Processing – Incorporation into Formulation, Mixture, or Reaction Product. Table 2-4 lists all facilities classified under these NAICS and SIC codes that reported direct or indirect water releases in the 2016 TRI or 2016 DMR. For the sites reporting for this scenario, the release estimates range from 0.2 to 5,785 kg/site-yr over 350 days/yr.

Table 2-4. Potential Industries Conducting Methylene Chloride Processing – Incorporation into Formulation, Mixture, or Reaction Product in 2016 TRI or DMR

NAICS Code	NAICS Description
325180	Other Basic Inorganic Chemical Manufacturing
325510	Paint and Coating Manufacturing
325998	All Other Miscellaneous Chemical Product and Preparation Manufacturing
2819	INDUSTRIAL INORGANIC CHEMICALS
2843	SURF ACTIVE AGENT, FIN AGENTS
2899	CHEMICALS & CHEM PREP, NEC

Table 2-5. Reported 2016 TRI and DMR Releases for Potential Processing—Incorporation into Formulation, Mixture, or Reaction Product Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
ARKEMA INC	CALVERT CITY	KY	31	300	0.1	Surface Water	U.S. EPA (2017f)
MCGEAN-ROHCO INC	LIVONIA	MI	113	300	0.4	POTW	U.S. EPA (2017f)

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
WM BARR & CO INC	MEMPHIS	TN	0.5	300	0.002	POTW	U.S. EPA (2017f)
BUCKMAN LABORATORIES INC	MEMPHIS	TN	254	300	1	POTW	U.S. EPA (2017f)
EUROFINS MWG OPERON LLC	LOUISVILLE	KY	5,785	300	19	POTW	U.S. EPA (2017f)
SOLVAY - HOUSTON PLANT	HOUSTON	TX	12	300	0.04	Surface Water	EPA (2016)
HONEYWELL INTERNATIONAL INC - GEISMAR COMPLEX	GEISMAR	LA	4	300	0.01	Surface Water	EPA (2016)
STEPAN CO MILLSDALE ROAD	ELWOOD	IL	2	300	0.01	Surface Water	EPA (2016)
ELEMENTIS SPECIALTIES, INC.	CHARLESTON	WV	0.2	300	0.001	Surface Water	EPA (2016)

2.2.2.4 Repackaging

EPA assumed that sites classified under NAICS 424690 (Other Chemical and Allied Products Merchant Wholesalers) or SIC 5169 (Chemicals and Allied Products) are potentially applicable to repackaging of methylene chloride. Table 2-6 lists all facilities in these industries that reported direct or indirect water release to the 2016 TRI or 2016 DMR. None of the potential repackaging sites listed in CDR reported water releases to TRI or DMR in reporting year 2016. For the sites reporting for this scenario, the release estimates range from 0.03 to 144 kg/site-yr over 250 days/yr.

Table 2-6. Reported 2016 TRI and DMR Releases for Repackaging Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
CHEMISPHERE CORP	SAINT LOUIS	MO	2	250	0.01	POTW	U.S. EPA (2017f)
HUBBARD-HALL INC	WATERBURY	CT	144	250	1	Non-POTW WWT	U.S. EPA (2017f)
WEBB CHEMICAL SERVICE CORP	MUSKEGON HEIGHTS	MI	98	250	0.4	POTW	U.S. EPA (2017f)
RESEARCH SOLUTIONS GROUP INC	PELHAM	AL	0.09	250	0.0003	Surface Water	EPA (2016)
EMD MILLIPORE CORP	CINCINNATI	OH	0.03	250	0.0001	Surface Water	EPA (2016)

2.2.2.5 Batch Open-Top Vapor Degreasing

EPA did not identify quantitative information about water releases during batch open-top vapor degreasing (OTVD). The primary source of water releases from OTVDs is wastewater from the water separator. Water in the OTVD may come from two sources: 1) Moisture in the atmosphere that condenses into the solvent when exposed to the condensation coils on the OTVD; and/or 2) steam used to regenerate carbon adsorbers used to control solvent emissions on OTVDs with enclosures ([Durkee, 2014](#); [Kanegsberg and Kanegsberg, 2011](#); [\(NIOSH\), 2002a, b](#); [Niosh, 2002a, b](#)). The water is removed in a gravity separator and sent for disposal ([\(NIOSH\), 2002a, b](#); [Niosh, 2002a, b](#)). The current disposal practices of the wastewater are unknown; however, a U.S. EPA ([1982](#)) report estimated 20% of water releases from metal cleaning (including batch systems, conveyORIZED systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

2.2.2.6 ConveyORIZED Vapor Degreasing

EPA did not identify quantitative information about water releases during vapor degreasing. The current disposal practices of the wastewater are unknown; however, a U.S. EPA ([1982](#)) report estimated 20% of water releases from metal cleaning (including batch systems, conveyORIZED systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

2.2.2.7 Cold Cleaning

EPA did not identify quantitative information about water releases during cold cleaning. The current disposal practices of the wastewater are unknown; however, a U.S. EPA ([1982](#)) report estimated 20% of water releases from metal cleaning (including batch systems, conveyORIZED systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

2.2.2.8 Commercial Aerosol Products

EPA does not expect releases of methylene chloride to water from the use of aerosol products. Due to the volatility of methylene chloride the majority of releases from the use of aerosol products will likely be to air as methylene chloride evaporates from the aerosolized mist and the substrate surface. There is a potential that methylene chloride that deposits on shop floors during the application process could possibly end up in a floor drain (if the shop has one) or could runoff outdoors if garage doors are open. However, EPA expects the potential release to water from this to be minimal as there would be time for methylene chloride to evaporate before entering one of these pathways. This is consistent with estimates from the International Association for Soaps, Detergents and Maintenance Products (AISE) Specific Environmental Release Categories (SpERC) for Wide Dispersive Use of Cleaning and Maintenance Products, which estimates 100% of volatiles are released to air ([AISE, 2012](#)). EPA expects residuals in the aerosol containers to be disposed of with shop trash that is either picked up by local waste management or by a waste handler that disposes shop wastes as hazardous waste.

2.2.2.9 Adhesives and Sealants

Based on a mass balance study on the Dutch use of methylene chloride as adhesives, the Netherlands Organisation for Applied Scientific Research (TNO) calculated an emission of 100% to air ([TNO \(CIVO\), 1999](#)). EPA did not find information on potential water releases. Water releases may occur if equipment is cleaned with water.

2.2.2.10 Paints and Coatings

EPA did not identify information about potential water releases during application of paints and coatings. Water releases may occur if equipment is cleaned with water; however, industrial and commercial sites would likely be expected to dispose of solvent-based paints as hazardous waste.

2.2.2.11 Adhesive and Caulk Removers

EPA did not find specific industry information or release data for use of adhesive and caulk removers. EPA did not identify quantitative information in the 2016 TRI or 2016 DMR for this use. Professional contractors who may use adhesive and caulk removers likely do not handle enough methylene chloride to meet the reporting thresholds of TRI and would not likely report to DMR because they are not industrial facilities. The majority of methylene chloride is expected to evaporate into the air, but releases to water may occur if equipment is cleaned with water.

2.2.2.12 Fabric Finishing

EPA did not identify quantitative information about potential water releases during use of methylene chloride in fabric finishing. The majority of methylene chloride is expected to evaporate into the air, but releases to water may occur if equipment or fabric is cleaned with water.

2.2.2.13 Spot Cleaning

The majority of methylene chloride in spot removers is expected to evaporate into the air, but releases to water may occur if residue remains in the garment during washing. EPA identified

one facility in the 2016 DMR with SIC code 7216 (Drycleaning Plants, Excluding Rug Cleaning). This facility reported 0.1 kg annual release of methylene chloride to surface water, as shown in Table 2-7. EPA did not identify any potential spot cleaning facilities in the 2016 TRI that reported water releases. Other facilities in this industry may not dispose to water or use methylene chloride in quantities that meet the TRI reporting threshold. For the site reporting for this scenario, the release estimate is 0.1 kg/site-yr over 250 days/yr.

Table 2-7. Surface Water Releases of Methylene Chloride During Spot Cleaning

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
BOISE STATE UNIVERSITY	BOISE	ID	0.1	250	0.0002	Surface Water	EPA (2016)

2.2.2.14 Cellulose Triacetate Film Production

EPA identified one facility in the 2016 DMR, potentially related to CTA manufacturing (SIC code 3861 - Photographic Equipment and Supplies) that reported water releases. Release for this facility is summarized in Table 2-8. EPA did not identify any potential CTA manufacturing facilities in the 2016 TRI that reported water releases. For the site reporting for this scenario, the release estimate is 29 kg/site-yr over 250 days/yr.

Table 2-8. Reported 2016 TRI and DMR Releases for CTA Manufacturing Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
KODAK PARK DIVISION	ROCHESTER	NY	29	250	0.1	Surface Water	EPA (2016)

2.2.2.15 Flexible Polyurethane Foam Manufacturing

EPA assumed that sites classified under NAICS code 326150 (Urethane and Other Foam Product (except Polystyrene) Manufacturing) are potentially applicable to polyurethane foam manufacturing.

Table 2-9 lists one facility under this NAICS code that reported direct or indirect water releases in the 2016 TRI. EPA did not identify water releases for polyurethane manufacturing sites in the 2016 DMR. This facility (Previs Innovative Packaging, Inc. in Wurtland, KY) reported 2 kilograms release to surface water ([U.S. EPA, 2017f](#)), and EPA estimates 250 days/yr release. Other facilities in this industry may not dispose to water or use methylene chloride in quantities that meet the TRI reporting threshold.

Table 2-9. Water Releases Reported in 2016 TRI for Polyurethane Foam Manufacturing

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
PREGIS INNOVATIVE PACKAGING INC	WURLAND	KY	2	250	0.01	Surface Water	U.S. EPA (2017f)

For chemical industries (including blowing agent in PUR production, which is applicable to this OES), calculations for the Dutch chemical industry estimated emissions of 0.2 % to water, 64.8 % to air and 35 % to waste, based on a mass balance study ([TNO \(CIVO\), 1999](#)).

2.2.2.16 Laboratory Use

EPA did not identify quantitative information about potential water releases during laboratory use of methylene chloride. The majority of methylene chloride is expected to evaporate into the air or disposed as hazardous waste, but releases to water may occur if equipment is cleaned with water.

2.2.2.17 Plastic Product Manufacturing

EPA identified facilities classified under four NAICS and SIC codes, listed in Table 2-10, that reported water releases in the 2016 TRI and 2016 DMR and may be related to plastic product manufacturing. Table 2-11 lists all facilities classified under these NAICS and SIC codes that reported direct or indirect water releases in the 2016 TRI or 2016 DMR. For the sites reporting for this scenario, the release estimates range from 0.02 to 28 kg/site-yr over 250 days/yr.

Table 2-10. Potential Industries Conducting Plastics Product Manufacturing in 2016 TRI or DMR

NAICS Code	NAICS Description
325211	Plastics Material and Resin Manufacturing
2821	PLSTC MAT./SYN RESINS/NV ELAST
2822	SYN RUBBER (VULCAN ELASTOMERS)
3081	UNSUPPORTED PLSTICS FILM/SHEET

Table 2-11. Reported 2016 TRI and DMR Releases for Potential Plastics Product Manufacturing Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
SABIC INNOVATIVE PLASTICS US LLC	BURKVILLE	AL	8	250	0.03	Surface Water	U.S. EPA (2017f)
SABIC INNOVATIVE	MOUNT VERNON	IN	28	250	0.1	Surface Water	EPA (2016)

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
PLASTICS MT. VERNON, LLC							
SABIC INNOVATIVE PLASTICS US LLC	SELKIRK	NY	9	250	0.03	Surface Water	EPA (2016)
EQUISTAR CHEMICALS LP	LA PORTE	TX	9	250	0.03	Surface Water	EPA (2016)
CHEMOURS COMPANY FC LLC	WASHINGTON	WV	7	250	0.03	Surface Water	EPA (2016)
SHINTECH ADDIS PLANT A	ADDIS	LA	3	250	0.01	Surface Water	EPA (2016)
STYROLUTION AMERICA LLC	CHANNAHON	IL	0.2	250	0.001	Surface Water	EPA (2016)
DOW CHEMICAL CO DALTON PLANT	DALTON	GA	0.3	250	0.001	Surface Water	EPA (2016)
PREGIS INNOVATIVE PACKAGING INC	WURTLAND	KY	0.02	250	0.0001	Surface Water	EPA (2016)

2.2.2.18 Lithographic Printing Plate Cleaning

EPA identified one facility in the 2016 DMR, potentially related to lithographic printing (SIC code 2752 - Commercial Printing, Lithographic) that reported water releases. Release for this facility is summarized in Table 2-12. EPA did not identify any potential lithographic printing facilities in the 2016 TRI that reported water releases. Other facilities in this industry may not dispose to water or use methylene chloride in quantities that meet the TRI reporting threshold. For the site reporting for this scenario, the release estimate is 0.001 kg/site-yr over 250 days/yr.

Table 2-12. Reported 2016 TRI and DMR Releases for Potential Lithographic Printing Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
FORMER REXON FACILITY AKA ENJEMS MILLWORKS	WAYNE TWP	NJ	0.001	250	0.000004	Surface Water	EPA (2016)

2.2.2.19 Non-Aerosol Commercial Uses

EPA did not identify quantitative information about potential water releases during non-aerosol use of methylene chloride. The majority of methylene chloride is expected to evaporate into the air, but releases to water may occur if equipment is cleaned with water.

2.2.2.20 Waste Handling, Disposal, Treatment, and Recycling

EPA identified facilities classified under five NAICS and SIC codes, listed in Table 2-13, that reported water releases in the 2016 TRI and 2016 DMR and may be related to recycling/disposal.

Table 2-14 lists all facilities classified under these NAICS and SIC codes that reported direct or indirect water releases in the 2016 TRI or 2016 DMR. To estimate the daily release, EPA used a default assumption of 250 days/yr of operation and averaged the annual release over the operating days. For the sites reporting for this scenario, the release estimates range from 0.02 to 115,059 kg/site-yr over 250 days/yr.

Table 2-13. Potential Industries Conducting Waste Handling, Disposal, Treatment, and Recycling in 2016 TRI or DMR

NAICS/SIC Code	NAICS/SIC Description
331492	Secondary Smelting, Refining, and Alloying of Nonferrous Metal (except Copper and Aluminum)
562211	Hazardous Waste Treatment and Disposal
4953	REFUSE SYSTEMS
7699	REPAIR SHOPS & RELATED SERVICE
9511	AIR & WATER RES & SOL WSTE MGT

Table 2-14. Reported 2016 TRI and DMR Releases for Potential Recycling/Disposal Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
JOHNSON MATTHEY	WEST DEPTFORD	NJ	620	250	2	Non-POTW WWT	U.S. EPA (2017f)
CLEAN HARBORS DEER PARK LLC	LA PORTE	TX	522	250	2	Non-POTW WWT	U.S. EPA (2017f)
CLEAN HARBORS EL DORADO LLC	EL DORADO	AR	113	250	0.5	Non-POTW WWT	U.S. EPA (2017f)
TRADEBE TREATMENT & RECYCLING LLC	EAST CHICAGO	IN	19	250	0.1	Non-POTW WWT	U.S. EPA (2017f)

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
VEOLIA ES TECHNICAL SOLUTIONS LLC	WEST CARROLLTON	OH	2	250	0.01	POTW	U.S. EPA (2017f)
VEOLIA ES TECHNICAL SOLUTIONS LLC	AZUSA	CA	0.5	250	0.002	POTW	U.S. EPA (2017f)
VEOLIA ES TECHNICAL SOLUTIONS LLC	MIDDLESEX	NJ	115,059	250	460	99.996% Non-POTW WWT 0.004% POTW	U.S. EPA (2017f)
CHEMICAL WASTE MANAGEMENT	EMELLE	AL	4	250	0.01	Surface Water	EPA (2016)
OILTANKING HOUSTON INC	HOUSTON	TX	1	250	0.003	Surface Water	EPA (2016)
HOWARD CO ALFA RIDGE LANDFILL	MARRIOTTSVILLE	MD	0.1	250	0.0002	Surface Water	EPA (2016)
CLIFFORD G HIGGINS DISPOSAL SERVICE INC SLF	KINGSTON	NJ	0.02	250	0.0001	Surface Water	EPA (2016)
CLEAN WATER OF NEW YORK INC	STATEN ISLAND	NY	2	250	0.01	Surface Water	EPA (2016)
FORMER CARBORUNDUM COMPLEX	SANBORN	NY	0.2	250	0.001	Surface Water	EPA (2016)

2.2.2.21 Other Unclassified Facilities

Table 2-15 summarizes TRI and DMR releases for facilities that were unable to be classified in one of the assessed scenarios. For the sites reporting for unclassified scenarios, the release estimates range from 0.0002 to 42 kg/site-yr over 250 days/yr.

Table 2-15. Reported 2016 TRI and DMR Releases for Other Unclassified Facilities

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
APPLIED BIOSYSTEMS LLC	PLEASANTON	CA	42	250	0.2	Non-POTW WWT	U.S. EPA (2017f)

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources & Notes
EMD MILLIPORE CORP	JAFFREY	NH	2	250	0.01	POTW	U.S. EPA (2017f)
GBC METALS LLC SOMERS THIN STRIP	WATERBURY	CT	0.2	250	0.001	Surface Water	EPA (2016)
HYSTER-YALE GROUP, INC	SULLIGENT	AL	0.0002	250	0.000001	Surface Water	EPA (2016)
AVNET INC (FORMER IMPERIAL SCHRADER)	ELLENVILLE	NY	0.005	250	0.00002	Surface Water	EPA (2016)
BARGE CLEANING AND REPAIR	CHANNELVIEW	TX	0.1	250	0.0003	Surface Water	EPA (2016)
AC & S INC	NITRO	WV	0.01	250	0.00005	Surface Water	EPA (2016)
MOOG INC - MOOG IN-SPACE PROPULSION ISP	NIAGARA FALLS	NY	0.003	250	0.00001	Surface Water	EPA (2016)
OILTANKING JOLIET	CHANNAHON	IL	1	250	0.003	Surface Water	EPA (2016)
NIPPON DYNAWAVE PACKAGING COMPANY	LONGVIEW	WA	22	250	0.1	Surface Water	EPA (2016)
TREE TOP INC WENATCHEE PLANT	WENATCHEE	WA	0.01	250	0.00003	Surface Water	EPA (2016)
CAROUSEL CENTER	SYRACUSE	NY	0.001	250	0.000002	Surface Water	EPA (2016)

2.2.3 Summary of Water Release Assessment

EPA found that most of the facilities reporting water releases to TRI and DMR could be classified into scenarios associated with conditions of use of methylene chloride. Magnitudes of releases of methylene chloride to water can vary highly (e.g., orders of magnitude) within most scenarios, ranging from 0.0002 to 115,059 kg/site-yr, likely due to site-specific processes and handling of methylene chloride. Some of the largest releases reported are associated with the Waste Handling, Disposal, Treatment, and Recycling; and Processing - incorporation into formulation, mixture, or reaction product scenarios. Data or information and methods needed to estimate releases were not found for Adhesives and Sealants, Paints and Coatings, Aerosol Degreasing/ Lubricants, Batch Open-Top Vapor Degreasing, Conveyorized Vapor Degreasing,

Cold Cleaning, Adhesive and Caulk Removers, Fabric Finishing, Laboratory Use, Non-Aerosol Industrial and Commercial Use scenarios. While some sites in some of these scenarios without quantitative water release estimates may have water releases, it is reasonable to assume that such water releases would be less than most releases reported to TRI and DMR, which are expected to have the highest volumes and releases of methylene chloride. A table of facilities for all scenarios is in Appendix E. Uncertainties are discussed in Key Assumptions and Uncertainties in the Environmental Exposure Assessment Section 4.4.1.

2.3 Environmental Exposures

2.3.1 Environmental Exposures Approach and Methodology

The environmental exposure characterization focuses on aquatic releases of methylene chloride from facilities that use, manufacture, or process methylene chloride under industrial and/or commercial conditions of use. To characterize environmental exposure, EPA assessed point estimate exposures derived from both measured and predicted concentrations of methylene chloride in surface water in the U.S. Measured surface water concentrations were obtained from EPA's Water Quality Exchange (WQX) using the Water Quality Portal (WQP) tool, which is the nation's largest source of water quality monitoring data and includes results from EPA's STORage and RETrieval (STORET) Data Warehouse, the United States Geological Service (USGS) National Water Information System (NWIS), and other federal, state, and tribal sources. A literature search was also conducted to identify other peer-reviewed or grey literature¹⁰ sources of measured surface water concentrations in the U.S., however, no data were found after 2000. Predicted surface water concentrations were modeled for facility releases as detailed in Section 2.2 for reporting year 2016, as determined from EPA's TRI and from DMR; through EPA's Water Pollutant Loading Tool). The aquatic modeling was conducted with EPA's Exposure and Fate Assessment Screening Tool, version 2014 (E-FAST 2014) ([EPA, 2007](#)), using reported annual release/loading amounts (kg/yr) and estimates of the number of days/yr that the annual load is released (see Section 2.2 for more information). As appropriate, two scenarios were modeled per release: release of the annual load over an estimated maximum number of operating days/yr and over only 20 days/yr. Twenty days of release was modeled as the low-end release frequency at which possible ecologic risk from chronic exposure could be determined. The 20-day risk from chronic exposure criterion is derived from partial life cycle tests (e.g., daphnid chronic and fish early life stage tests) that typically range from 21 to 28 days in duration. Additionally, the Probabilistic Dilution Model (PDM), a module of E-FAST 2014 was run to predict the number of days a stream concentration will exceed the designated concentration of concern (COC) value. The measured concentrations reflect localized ambient exposures at the monitoring sites, and the modeled concentrations reflect near-site estimates at the point of release. A geospatial analysis at the subbasin and subwatershed level (Hydrologic Unit Code (HUC)-8 and HUC-12 level respectively) was conducted to compare the measured and predicted surface water concentrations from known facility releases and investigate if the facility releases

¹⁰ Gray literature refers to sources of scientific information that are not formally published and distributed in peer reviewed journal articles. These references are still valuable and consulted in the TSCA risk evaluation process. Examples of grey literature are theses and dissertations, technical reports, guideline studies, conference proceedings, publicly-available industry reports, unpublished industry data, trade association resources, and government reports. ([ENREF 388](#))

may be associated with the observed concentrations in surface water. Hydrologic Unit Codes are a geographically hierarchical tiered approach to organizing stream networks across the United States from regions to subwatersheds and part of the Watershed Boundary Dataset developed by U.S. Geological Survey and U.S. Department of Agriculture ([USGS, 2013](#)). HUC-8 and HUC-12 sized units were selected as relevant sized units as they were expected to give a representative geographic size range over which potentially collocated predicted SWCs from known facility releases and measured SWCs would be spatially relevant.

2.3.1.1 Methodology for Obtaining Measured Surface Water Concentrations

To characterize environmental exposure in ambient water for methylene chloride, EPA used two approaches to obtain measured surface water concentrations. One approach was to pull monitoring data on surface water concentrations from the WQP, and the second was to conduct a systematic review of surface water concentrations in peer reviewed and gray literature.

The primary source of ambient surface water monitoring data was the WQP, which integrates publicly available U.S. water quality data from multiple databases: 1) USGS NWIS, 2) STORET, and 3) the USDA ARS Sustaining The Earth's Watersheds - Agricultural Research Database System (STEWARDS). For methylene chloride, the data retrieved originated from the NWIS and STORET databases. NWIS is the Nation's principal repository of water resources data USGS collects from over 1.5 million sites, including sites from the National Water-Quality Assessment (NAWQA). STORET refers to an electronic data system originally created by EPA in the 1960's to compile water quality monitoring data. NWIS and STORET now use common web services, allowing data to be published through WQP tool. The WQP tool and User Guide is accessed from the following website: (<http://www.waterqualitydata.us/portal.jsp>).

Surface water data for methylene chloride were downloaded from the WQP on October 3, 2018. The WQP can be searched through three different search options: Location Parameters, Site Parameters, and Sampling Parameters. The methylene chloride data were queried through the Sampling Parameters search using the Characteristics parameter (selected "Methylene Chloride (NWIS, STORET)") and Date Range parameter (selected "01-01-2013 to 12-31-2017"). Both the "Site data only" and "Sample results (physical/chemical metadata)" were selected for download in "MS Excel 2007+" format. The "Site data only" file contains monitoring site information (i.e., location in hydrologic cycle, HUC and geographic coordinates); whereas the "Sample result" file contains the sample collection data and analytical results for individual samples.

The "Site data only" and "Sample results (physical/chemical metadata)" files were linked together using the common field "Monitoring Location Identifier" and then filtered and cleansed to obtain surface water samples for years 2013 through 2017. Specifically, cleansing focused on obtaining samples that were only for the media of interest (i.e., surface water), were not quality control (QC) samples (i.e., field blanks), were of high analytical quality (i.e., no QC issues, sample contamination, or estimated values), and were not associated with contaminated sites (i.e., Superfund).

Following filtering to obtain the final dataset, additional domains were examined to identify samples with non-detect concentrations. All non-detect samples were tagged and the concentrations were converted to ½ the reported detection limit for summary calculation

purposes. If a detection limit was not provided, calculations were performed using the average of the reported detection limits in all samples (calculated as 1.46 µg/L).

In addition to using data from WQP, EPA conducted a full systematic review of published literature to identify studies reporting concentrations of methylene chloride in surface water associated with background levels of contamination or potential releases from facilities that manufacture, process, use and/or dispose of methylene chloride in the U.S. Studies clearly associated with releases from Superfund sites, improper disposal methods, and landfills were considered out of scope due to being regulated under other environmental statutes administered by EPA and excluded from data evaluation and extraction. The systematic review process is described in detail in Section 1.5. A total of seven surface water studies were extracted and the results are summarized in Section 2.3.2.1. No concentration data from the U.S. was identified prior to 2000.

2.3.1.2 Methodology for Modeling Surface Water Concentrations from Facility Releases (E-FAST 2014)

Surface water concentrations resulting from wastewater releases of methylene chloride from facilities that use, manufacture, or process methylene chloride were modeled using EPA's E-FAST, Version 2014 ([EPA, 2007](#)). E-FAST 2014 is a model that estimates chemical concentrations in water to which aquatic life may be exposed using upper percentile and/or mean exposure parametric values, resulting in possible conservative exposure estimates. Other assumptions and uncertainties in the model, including ways it may be underestimating or overestimating exposure, are discussed in the Sections 4.4.1 and 4.4.6. Advantages to this model are that it requires minimal input parameters and it has undergone extensive peer review by experts outside of EPA. A brief description of the calculations performed within the tool, as well as a description of required inputs and the methodology to obtaining and using inputs specific to this assessment is described in Section 2.3.2.1. To obtain more detailed information on the E-FAST 2014 tool from the user guide/background document, visit this web address: <https://www.epa.gov/tsca-screening-tools/e-fast-exposure-and-fate-assessment-screening-tool-version-2014/>. All model runs for this assessment were conducted between December 2018 and June 2019.

In some ways the E-FAST estimates are underestimating exposure, because data used in E-FAST include TRI and DMR data, and TRI does not include smaller facilities with fewer than 10 full time employees, nor does it cover certain sectors, such as dry cleaners, or oil and gas extraction. In some ways the E-FAST estimates are overestimating exposure, because methylene chloride is a volatile chemical, but E-FAST doesn't take volatilization into consideration; and, for static water bodies, E-FAST doesn't take dilution into consideration.

2.3.1.2.1 E-FAST Calculations

Surface Water Concentrations

EPA used E-FAST 2014 to estimate site-specific surface water concentrations for discharges to both free-flowing water bodies (i.e., rivers and streams) and for still water bodies (i.e., bays, lakes, and estuaries).

For free-flowing water body assessments, E-FAST 2014 calculates surface water concentrations for four streamflow conditions (7Q10, harmonic mean, 30Q5, and 1Q10 flows) using the following equation:

$$SWC = \frac{WWR \times CF1 \times \left(1 - \frac{WWT}{100}\right)}{SF \times CF2} \quad (\text{Eq. 2-1})$$

where:

SWC	=	Surface water concentration (parts per billion (ppb) or $\mu\text{g/L}$)
WWR	=	Chemical release to wastewater (kg/day)
WWT	=	Removal from wastewater treatment (%)
SF	=	Estimated flow of the receiving stream (million liters/day (MLD))
CF1	=	Conversion factor ($10^9 \mu\text{g/kg}$)
CF2	=	Conversion factor (10^6 L/day/MLD)

For still water body assessments, no simple streamflow value represents dilution in these types of water bodies. As such, E-FAST 2014 accounts for dilution by incorporating an acute or chronic dilution factor for the water body of interest instead of stream flows. Dilution factors in E-FAST 2014 are typically 1 (representing no dilution) to 200, based on NPDES permits or regulatory policy. The following equation is used to calculate surface water concentrations in still water bodies:

$$SWC = \frac{WWR \times \left(1 - \frac{WWT}{100}\right) \times CF1}{PF \times CF2 \times DF} \quad (\text{Eq. 2-2})$$

where:

SWC	=	Surface water concentration (ppb or $\mu\text{g/L}$)
WWR	=	Chemical release to wastewater (kg/day)
WWT	=	Removal from wastewater treatment (%)
PF	=	Effluent flow of the discharging facility (MLD)
DF	=	Acute or chronic dilution factor (DF) used for the water body
	(typically	between 1 and 200)
CF1	=	Conversion factor ($10^9 \mu\text{g/kg}$)
CF2	=	Conversion factor (10^6 L/day/MLD)

Outputs

There are two main outputs from E-FAST that EPA used in characterizing environmental exposures: surface water concentration estimates, and the number of days a certain surface water concentration was exceeded. Site-specific surface water concentration estimates for free-flowing water bodies are reported for the 7Q10 stream flows. The 7Q10 stream flow is the lowest consecutive 7-day average flow during any 10-year period. Site-specific surface water concentration estimates for still water bodies are reported for calculations using the acute dilution factors. In cases where site-specific flow/dilution data were not available, the releases were modeled using stream flows of a representative industry sector, as calculated from all facilities assigned to the industry sector in the E-FAST database (discussed below). Estimates from this calculation method are reported for the 10th percentile 7Q10 stream flows.

The PDM portion of E-FAST 2014 was also run for free-flowing water bodies. The PDM predicts the number of days/yr a chemical's COC in an ambient water body will be exceeded. COCs are threshold concentrations below which adverse effects on aquatic life are expected to be minimal. The model is based on a simple mass balance approach presented by ([Di Toro, 1984](#)) that uses probability distributions as inputs to reflect that streams follow a highly variable seasonal flow pattern and there are numerous variables in a manufacturing process that can affect the chemical concentration and flow rate of the effluent. PDM does not estimate exceedances for chemicals discharged to still waters, such as lakes, bays, or estuaries. For these water bodies, the days of exceedance is assumed to be zero unless the predicted surface water concentration exceeds the COC. In these cases, the days of exceedance is set to the number of release days/yr (see required inputs in 2.3.1.2.2).

2.3.1.2.2 Model Inputs

Individual model inputs and accompanying considerations for the surface water modeling are described in this section.

Chemical Release to Wastewater (WWR)

Annual wastewater loading estimates (kg/site/year or lb/site/year) were obtained from 2016 TRI and 2016 DMR, as discussed in Section 2.2. To model these releases within E-FAST 2014, the annual release is converted to a daily release using an estimated days of release per year. Below is an example calculation:

$$\text{WWR (kg/day)} = \text{Annual loading (kg/site/year)} * \text{Days released per year (days/year)} \quad (\text{Eq. 2-3})$$

In cases where the total annual release amount from one facility was discharged via multiple mechanisms (i.e., direct to surface water and/or indirectly through one or more WWTPs), the annual release amount was divided accordingly based on reported information in TRI (Form R).

Release Days (days/yr)

The number of days/yr that the chemical is discharged is used to calculate a daily release amount from annual loading estimates (see above). Current regulations do not require facilities to report the number of days associated with reported releases. Therefore, two release scenarios were modeled for direct discharging facilities to provide upper and lower bounds for the range of surface water concentrations predicted by E-FAST 2014. The two scenarios modeled are a maximum release frequency (250 to 365 days) based on estimates specific to the facility's condition of use (see Section 2.2.1 for more details) and a low-end release frequency of 20 days of release per year as an estimate of releases that could lead to risk from chronic exposure. The 20-day risk from chronic exposure criterion is derived from partial life cycle tests (e.g., daphnid chronic and fish early life stage tests) that typically range from 21 to 28 days in duration. For indirect dischargers, only the maximum estimated days of release per year was modeled because it was assumed that the actual release to surface water would mostly occur at receiving treatment facilities, which were assumed to typically operate greater than 20 days/yr.

Removal from Wastewater Treatment (WWT%)

The WWT% is the percentage of the chemical removed from wastewater during treatment before discharge to a body of water. As discussed in Section 2.1, the WWT% for methylene chloride was estimated as 57% using the "STP" module within EPI Suite™, which was run using default

settings to evaluate the potential for methylene chloride to volatilize to air or sorb to sludge during wastewater treatment. The WWT% of 54% was applied to releases from indirect discharging facilities because the releases are transferred off-site for treatment at a WWTP prior to discharge to surface water. A WWT% of zero was used for direct releasing facilities because the release reported in TRI and DMR already accounts for any wastewater treatment which may have occurred.

Facility or Industry Sector

The required site-specific stream flow or dilution factor information for a given facility is contained in the E-FAST 2014 database and is selected by searching by a facility's NPDES permit number, name, or the known discharging waterbody reach code. For facilities that directly discharge to surface water (i.e., "direct dischargers"), the NPDES code of the direct discharger was selected from the database. For facilities that indirectly discharge to surface water (i.e., "indirect dischargers" because the release is sent to a WWTP prior to discharge to surface water), the NPDES of the receiving WWTP was selected. The receiving facility name and location was obtained from the TRI database (Form R), if available. As TRI does not contain the NPDES code of receiving facilities, the NPDES was obtained using EPA's EnviroFacts search tool (<https://www3.epa.gov/enviro/facts/multisystem.html>). If a facility NPDES was not available in the E-FAST-2014 database, the release was modeled using water body data for a surrogate NPDES code (preferred) or an industry sector, as described below.

Surrogate NPDES: In cases where the site-specific NPDES code was not available in the E-FAST 2014 database, the preferred alternative was to select the NPDES for a nearby facility that discharges to the same waterbody. The surrogate NPDES was chosen to best represent flow conditions in the waterbody that both the methylene chloride releasing facility and surrogate facility discharge to and not actual releases associated with the surrogate facility NPDES.

Industry Sector (SIC Code Option): If the NPDES code is unknown, no close analog could be identified, or the exact location of a chemical loading is unknown, surface water concentrations were modeled using the "SIC Code Option" within E-FAST 2014. This option uses the 10th and 50th percentile receiving 7Q10 stream flows for dischargers in a given industry sector, as defined by the SIC codes of the industry. The industrial activity associated with the SIC or alternatively the NAICS of the facility in question was examined to select the most representative industry sector for modeling in E-FAST 2014.

2.3.1.3 Methodology for Geospatial Analysis of Measured Surface Water Monitoring and Modeled Facility Releases

Using 2016 data, the measured surface water concentrations from the WQP and predicted concentrations from the modeled facility releases were mapped in ArcGIS Version 10.6 to conduct a watershed analysis at the HUC-8 and HUC-12 level (these results are shown in Section 2.3.2.3 in Figure 2-6 through Figure 2-8). The purpose of the analysis was to identify if any of the observed surface water concentrations could be attributable to the modeled facility releases. In addition, the analysis included a search for Superfund sites within 1 to 5 miles of the surface water monitoring stations.

The locations of the monitoring stations were determined from the geographic coordinates (latitude and longitude) provided in WQP. Location of releases from facilities were located based

on the geographic coordinates for the NPDES, TRI, and/or Facility Registry Service Identification (FRS ID) of the mapped facility, as provided by FRS. For indirect dischargers, the location of the receiving facility was mapped if known. If the receiving facility was not known, the location of the indirect discharger was mapped. Superfund sites in 2016 were identified and mapped using geographic coordinates as reported in the Superfund Enterprise Management System (SEMS) database in EnviroFacts (<https://www.epa.gov/enviro/sems-search>).

A U.S. scale map was developed to provide a spatial representation of the measured concentrations from monitoring and predicted instream concentrations from discharging facilities (Section 2.3.2.3). HUC-8s or HUC-12s with co-located monitoring stations and facility releases were identified and examined further through development of localized maps at the HUC scale.

2.3.2 Environmental Exposure Results

2.3.2.1 Measured Surface Water Concentrations

Measured Surface Water Concentrations from WQX/WQP

The original dataset downloaded contained 29,084 entries for sample years 2013 through 2017. Following the filtering and cleansing procedure, only 8% of the samples remained (n = 2,286 for 2013-2017). The majority of the samples were removed because they were an off-topic media (i.e., groundwater, artificial, bulk deposition, leachate, municipal waste, or stormwater) or location type (i.e., landfill, seep, spring, or well). Those media and locations deemed off-topic are discussed more fully in Section 1 and ([U.S. EPA, 2018c](#)). Of the surface water samples that were removed, ~99% were QC samples (field or laboratory blanks, spikes, or replicates). Other samples were removed because of monitoring conducted at a Superfund site (i.e., Palermo Wellfield Superfund Site) or QC issues.

For the 2016 final dataset (n = 471 samples), observations were made in 10 states (AZ, KS, MN, MO, NJ, NM, NC, PA, TN, TX) at 109 unique monitoring sites, with 1 to 47 samples collected per site. On a watershed level, observations were made in 44 HUC-8 areas and 98 HUC-12 areas. The majority of HUCs had only one monitoring site (55% for HUC-8; 93% for HUC-12). Up to 12 sites were present in an HUC-8 and up to 4 sites in an HUC-12. A list of individual HUCs, including the number of monitoring sites and samples in each HUC, is provided in Table_Apx E-1 for HUC-8 and Table_Apx E-2 for HUC-12. For geospatial representation of these measured samples see Figure 2-2 to Figure 2-5.

A summary of the WQX data obtained from the WQP is provided in Table 2-16 below for years 2013-2017. Per year, the final evaluated datasets contained between 52 and 797 surface water samples collected from 28 to 116 unique monitoring stations. Detection frequencies were low, ranging from 1.1 to 5.1%. Concentrations ranged from not detected (ND; <0.04-10) to 2.5 µg/L in 2013, ND (<0.04-5) to 1.2 µg/L in 2014, ND (<0.04-4) to 0.5 µg/L in 2015, ND (<0.04-5) to 29 µg/L in 2016, and ND (<0.04-5) to 0.61 µg/L in 2017. Non detect values are reported as a range because of differences in reported detection limits in measured samples due to likely differences in sampling routine, methodology, and precision in available analysis tools. The highest measured value was observed in 2016; however, caution should be used in interpreting

trends with this data due to the small number of samples and the lack of samples collected from the same sites over multiple years.

Table 2-16. Measured Concentrations of Methylene Chloride in Surface Water Obtained from the Water Quality Portal (WQP): 2013-2017^a

Year	Detection Frequency	Concentration in All Samples (µg/L)			Concentrations (µg/L) in Only Samples Above the Detection Limit		
		No. of Samples (No. of Unique Stations)	Range ^b	Average ± Standard Deviation (SD) ^c	No. of Samples (No. of Unique Stations)	Range	Average ± SD ^c
2013	5.1%	797 (166)	ND (<0.04-10) to 2.5	1.38 ± 2.0	41 (26)	0.5 to 2.5	0.57 ± 0.33
2014	1.8%	611 (157)	ND (<0.04-5) to 1.2	0.34 ± 0.32	11 (11)	0.13 to 1.2	0.53 ± 0.29
2015	1.1%	355 (94)	ND (<0.04-4) to 0.5	0.43 ± 0.21	4 (2)	0.04 to 0.07	0.05 ± 0.02
2016	1.1%	471 (109)	ND (<0.04-5) to 29	0.61 ± 1.9	5 (3)	1.2 to 29	13.1 ± 14.6
2017	1.9%	52 (28)	ND (<0.04-5) to 0.61	0.59 ± 1.0	1 (1)	0.61	0.61
All 5 Years	2.7%	2,286 (389)	ND (<0.04-10) to 29	0.78 ± 1.5	62 (42)	0.04 to 29	1.54 ± 5.10

- a. Data were downloaded from the WQP (www.waterqualitydata.us) on 10/3/2018. NWIS and STORET surface water data were obtained by selecting “Methylene chloride (NWIS, STORET)” for the Characteristic and selecting for surface water media and locations only. Results were reviewed and filtered to obtain a cleansed dataset (i.e., samples/sites were eliminated if identified as estimated, QC, media type other than surface water, Superfund, landfill, failed laboratory QC, etc.).
- b. ND = Not Detected. Reported detection limits in all samples ranged from 0.04 to 10 µg/L.
- c. Calculations were performed using ½ the reported detection limit when results were reported as not detected. If a detection limit was not provided, calculations were performed using the average of the reported detection limits in all samples (1.46 µg/L).

The quantitative environmental assessment used the 2016 data set only to allow direct comparison with known TRI and DMR releasers from the same year. For the 2016 data, only 5 samples from 3 monitoring sites (all in North Carolina) had methylene chloride concentrations above the detection limit, as shown in Table 2-17. The average of these samples was 13.1 µg/L. It should be noted that two of the sites (Clinton, NC and Mills River, NC) each had two samples collected on the same day within 5-15 minutes (min) of each other. Both samples had identical measured concentrations: 1.2 µg/L in Clinton, NC and 29 µg/L in Mills River, NC. The last site (Asheville, NC) had a concentration of 5 µg/L in one sample. No samples were collected at these three sites in other years between 2013 and 2017.

A detailed summary of results for all samples collected between 2013 and 2017 with concentrations above the detection limit is provided in Table_Apx E-3.

Table 2-17. Sample Information for Water Quality Exchange (WQX) Surface Water Observations With Concentrations Above the Reported Detection Limit: Year 2016^a

Monitoring Site Information				Sample Information		
Monitoring Site ID and Organization	Waterbody Type and Location	Lat/Long	HUC 8	Sample ID	Date and Time	Concentration (µg/L) ^b
21NC03WQ-B8484000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	River/Stream BEARSKIN SWAMP AT SR 1325 NR Clinton, NC	35.08754/ -78.43463	3030006	21NC03WQ- AMS20161206- B8484000- 370870277	2016-12-06 11:40:00 EST	1.2
				21NC03WQ- AMS20161206- B8484000- 381057619	2016-12-06 11:55:00 EST	1.2
21NC03WQ-E1485000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	River/Stream North Mills River at SR 1343 (River Loop Rd) nr Mills River, NC	35.39412/ -82.61646	6010105	21NC03WQ- AMS20160822- E1485000- 381059366	2016-08-22 15:55:00 EST	29
				21NC03WQ- AMS20160822 -E1485000- 381059612	2016-08-22 16:00:00 EST	29
21NC03WQ-E3475000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	River/Stream Hominy Creek at Pond Rd in Asheville, NC ^c	35.54683/ -82.60264	6010105	21NC03WQ- RAMS20160817- E3475000- 370533933	2016-08-17 17:05:00 EST	5

a. Data were downloaded from the WQP (www.waterqualitydata.us) on 10/3/2018. NWIS and STORET surface water data were obtained by selecting "Methylene chloride (NWIS, STORET)" for the Characteristic and selecting for surface water media and locations only. Results were reviewed and filtered to obtain a cleansed dataset (i.e., samples/sites were eliminated if identified as estimated, QC, media type other than surface water, Superfund, landfill, failed laboratory QC, etc.).

Measured Concentrations in Published Literature

Using systematic review, the published literature yielded only a minimal amount of surface water monitoring data for methylene chloride; a summary of the individual studies is provided in Table 2-18. Only two U.S. studies were identified. In one, a USGS nation-wide random survey of rivers and reservoirs used for drinking water sources, methylene chloride was detected at 2.6 µg/L in one out of 375 samples collected between 1999 and 2000 (detection limit of 0.2 µg/L) ([USGS, 2003](#)). In the other U.S. study, conducted in 1979-1981, methylene chloride was detected in 93% of samples collected from the Eastern Pacific Ocean ([Singh et al., 1983](#)). Concentrations ranged from below the detection limit (<0.0004) to 0.008 µg/L, with a mean of 0.0031 µg/L (n=30). No U.S. monitoring data were identified for year 2016.

The systematic review approach also identified data from various other countries and regions, including Brazil, China, Japan, France, and Europe ([Bianchi et al., 2017](#); [Ma et al., 2014](#); [Christof et al., 2002](#); [Duclos et al., 2000](#); [Yamamoto et al., 1997](#)). Collectively, these studies encompass 332 samples collected between 1993 and 2013 from rivers and estuaries. The

reported methylene chloride concentrations range from below the detection limit to 134 µg/L, with reported central tendency values ranging from 0.0019 to 1.7 µg/L. The highest concentration was from an industrialized area of Osaka, Japan in 1993-1995 with a maximum concentration of 134 µg/L ([Yamamoto et al., 1997](#)). The next highest reported concentrations were in the range of 4.5 to 5 µg/L in industrialized or urban areas of China, France, and Europe (1993-2011).

Table 2-18. Summary of Published Literature with Surface Water Monitoring Data

Country	Site Information	Date Sampled	N (Detection Frequency)	Concentration (µg/L)		Source	Data Quality Score
				Range	Central Tendency ±SD		
North America							
U.S.	Nation-wide; Surface water for drinking water sources (rivers and reservoirs)	1999-2000	375 (0.0027)	ND (<0.2) - 2.6	NR	(USGS, 2003)	Medium
U.S. to Chile	Eastern Pacific Ocean (California, U.S. to Valparaiso, Chile)	1979-1981	30 (0.93)	ND (<0.0004) - 0.008	Mean: 0.0031 ± 0.0032	(Singh et al., 1983)	Medium
Europe and Asia							
Brazil	Santo Antonio da Patrulha, Tres Coroas, and Parobe in the Sinos River Basin; River samples collected from seven points on the three main rivers of the Sinos River Basin	2012-2013	60 (0.72)	ND - 0.0058	Mean: 0.0019	(Bianchi et al., 2017)	Medium
China	Daliao River (n=20 sites), heavily industrialized	2011	20 (0.75)	ND (<0.675) - 4.47	Mean: 0.678	(Ma et al., 2014)	High
Europe	Estuaries of the Scheldt, Thames, Loire, Rhine	1997-1999	73 (1)	0.0003 - 4.98	NR	(Christof et al., 2002)	High
France	Paris; River samples (raw) collected from the River Seine (n=14 stations), River Marne (n=1 station) and River Oise (n=1 station). WWTPs are located on the river.	1994-1995	43 (1)	0.016 - 4.92	Mean: 1.004 ± 1.218; Median: 0.473	(Duclos et al., 2000)	Medium
Japan	Osaka; Rivers and estuaries (30 sites) in industrialized city	1993-1995	136 (NR)	NR - 134	Median: 1.7	(Yamamoto et al., 1997)	High

NR = Not reported

ND = Not detected; detection limit reported in parenthesis if available.

2.3.2.2 E-FAST Modeling Results

Summary

As discussed in Section 2.2, releases of methylene chloride were determined from two data sources (TRI and DMR) for the 2016 calendar year and assigned to 14 TSCA condition of use categories. Overall, 106 releases originating from 22 states were modeled, with the most in California (15%) and New York (12%). The location of the actual releases, when accounting for indirect dischargers, occurred in 21 U.S. states/territories (AL, AZ, CA, CT, GA, ID, IL, IN, KY, LA, MD, MI, MO, NH, NJ, NY, OH, TN, TX, WA, WV). With respect to watersheds, the releases occurred across 74 HUC-8 areas and 87 HUC-12 areas. At the HUC-8 level, approximately three quarters of the HUCs contained only one identified facility release (**73%**), and the remaining HUCs contained 2 to 5 facility releases. Direct and indirect dischargers accounted for 77% and 23% of the total releases modeled, respectively. The majority of the releases were modeled using site-specific NPDES codes (63%); surrogate NPDES codes were used in only 9% of the cases, with the remaining cases (27%) run using a representative industry sector SIC code. For releases modeled with a NPDES code (including a surrogate NPDES), surface water concentrations were calculated for free-flowing water bodies in 82% of the cases, and still water bodies for the remaining cases (18%). A detailed summary table by facility is provided in Table_Apx E-4.

Summary by Occupational Exposure Scenarios (OES)

A summary of the surface water concentration estimates modeled using E-FAST 2014 is summarized by OES category in Table 2-21 for the maximum release scenario and Table 2-20 for the 20-day release scenario. Release estimates are based on reported 2016 releases to TRI and DMR as summarized in Section 2.2.2. For the maximum days of release scenarios, surface water concentrations under 7Q10 flow conditions ranged from 3.5E-07 to 1.8E+04 ppb. For the 20-day release scenarios, surface water concentrations ranged from 4.4E-06 to 5,857 ppb. On a per facility basis, the 20-day release scenario yielded higher surface water concentrations than the maximum day of release scenario.

Table 2-19. Summary of Surface Water Concentrations by Occupational Exposure Scenarios (OES) for Maximum Days of Release Scenario

OES	No. of Releases Modeled	Sum of Annual Releases Modeled (kg/yr)	Annual Release by Facility (kg/site-yr)		Surface Water Concentration (7Q10 Flow) (µg/L)	
			Min	Max	Min	Max
Manufacturing	20	162	8.28E-03	76	1.2E-05	5.0
Import and Repackaging	5	245	2.81E-02	144	5.1E-05	34
Processing as a Reactant	3	238	0.12	213	1.5E-02	0.26
Processing: Formulation	9	6,202	0.23	5,785	2.8E-06	1,659
Polyurethane Foam	1	2.3	2.3	2.3	1.1	1.1
Plastics Manufacturing	9	64	2.3E-02	28	4.2E-05	4.3
CTA Film Manufacturing	1	29	29	29	0.11	0.11

OES	No. of Releases Modeled	Sum of Annual Releases Modeled (kg/yr)	Annual Release by Facility (kg/site-yr)		Surface Water Concentration (7Q10 Flow) (µg/L)	
			Min	Max	Min	Max
Lithographic Printer Cleaner	1	9.3E-04	9.3E-04	9.3E-04	5.4E-05	5.4E-05
Spot Cleaner	1	6.0E-02	6.0E-02	6.0E-02	6.0E-03	6.0E-03
Recycling and Disposal	14	7.8E+04	2.4E-02	7.6E+04	3.9E-03	1.8E+04
Other	12	67	2.4E-04	42	3.5E-07	10
Department of Defense (DoD)	1	0.45	0.45	0.45	1.8E-03	1.8E-03
WWTP	29	5,596	0.11	2,730	7.4E-05	322
Overall	106	9.1E+04	2.3E-04	7.6E+04	3.5E-07	1.8E+04

Table 2-20. Summary of Surface Water Concentrations by Occupational Exposure Summary (OES) for 20 Days of Release Scenario

OES	No. of Releases Modeled	Sum of Annual Releases (kg/yr)	Annual Release by Facility (kg/site-yr)		Surface Water Concentration (7Q10) (ppb)	
			Min	Max	Min	Max
Manufacturing	14	95	8.3E-03	76	2.4E-04	83
Import and Repackaging	2	0.11	2.8E-02	8.6E-02	0.18	0.55
Processing as a Reactant	2	25	0.12	25	2.0	4.6
Processing: Formulation	5	49	0.23	31	8.9E-04	107
Polyurethane Foam	1	2.3	2.3	2.3	14	14
Plastics Manufacturing	9	64	2.3E-02	28	5.3E-04	54
CTA Film Manufacturing	1	29	29	29	1.4	1.4
Lithographic Printer Cleaner	1	9.3E-04	9.3E-04	9.3E-04	6.8E-04	6.8E-04
Spot Cleaner	1	6.0E-02	6.0E-02	6.0E-02	7.5E-02	7.5E-02
Recycling and Disposal	6	7.1	2.4E-02	3.6	0.16	353
Other	10	23	2.4E-04	22	4.4E-06	1.3
DoD	1	0.45	0.45	0.45	2.3E-02	0.02
WWTP	29	5,596	0.11	2,730	1.4E-03	5,857
Overall	82	5,891	2.3E-04	2,730	4.4E-06	5,857

2.3.2.3 Geospatial Analysis

A geospatial analysis at the watershed level (HUC-8 and HUC-12) was conducted to compare the measured and predicted surface water concentrations in 2016 and investigate if the facility releases may be associated with the observed concentrations in surface water. A geographic distribution of the concentrations is shown in Figure 2-2 and Figure 2-3 (east and west U.S.) for the maximum days of release scenario, and in Figure 2-4 and Figure 2-5 (east and west U.S.) for the 20-days of release scenario. Overall, there are 26 U.S. states/territories with either a measured concentration (n=10) or a predicted concentration (n=21); at the watershed level, there

are 116 HUC-8 areas and 184 HUC-12 areas with either measured or predicted concentrations. Table_Apx E-5 provides a list of states/territories with facility releases (as mapped) and/or monitoring sites.

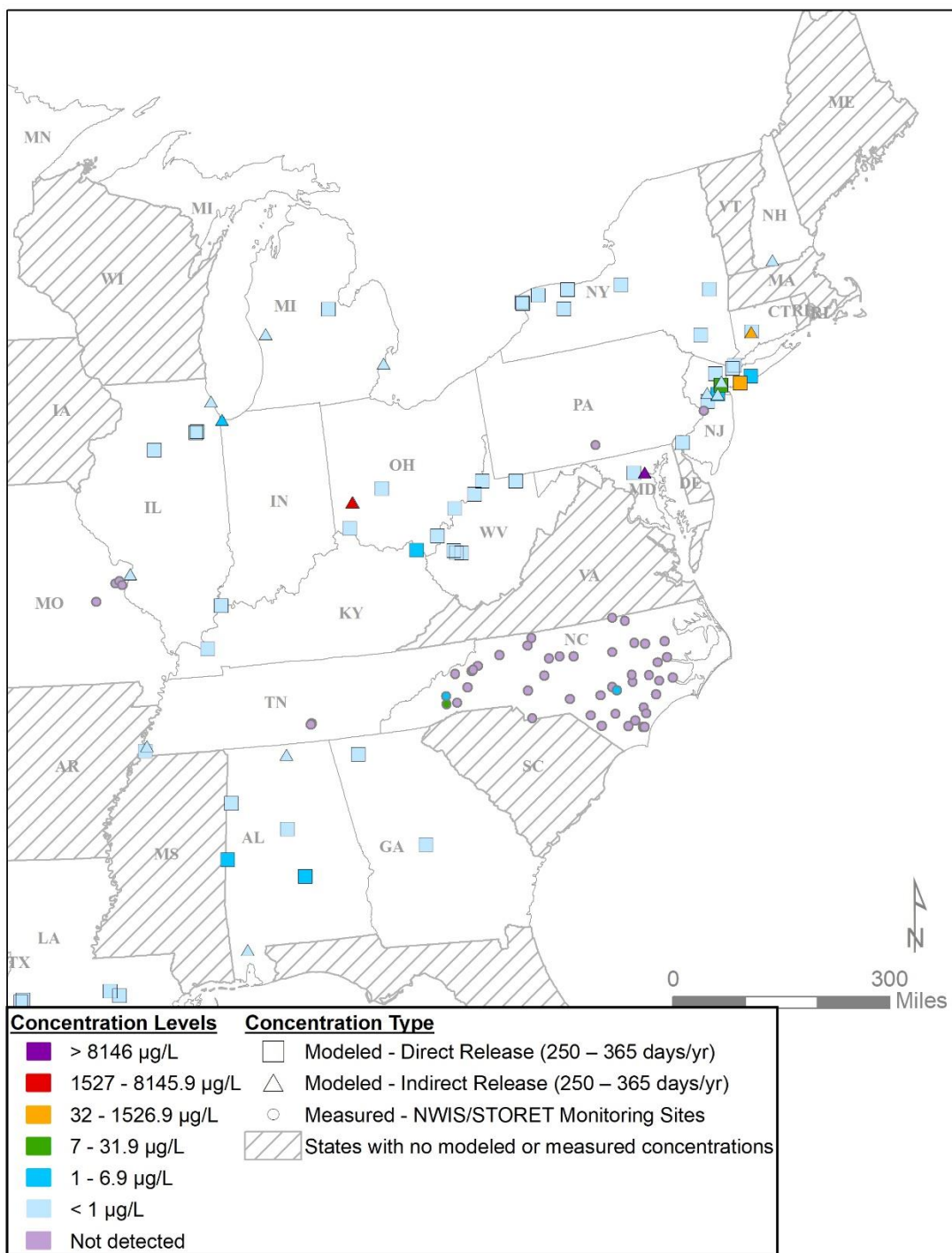


Figure 2-2. Surface Water Concentrations of Methylene Chloride from Releasing Facilities (Maximum Days of Release Scenario) and Water Quality Exchange (WQX) Monitoring Stations: Year 2016, Eastern U.S.

All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.

Puerto Rico and the U.S. Virgin Islands not shown due to no modeled releases or measured monitoring information.

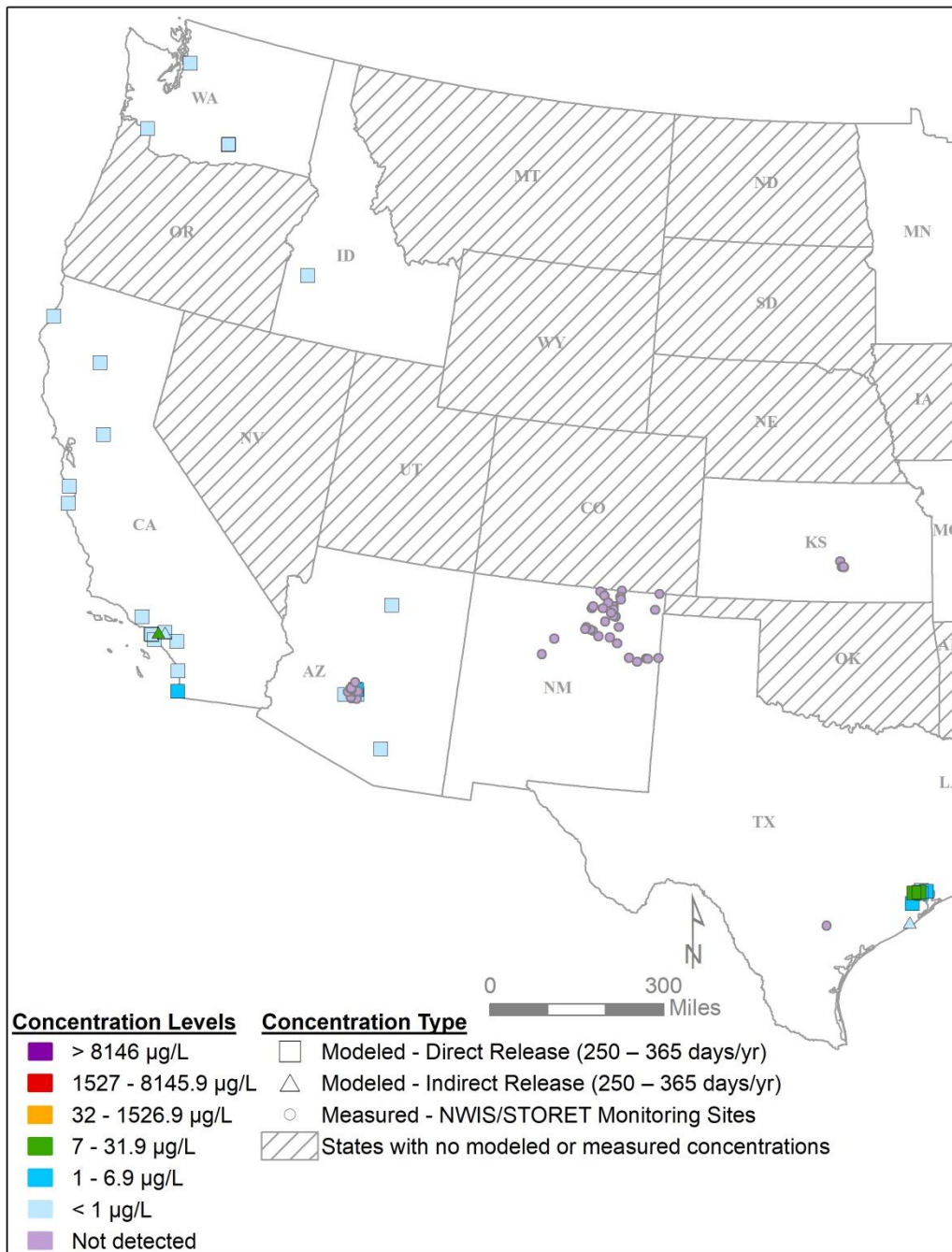


Figure 2-3. Surface Water Concentrations of Methylene Chloride from Releasing Facilities (Maximum Days of Release Scenario) and Water Quality Exchange (WQX) Monitoring Stations: Year 2016, Western U.S.

All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.

Alaska, Hawaii, Guam, N. Mariana Islands and American Somoma not shown due to no modeled releases or measured monitoring information.

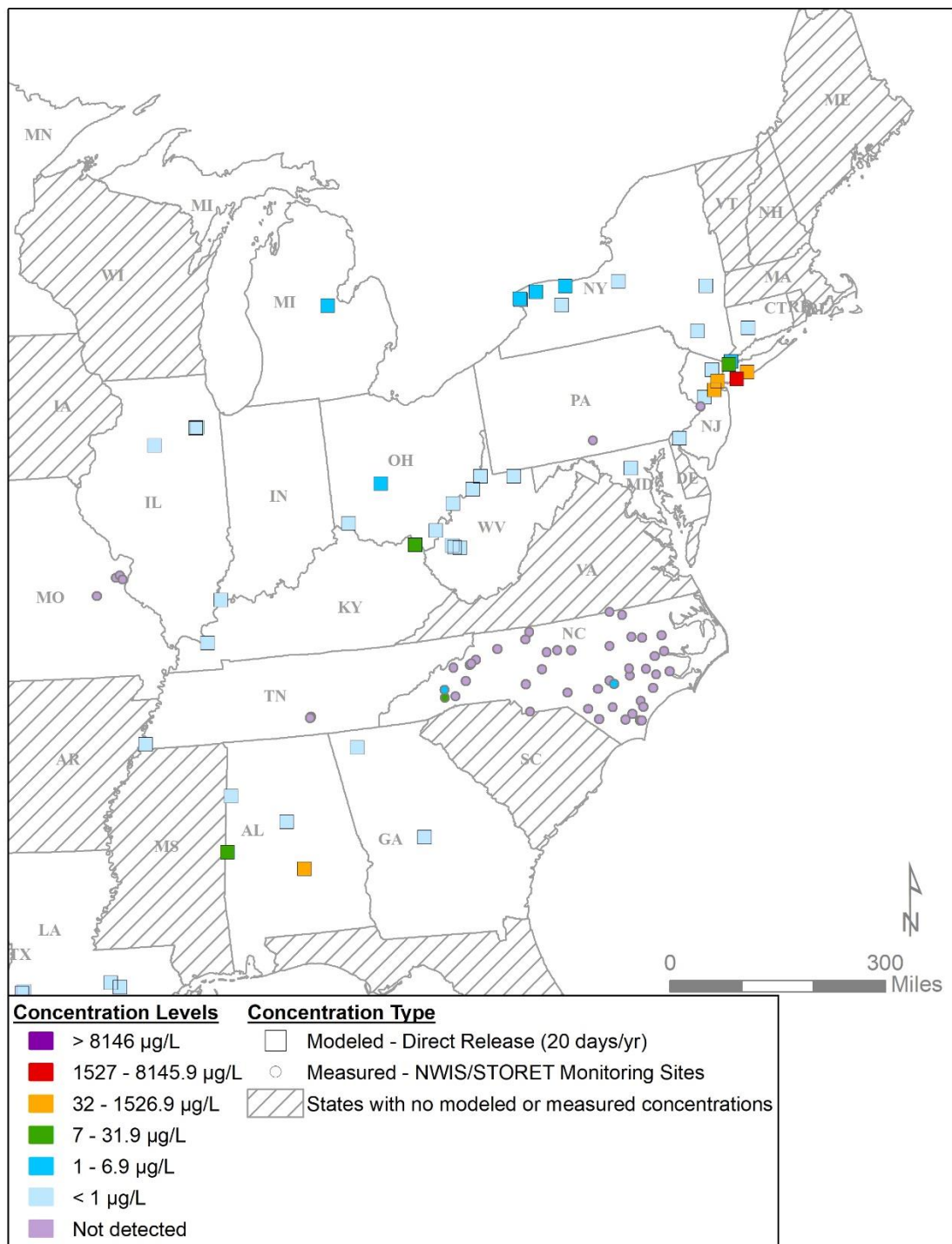


Figure 2-4. Concentrations of Methylene Chloride from Releasing Facilities (20 Days of Release Scenario) and Water Quality Exchange (WQX) Monitoring Stations: Year 2016, East U.S.

Puerto Rico and U.S. Virgin Islands not shown due to no modeled releases or measured monitoring information.

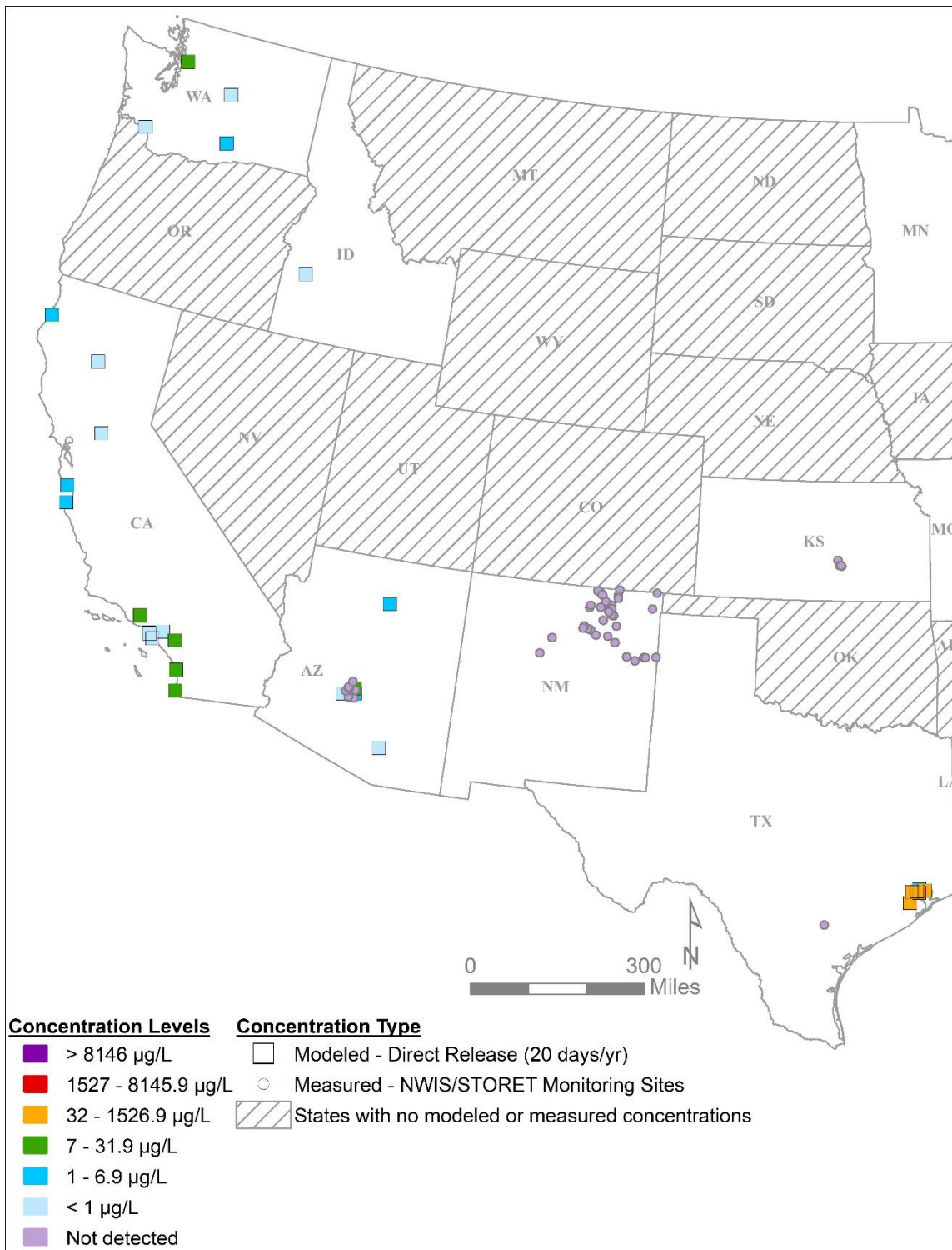


Figure 2-5. Concentrations of Methylene Chloride from Releasing Facilities (20 Days of Release Scenario) and Water Quality Exchange (WQX) Monitoring Stations: Year 2016, West U.S.

Alaska, Hawaii, Guam, N. Mariana Islands and American Somoa not shown due to no modeled releases or measured monitoring information.

Superfund Analysis

An analysis of the 2016 dataset was conducted to determine if any monitoring stations may be associated with nearby Superfund sites that may potentially contain methylene chloride releases, and thus would not fall under the scope of this TSCA evaluation. In the dataset, six surface water monitoring stations were within 1 mile of one or more Superfund sites in SEMS. Overall, 12 Superfund sites were identified, although only one of the 12 Superfund sites is on the National Priority List (NPL), the others are identified as Non-NPL. All measured surface water concentrations at the six monitoring sites were below the detection limit. For monitoring stations that had detectable concentrations in 2016, the search was expanded to 5 miles. Sample 21NC03WQ-E3475000, located at Hominy Creek at Pond Rd in Asheville, NC, met this criterion. However, the monitoring station is located on a separate tributary to the French Broad River and its catchment does not include the Superfund site. Therefore, no monitoring stations were removed from the geospatial analysis based on proximity to Superfund sites.

Co-location of Methylene Chloride Releasing Facilities and Monitoring Stations

The co-occurrence of methylene chloride releasing facilities and monitoring stations in a HUC is shown in Figure 2-6. There are two adjacent HUC-8 areas (and one HUC-12) in Arizona that have both measured and predicted concentrations. The associated facility and monitoring site information are provided in Table 2-21. HUC 15070102 (Aqua Fria), has three direct releasing facilities with modeled 7Q10 SWCs ranging from 0.11 to 7.99 $\mu\text{g/L}$, and 7 monitoring stations all with concentration less than the reported detection limit (0.8 to 5 $\mu\text{g/L}$). Three of the monitoring sites were 7.5 to 15.8 miles downstream of two facilities, the remaining monitoring sites were neither up or downstream of facilities. HUC 15060106 (Lower Salt), has one direct releasing facility with modeled 7Q10 SWCs ranging from 0.13 to 1.95 $\mu\text{g/L}$, and 5 monitoring stations all with concentration less than the reported detection limit (0.8 to 5 $\mu\text{g/L}$).

As the measured concentrations were below the detection limit and the number of samples collected was small, definitive conclusions could not be drawn on possible associations between measured concentrations in surface water and predicted concentrations from facility releases.

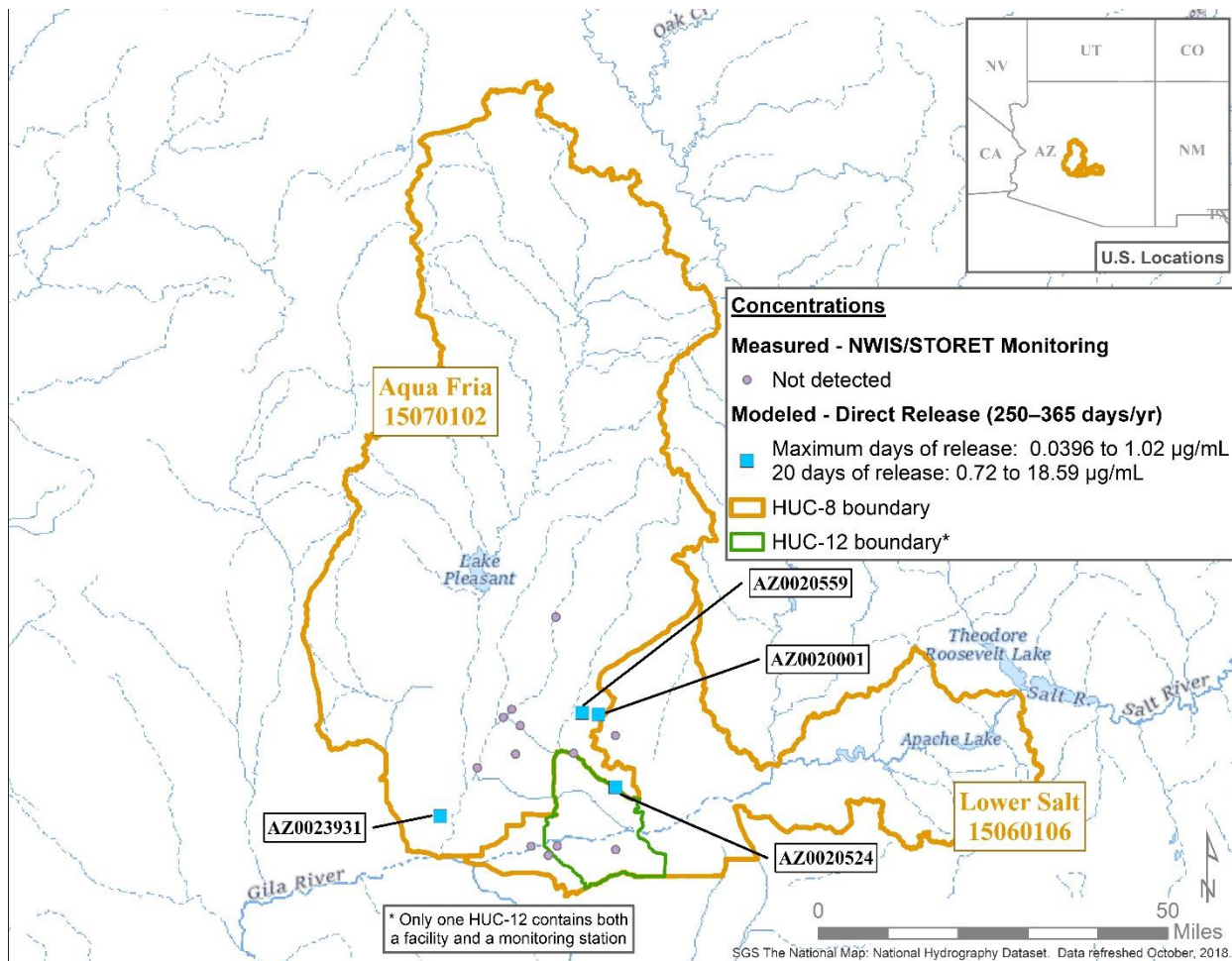


Figure 2-6. Co-location of Methylene Chloride Releasing Facilities and Water Quality Exchange (WQX) Monitoring Stations at the HUC 8 and HUC 12 Level

Table 2-21. Co-Location of Facility Releases and Monitoring Sites within HUC 8 Boundaries (Year 2016)

Facilities in HUC		Monitoring Sites in HUC			
Site	Modeled 7Q10 SWCs ^a (µg/L)	Monitoring Site ID	No. of Samples	Measured Surface Water Concentrations (µg/L)	Location Comments Relative to Facilities ^b
HUC 15070102: Aqua Fria					
<u>3 Direct Releasing Facilities</u>		<u>7 Monitoring Sites</u>			
1. PIMA COUNTY - INA ROAD WWTP; <i>TUCSON, AZ</i> NPDES: AZ0020001	365 days: 1.36* 20 days: 18.59*	USGS-333238112165201	1	ND (< 5)	Downstream of AZ0020001 (14 mi) and AZ0020559 (15.8 mi)
		USGS-333658112113200	1	ND (< 5)	Downstream of AZ0020001 (7.5 mi) and AZ0020559 (9.4 mi)
		USGS-333751112133801	1	ND (< 5)	Downstream of AZ0020001 (9.4 mi) and AZ0020559 (11.4 mi)
2. 23RD AVENUE WWTP; <i>PHOENIX, AZ</i> NPDES: AZ0020559	365 days: 0.26 20 days: 2.49	USGS-09513925	1	ND (< 5)	Upstream or neither up or down stream
		USGS-333407112045401 ^d	3	ND (< 0.3 - < 0.8)	Upstream or neither up or down stream
3. APACHE JUNCTION WWTP <i>APACHE JUNCTION, AZ</i> ; NPDES: AZ0023931	365 days: 0.0387 20 days: 0.72	USGS-333840112123601	1	ND (< 5)	Upstream or neither up or down stream
		USGS-334811112070700	3	ND (< 0.3 - < 4)	Upstream or neither up or down stream
HUC 15060106: Lower Salt					
<u>1 Direct Releasing Facility</u>		<u>5 Monitoring Sites</u>			
1. 91ST AVE WWTP; <i>TOLLESON, AZ</i> NPDES: AZ0020524	365 days: 0.29 20 days: 4.52	USGS-09512403 ^{c, d}	2	ND (< 0.3 - < 0.8)	Neither up or down stream
		USGS-332333112080301	3	ND (< 0.3 - < 0.8)	Neither up or down stream
		USGS-332409111594101 ^{c, d}	2	ND (< 0.3 - < 0.8)	Neither up or down stream
		USGS-332430112101001	2	ND (< 0.3 - < 0.8)	Neither up or down stream
		USGS-333557111594201	3	ND (< 0.3)	Neither up or down stream

a. Concentrations leading to modeled days of exceedance are indicated by an asterisks (*).

b. The number of miles between the facility and monitoring site are based on Euclidean distance.

c. The monitoring sites are also co-located with the facility in the same HUC 12 (150601060306; City of Phoenix-Salt River).

d. The monitoring sites are located within 1.02 to 1.08 miles of Superfund sites.

1.3.1 Co-location of Monitoring Stations and DMR/TRI/CDR/Superfund Sites

Three monitoring sites in the 2016 dataset had detectable concentrations but were not co-located with other identified methylene chloride-releasing facilities. As such these monitoring stations were further characterized by evaluating their location with respect to any DMR (NPDES), TRI, CDR, or Superfund site in 2016 as shown in Figure 2-7 and Figure 2-8.

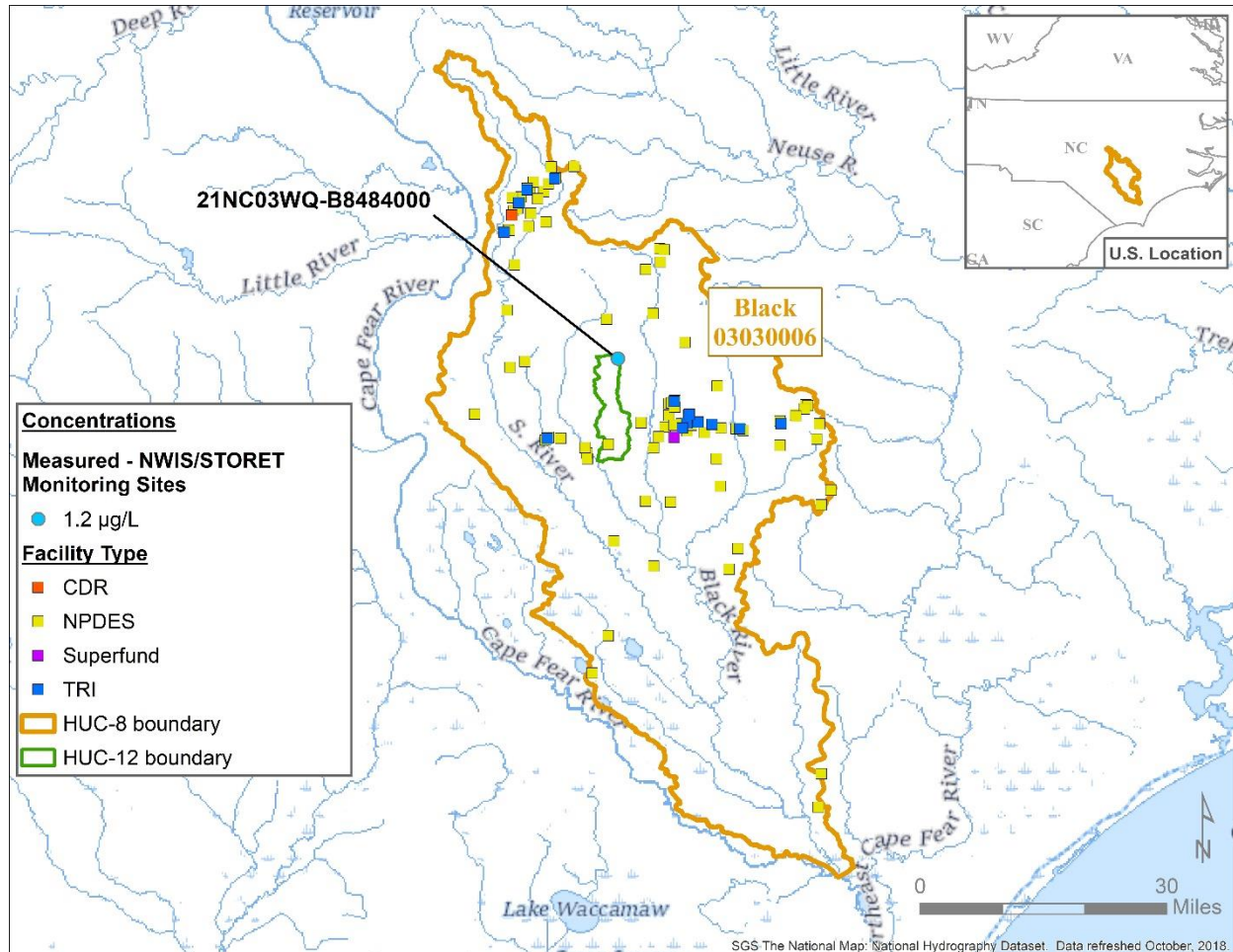


Figure 2-7. Search of CDR, DMR (NPDES), Superfund, and TRI facilities in 2016 within HUC-8 of Water Quality Portal (WQP) Station 21NC03WQ-AMS20161206 -B8484000. Two samples with concentrations of 1.2 ppb were detected at this monitoring site on 2016.

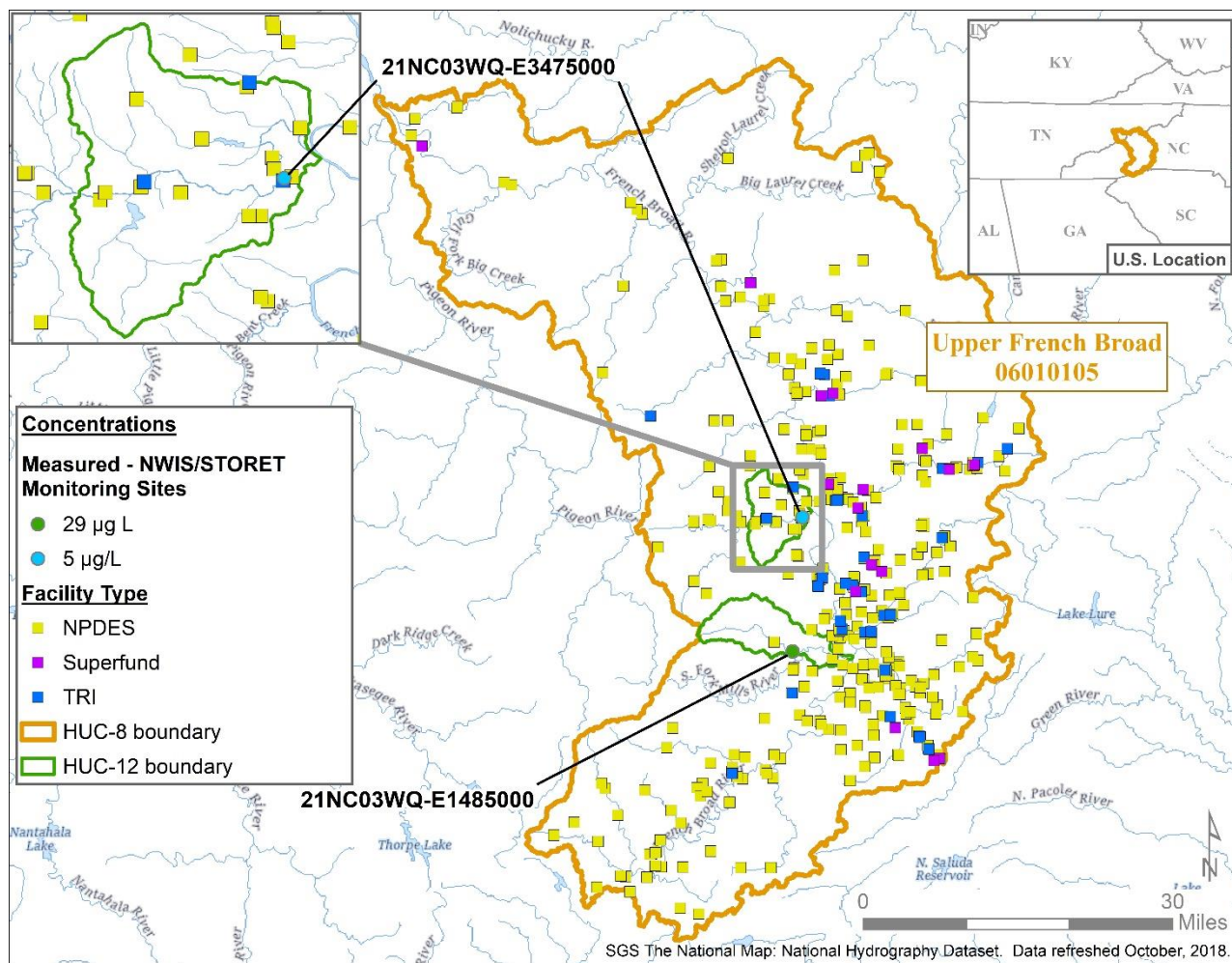


Figure 2-8. Search of CDR, NPDES, Superfund, and TRI facilities in 2016 within HUC-8 of Water Quality Portal (WQP) Stations 21NC03WQ-E1485000 and 21NC03WQ-E3475000. Station 21NC03WQ-E1485000 had two samples with concentrations of 29 ppb and station 21NC03WQ-E3475000 had one sample with concentration of 5 ppb.

2.4 Human Exposures

EPA evaluated acute and chronic exposures to workers and occupational non-users (ONUs) and acute exposures to consumers by dermal and inhalation routes in association with methylene chloride use in industrial, commercial and consumer applications. The assessed conditions of use are described in Table 1-4; however, due to expected similarities in or lack of data to distinguish some conditions of use, both exposures/releases and occupational and consumer exposures for several of the subcategories of use in Table 1-4 were grouped and assessed together during risk evaluation. For example, formulation of paints, coatings, adhesives, sealants, and other product subcategories may generally have similar worker activities, and EPA does not have data to distinguish whether workers are exposed differently for these different formulations. Therefore, EPA has grouped these formulating conditions of use into one occupational scenario. A crosswalk of the conditions of use in Table 1-4 to the occupational and consumer scenarios assessed in this report is provided in Table 2-22 below. It is possible that an individual can fall

into multiple PESS categories. For example, an individual may be exposed as a worker or ONU and also outside of the workplace as a consumer.

Table 2-22. Crosswalk of Conditions of Use to Occupational and Consumer Scenarios Assessed in the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Occupational Scenario	Consumer Scenario
Manufacturing	Domestic manufacturing	Manufacturing	Manufacturing	N/A
	Import	Import	Repackaging	N/A
Processing	Processing as a reactant	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants)	Processing as a Reactant	N/A
		Intermediate for pesticide, fertilizer, and other agricultural chemical manufacturing		
		Petrochemical manufacturing		
		Intermediate for other chemicals		
	Incorporated into formulation, mixture, or reaction product	Solvents (for cleaning or degreasing), including manufacturing of: <ul style="list-style-type: none"> All other basic organic chemical Soap, cleaning compound and toilet preparation 	Processing - Incorporation into Formulation, Mixture, or Reaction Product	N/A
		Solvents (which become part of product formulation or mixture), including manufacturing of: <ul style="list-style-type: none"> All other chemical product and preparation Paints and coatings 		
Propellants and blowing agents for all other chemical product and		N/A		

Life Cycle Stage	Category ^a	Subcategory ^b	Occupational Scenario	Consumer Scenario
		preparation manufacturing		
		Propellants and blowing agents for plastics product manufacturing		
		Paint additives and coating additives not described by other codes		
		Laboratory chemicals for all other chemical product and preparation manufacturing		
		Laboratory chemicals for other industrial sectors		
		Processing aid, not otherwise listed for petrochemical manufacturing		
		Adhesive and sealant chemicals in adhesive manufacturing		
		oil and gas drilling, extraction, and support activities		
	Repackaging	Solvents (which become part of product formulation or mixture) for all other chemical product and preparation manufacturing	Repackaging	N/A
	all other chemical product and preparation manufacturing			
	Recycling	Recycling	Waste Handling, Disposal, Treatment, and Recycling	N/A
Distribution in commerce	Distribution	Distribution	Repackaging	
Industrial, commercial and consumer uses	Solvents (for cleaning or degreasing) ^c	Batch vapor degreaser (e.g., open-top, closed-loop)	Batch Open-Top Vapor Degreasing	N/A
		In-line vapor degreaser (e.g., conveyORIZED, web cleaner)	Conveyorized Vapor Degreasing	N/A
		Cold cleaner	Cold Cleaning	N/A
		Aerosol spray degreaser/cleaner	Commercial Aerosol Products (Aerosol Degreasing, Aerosol	Brake Cleaner, Carbon Remover,

Life Cycle Stage	Category ^a	Subcategory ^b	Occupational Scenario	Consumer Scenario
			Lubricants, Automotive Care Products)	Carburetor Cleaner, Coil Cleaner, Electronics Cleaner, Engine Cleaner, Gasket Remover
	Adhesives and sealants	Single component glues and adhesives and sealants and caulks	Adhesives and Sealants	Adhesives, Sealants
	Paints and coatings including commercial paint and coating removers	Paints and coatings use and paints and coating removers, including furniture refinisher	Paints and Coatings Paint and Coating Removers	Brush Cleaner
		Adhesive/caulk removers	Adhesive and Caulk Removers	
	Metal products not covered elsewhere	Degreasers – aerosol and non-aerosol degreasers and cleaners e.g., coil cleaners	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products) Miscellaneous Non-Aerosol Industrial and Commercial Uses	Carbon Remover, Coil Cleaner, Electronics Cleaner
	Fabric, textile and leather products not covered elsewhere	Textile finishing and impregnating/ surface treatment products e.g., water repellent	Fabric Finishing	N/A
	Automotive care products	Function fluids for air conditioners: refrigerant, treatment, leak sealer	Miscellaneous Non-Aerosol Industrial and Commercial Uses	Automotive Air Conditioning Leak Sealer, Automotive Air Conditioning Refrigerant
		Interior car care – spot remover	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	N/A
	Automotive care products	Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Brake Cleaner, Carburetor Cleaner, Engine Cleaner, Gasket Remover
	Apparel and footwear care products	Post-market waxes and polishes applied to footwear e.g., shoe polish	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	N/A
	Laundry and dishwashing products	Spot remover for apparel and textiles	Spot Cleaning	N/A

Life Cycle Stage	Category ^a	Subcategory ^b	Occupational Scenario	Consumer Scenario
	Lubricants and greases	Liquid and spray lubricants and greases	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products) Miscellaneous Non-Aerosol Industrial and Commercial Uses	Brake Cleaner, Carburetor Cleaner, Engine Cleaner, Gasket Remover
		Degreasers – aerosol and non-aerosol degreasers and cleaners		
	Building/ construction materials not covered elsewhere	Cold pipe insulation	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Cold Pipe Insulation
	Solvents (which become part of product formulation or mixture)	All other chemical product and preparation manufacturing	Processing - Incorporation into Formulation, Mixture, or Reaction Product	N/A
	Processing aid not otherwise listed	In multiple manufacturing sectors ^c	Cellulose Triacetate Film Production	N/A
	Propellants and blowing agents	Flexible polyurethane foam manufacturing	Flexible Polyurethane Foam Manufacturing	N/A
	Arts, crafts and hobby materials	Crafting glue and cement/concrete	N/A	Adhesives
	Other Uses	Laboratory chemicals - all other chemical product and preparation manufacturing	Laboratory Use	N/A
		Electrical equipment, appliance, and component manufacturing	Miscellaneous Non-Aerosol Industrial and Commercial Uses	N/A
		Plastic and rubber products	Plastic Product Manufacturing	N/A
			Cellulose Triacetate Film Production	N/A
		Anti-adhesive agent - anti-spatter welding aerosol	Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Weld Spatter Protectant
		Oil and gas drilling, extraction, and support activities	Miscellaneous Non-Aerosol Industrial and Commercial Uses	N/A
		Toys, playground, and sporting equipment - including novelty articles (toys, gifts, etc.)	Miscellaneous Non-Aerosol Industrial and Commercial Uses	N/A

Life Cycle Stage	Category ^a	Subcategory ^b	Occupational Scenario	Consumer Scenario
		Carbon remover, lithographic printing cleaner, wood floor cleaner, brush cleaner	Lithographic Printing Plate Cleaning Miscellaneous Non-Aerosol Industrial and Commercial Uses	Brush Cleaner, Carbon Remover
Disposal	Disposal	Industrial pre-treatment	Waste Handling, Disposal, Treatment, and Recycling	N/A
		Industrial wastewater treatment		
		Publicly owned treatment works (POTW)		
		Underground injection		
		Municipal landfill		
		Hazardous landfill		
		Other land disposal		
		Municipal waste incinerator		
		Hazardous waste incinerator		
		Off-site waste transfer		

a – These categories of conditions of use appear in the initial life cycle diagram, reflect CDR codes and broadly represent conditions of use for methylene chloride in industrial and/or commercial settings.

b – These subcategories reflect more specific uses of methylene chloride.

c – Reported for the following sectors in the 2016 CDR for manufacturing of: plastic materials and resins, plastics products, miscellaneous, all other chemical product and preparation ([U.S. EPA, 2016](#)).

e – Reported for the following sectors in the 2016 CDR for manufacturing of: petrochemicals, plastic materials and resins, plastics products, miscellaneous and all other chemical products ([U.S. EPA, 2016](#)) which may include chemical processor for polycarbonate resins and cellulose triacetate – photographic film, developer EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)).

N/A means these scenarios are not occupational or consumer conditions of use

2.4.1 Occupational Exposures

For the purpose of this assessment, EPA considered occupational exposure of the total workforce of exposed users and non-users, which include but are not limited to male and female workers of reproductive age who are >16 years of age. Female workers of reproductive age are >16 to less than 50 years old. Adolescents (>16 to <21 years old) are a small part of this total workforce. The occupational exposure assessment is applicable to and covers the entire workforce who are exposed to methylene chloride.

Occupational Exposures Approach and Methodology Section 2.4.1.1 summarizes the occupational acute and chronic inhalation exposure concentration and dermal dose models for methylene chloride.

These models were then applied for the various industries and scenarios identified in Table 2-24. Occupational Exposure Estimates by Scenario Section 2.4.1.2 summarizes air concentrations, including both 8-hr time-weighted averages (TWA) and shorter-term averages, and inhalation

exposure concentrations and dermal doses by occupational exposure scenario (OES), and overall summaries of model outputs and numbers of workers by OES.

The supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)) provides background details on industries that may use methylene chloride, worker activities, processes, numbers of sites and number of potentially exposed workers. This supplemental document also provides detailed discussion on the values of the exposure parameters and air concentrations and associated worker inhalation and dermal exposure results presented in this section.

For each scenario, EPA distinguishes exposures for workers and occupational non-users (ONUs). Normally, a primary difference between workers and ONUs is that workers may handle chemical substances and have direct dermal contact with chemicals that they handle, while ONUs are working in the general vicinity of workers but do not handle chemical substances and do not have direct dermal contact with chemicals being handled by the workers. EPA expects that ONUs may often have lower inhalation exposures than workers since they may be further from the exposure source than workers. For inhalation, if EPA cannot distinguish ONU exposures from workers, EPA assumes that ONU inhalation to be less than the inhalation estimates for workers.

2.4.1.1 Occupational Exposures Approach and Methodology

This section summarizes the key occupational acute and chronic inhalation exposure concentration and dermal dose models for methylene chloride. The supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)) provides detailed discussion on the values of the exposure parameters and air concentrations input into these models.

Acute and Chronic Inhalation Exposure Concentrations Models

A key input to the acute and chronic models for occupational assessment is 8-hr time-weighted average (TWA) air concentration. The 8-hr TWA air concentrations are time averaged to calculate acute exposure, average daily concentration (ADC) for chronic, non-cancer risks, and lifetime average daily concentration (LADC) for chronic, cancer risks.

Acute workplace exposures are assumed to be equal to the contaminant concentration in air (8- or 12-hr TWA), per Equation 2-4.

$$AEC = \frac{C \times ED}{AT_{acute}} \quad (\text{Eq. 2-4})$$

Where:

- AEC = acute exposure concentration (mg/m³)
- C = contaminant concentration in air (mg/m³, 8- or 12-hr TWA)
- ED = exposure duration (8 or 12 hr/day)

AT_{acute} = acute averaging time (8 or 12 hr)

ADC and LADC are used to estimate workplace chronic exposures for non-cancer and cancer risks, respectively. These exposures are estimated as follows:

$$ADC \text{ or } LADC = \frac{C \times ED \times EF \times WY}{AT \text{ or } AT_c} \quad (\text{Eq. 2-5})$$

Where:

- ADC = average daily concentration (mg/m^3) used for chronic non-cancer risk calculations
- LADC = lifetime average daily concentration (mg/m^3) used for chronic cancer risk calculations
- C = contaminant concentration in air (mg/m^3 , 8-hr TWA or 12-hr TWA)
- ED = exposure duration (8 or 12 hr/day depending upon TWA of C)
- EF = exposure frequency (250 days/yr for 8 hr/day ED or 167 days/yr for 12 hr/day ED)
- WY = exposed working years per lifetime (tenure values used to represent: 50th percentile = 31; 95th percentile = 40)
- AT = averaging time, non-cancer risks ($WY \times 365 \text{ days/yr} \times 24 \text{ hr/day}$)
- AT_c = averaging time, cancer risks (lifetime (LT) \times 250 days/year \times 8 hr/day for 8 hr/day ED or 167 days/yr for 12 hr/day for 12 hr/day ED; where LT = 78 years); this averaging time corresponds to the cancer benchmark as indicated in Chapter 3 HAZARDS

EPA reviewed workplace inhalation monitoring data collected by government agencies such as OSHA and NIOSH, and monitoring data found in published literature (i.e., personal exposure monitoring data and area monitoring data).

OSHA data are collected as part of compliance inspections at various types of facilities. Certain industries are typically targeted based on national and regional emphasis programs. These inspections are aimed at specific high-hazard industries or individual workplaces that have experienced high rates of injuries and illnesses. Emphasis programs do use injury and illness rates to inform their creation, but the bulk the sampling from programmed inspections would come from scheduling that is based on objective or neutral selection criteria. Unprogrammed inspections may also collect data and those inspections result from complaints, referrals, or fatality/ catastrophe incidents. These data are compiled in the Chemical Exposure Health Data (CEHD) database, available on the OSHA website, which contains the facility name, NAICS code, sampling date, sampling time, and sample result. However, OSHA provided a subset of data that also included worker activity descriptions and were verified for quality and were subsequently used in the risk evaluation ([OSHA, 2019](#)). A comment from Dr. Finkel also provided an OSHA dataset originating from a Freedom of Information Act (FOIA) request. However, this dataset only included Standard Industrial Classification (SIC) codes which are less specific than NAICS codes and also did not identify worker activities. Where possible, EPA associated SIC codes with NAICS to pair the exposure data from Finkel ([2017](#)) with some OESs.

NIOSH data were primarily from Health Hazard Evaluations (HHEs) conducted at specific processing or use sites.

Data were evaluated using the evaluation strategies laid out in the Application of Systematic Review in TSCA Risk Evaluations ([U.S. EPA, 2018a](#)), and the evaluation details are shown in two supplemental files: Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data ([EPA, 2019d](#)) Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Common Sources ([EPA, 2019c](#)). Where available, EPA used air concentration data and estimates found in government or published literature sources. Where air concentration data were not available, modeling estimates were used. Details on which models EPA used are included in Section 2.4.1.2 for the applicable OESs and discussion of the uncertainties associated with these models is included in Section 4.4.2. Beyond the modeling conducted for this Risk Evaluation, EPA did not find reasonably available models and associated parameter sets to conduct additional modeling.

EPA evaluated inhalation exposure for workers using personal monitoring data or modeled near-field exposure concentrations. Since ONUs do not directly handle methylene chloride, EPA reviewed personal monitoring data, modeled far-field exposure concentrations, and area monitoring data in evaluating potential inhalation exposures for ONUs. Because modeled results are typically intended to capture exposures in the near-field, modeling that does not contain a specific far-field component are not considered to be suitable for ONUs. Area monitoring data may potentially represent ONU exposures depending on the monitor placement and the intended sample population.

Consideration of Engineering Controls and Personal Protective Equipment

OSHA requires and NIOSH recommends that employers utilize the hierarchy of controls to address hazardous exposures in the workplace. The hierarchy of controls strategy outlines, in descending order of priority, the use of elimination, substitution, engineering controls, administrative controls, and lastly personal protective equipment (PPE). The hierarchy of controls prioritizes the most effective measures first which is to eliminate or substitute the harmful chemical (e.g., use a different process, substitute with a less hazardous material), thereby preventing or reducing exposure potential. Following elimination and substitution, the hierarchy recommends engineering controls to isolate employees from the hazard, followed by administrative controls, or changes in work practices to reduce exposure potential (e.g., source enclosure, local exhaust ventilation systems). Administrative controls are policies and procedures instituted and overseen by the employer to protect worker exposures. As the last means of control, the use of personal protective equipment (e.g., respirators, gloves) is recommended, when the other control measures cannot reduce workplace exposure to an acceptable level. The National Institute for Occupational Safety and Health (NIOSH) and the U.S. Department of Labor's Bureau of Labor Statistics (BLS) conducted a voluntary survey of U.S. employers regarding the use of respiratory protective devices between August 2001 and January 2002 ([NIOSH, 2003](#)). For additional information, please also refer to [*Memorandum NIOSH BLS Respirator Usage in Private Sector Firms. Docket # 1354 EPA-HQ-OPPT-2019-0500*] ([EPA, 2020a](#)).

OSHA Standards and Respiratory Protection

The Occupational Safety and Health Administration (OSHA) Respiratory Protection Standard (29 CFR 1910.134) provides a summary of respirator types by their assigned protection factor (APF). Assigned Protection Factor (APF) “means the workplace level of respiratory protection that a respirator or class of respirators is expected to provide to employees when the employer implements a continuing, effective respiratory protection program” according to the requirements of OSHA's Respiratory Protection Standard. Because methylene chloride may cause eye irritation or damage, the OSHA standard for methylene chloride (29 CFR 1910.1052) prohibits use of quarter and half mask respirators; additionally, only supplied air respirators (SARs) can be used because methylene chloride may pass through air purifying respirators.

Respirator types and corresponding APFs indicated in bold font in Table 2-25. comply with the OSHA standard for protection against methylene chloride. APFs are intended to guide the selection of an appropriate class of respirators to protect workers after a substance is determined to be hazardous, after an occupational exposure limit is established, and only when the exposure limit is exceeded after feasible engineering, work practice, and administrative controls have been put in place. For methylene chloride, the OSHA PEL is 25 ppm, or 87 mg/m³ as an 8-hr TWA, and the OSHA short-term exposure limit (STEL) is 125 ppm, or 433 mg/m³ as a 15-min TWA. For each occupational exposure scenario in Section 2.4.1.2, EPA compares the exposure data and estimates to the PEL and STEL.

The current OSHA PEL was updated in 1997; prior to the change the OSHA PEL had been 500 ppm as an 8-hr TWA, which was 20 times higher than the current PEL of 25 ppm. EPA received a public comment that included over 12,000 samples taken during OSHA or state health inspections from 1984 to 2016 ([Finkel, 2017](#)). After the draft Risk Evaluation, EPA conducted a more robust statistical analysis on these samples to evaluate how occupational exposures to methylene chloride changed with time; in particular, any changes after the new PEL was fully implemented (the 1997 OSHA rule required all facilities to comply with all parts of the rule no later than April 9, 2000, which was three years after the final rule's effective date of April 10, 1997) (62 FR 1494). An appendix in the supplemental document titled "Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment" ([EPA, 2019b](#)) provides detailed discussion on EPA's analysis. EPA filtered the samples to personal samples only, combined sequential samples taken on the same worker, and calculated about 3,300 8-hr TWA exposures. To account for the presence of non-detects, EPA replaced sample results of 0 ppm with the limit of detection (LOD) divided by the square root of two. The exact LOD of the sampling and analysis method used in each inspection conducted from 1984 to 2016 is not known. EPA estimated the exposure concentrations for these data, following EPA/OPPT's Guidelines for Statistical Analysis of Occupational Exposure Data ([1994](#)), which recommends using the LOD divided by the square root of two if the geometric standard deviation of the data is less than 3.0 and LOD divided by two if the geometric standard deviation is 3.0 or greater. OSHA method 80 for methylene chloride (fully validated in 1990) reports an LOD of 0.201 ppm ([Osha, 1990](#)). NIOSH method 1005 for methylene chloride (issued January 15, 1998) reports an LOD of 0.4 micrograms per sample, with a minimum and maximum air sample volume of 0.5 and 2.5 liters, respectively ([Niosh, 1998](#)). EPA calculated a range in LOD for the NIOSH method of 0.046 to 0.231 ppm.

For this analysis, EPA used an LOD of 0.046 ppm (the smallest of these three LOD values) and an LOD divided by the square root of two of 0.0326 ppm.

EPA analyzed 1,407 and 1,471 8-hr TWA exposures measured prior to April 10, 1997 (pre-rule) and after April 10, 2000 (post-rule). The arithmetic mean of the pre-rule and post-rule distributions was 27.3 ppm and 17.9 ppm, respectively, a reduction of about 34%. The median of the pre-rule and post-rule distributions was 3.7 ppm and 2.5 ppm, respectively, a reduction of about 31%, similar to the reduction in the mean. EPA calculated the percentile ranks of 25 ppm in the pre-rule and post-rule distributions: approximately 23% and 15% of the exposures exceeded 25 ppm in the pre-rule and post-rule distributions, respectively. This is a reduction of about 35%, similar to the reductions in the mean and median. While exposures in the distributions showed consistent reductions of about 30% to 35%, this followed a reduction in the PEL of 95%. Hence, a twentyfold reduction in the PEL resulted in only an approximately 1.5-fold reduction in actual exposures. Due to the small reduction in exposures relative to the reduction in PEL, EPA included the pre-rule samples as well as the post-rule samples in the occupational exposure assessment to provide a more robust data set.

In addition to analyzing the entire distributions, EPA crosswalked reported SIC codes to 2017 NAICS codes and analyzed exposure trends in certain industry sectors. Table 2-23 summarizes an analysis of industry codes representing the larger shares of the data set, while table 2-24 summarizes an analyses by OES (using the same NAICS codes used for the Number of Workers analyses discussed Section 2.4.1.2). The summaries generally show a range in exposure reductions across the industry sectors. The largest OES decreases were for spot cleaning (94.5%) and fabric finishing (93.4%). On the other hand, exposures increased for plastics manufacturing (617%) and aerosol degreasing (130%).

Table 2-23. Summary of Pre- and Post-Rule Exposure Concentrations for Industries with Largest Number of Data Points

NAICS Code	NAICS Description	Pre-Rule Update (prior to April 10, 1997)			Post-Rule Update, after all requirements in effect (after April 10, 2000)			Percent Reduction in Mean (%)
		Number of Samples	Arithmetic Mean (ppm)	% of Samples Above 25 ppm	Number of Samples	Arithmetic Mean (ppm)	% of Samples Above 25 ppm	
811420	Reupholstery and Furniture Repair	36	98.73	53.8%	121	29.38	30.8%	70.2%
337110	Wood Kitchen Cabinet and Countertop Manufacturing	35	9.91	11.7%	80	6.96	4.7%	29.8%
326121	Unlaminated Plastics Profile Shape Manufacturing	76	35.00	30.2%	78	14.24	11.5%	59.3%
326140	Polystyrene Foam Product Manufacturing	12	19.27	31.9%	15	11.44	12.0%	40.6%

336211	Motor Vehicle Body Manufacturing	32	50.69	30.3%	6	3.04	N/A ^a	94.0%
323111	Commercial Printing (except Screen and Books)	55	9.54	11.1%	28	5.02	5.8%	47.4%
541380	Testing Laboratories	16	2.43	N/A ^a	29	3.65	2.2%	-50.6% ^b
316110	Leather and Hide Tanning and Finishing	10	8.14	5.8%	40	8.90	12.9%	-9.4% ^b
All NAICS Codes Together		1,407	27.26	23.0%	1,471	17.86	15.0%	34%

Source of all samples: [Finkel \(2017\)](#)

a – N/A: Not applicable. There are no exposures above 25 ppm.

b – A negative reduction means the mean exposure increased from the pre-rule to post-rule periods.

Table 2-24. Summary of Pre- and Post-Rule Exposure Concentrations Mapped to Occupational Exposure Scenarios

OES	Potential NAICS	Pre-Rule Update (prior to April 10, 1997)			Post-Rule Update, after all requirements in effect (after April 10, 2000)			Percent Reduction in Mean (%)
		Number of Samples	Arithmetic Mean (ppm)	Percent of Samples Above 25 ppm	Number of Samples	Arithmetic Mean (ppm)	Percent of Samples Above 25 ppm	
Processing as a Reactant	325120, 325320	12	15.2	16.7%	0	N/A ^a	N/A ^a	N/A ^a
Processing - Incorporation into Formulation	325510, 325520, 325998	23	46.2	52.2%	17	28.1	47.1%	39.3%
Aerosol degreasing	811111, 811112, 811113, 811118, 811121, 811122, 811191, 811198, 811211, 811212, 811213, 811219, 811310, 811411, 811490, 451110, 441100	13	6.6	7.7%	15	15.1	13.3%	-129.7%
Adhesives and Sealants	326150, 332300, 333900, 334100, 334200, 334300, 334400, 334500, 334600, 335100, 335200, 335300, 335900, 336100, 336200, 336300, 336400, 336500, 336600, 337100, 811420	256	44.8	32.0%	230	24.4	24.4%	45.5%

Paints and Coatings	238320, 323113, 332000, 337100, 448100, 713100, 811111	78	23.5	19.2%	169	12.3	7.7%	47.8%
Fabric Finishing	313210, 313220, 313230, 313240, 313310, 313320	27	15.3	18.5%	6	1.0	0.0%	93.4%
Spot Cleaning	812320, 812332	14	14.1	21.4%	3	0.8	0.0%	94.5%
Laboratory Use	541380, 621511	19	5.2	5.3%	36	3.2	2.8%	38.9%
Plastic Product Mfg	325211, 325212, 325220, 325991, 326199	14	3.6	0.0%	20	26.1	5.0%	-616.9%
Lithographic Printing Plate Cleaning	323111	55	9.5	10.9%	28	5.0	7.1%	47.4%
Waste Handling, Disposal, Treatment, and Recycling	562211, 562213, 562920	15	6.0	6.7%	0	N/A ^a	N/A ^a	N/A ^a

Source of all samples: [Finkel \(2017\)](#)

a – N/A: Not applicable. Insufficient data points available.

b – N/A: Not applicable. There are no exposures above 25 ppm.

c – A negative reduction means the mean exposure increased from the pre-rule to post-rule periods. EPA does not have reasonably available information to indicate possible reasons for increases.

EPA has sought additional data regarding exposures, particularly during the public comment phases on the documents preceding the draft version of this risk evaluation (e.g., the methylene chloride Section 6 rule and the problem formulation). With the exception of paint and coating removers, EPA has not received information to date to indicate that workplace changes have occurred broadly in particular sectors over the past 40 years.

Based on the protection standards, inhalation exposures may be reduced by a factor of 25, 50, 1,000, or 10,000, if respirators are required and properly worn and fitted. Air concentration data are assumed to be pre-APF unless indicated otherwise in the source, and APFs acceptable under the OSHA standards are not otherwise considered or used in the occupational exposure assessment but are considered in the risk characterization and risk determination.

Table 2-25. Assigned Protection Factors for Respirators in OSHA Standard 29 CFR 1910.134^a

Type of Respirator	Quarter Mask	Half Mask	Full Facepiece	Helmet/Hood	Loose-fitting Facepiece
1. Air Purifying Respirator	5	10	50		
2. Powered Air-Purifying Respirator		50	1,000	25/1,000	25

Type of Respirator	Quarter Mask	Half Mask	Full Facepiece	Helmet/Hood	Loose-fitting Facepiece
3. Supplied-Air Respirator (SAR) or Airline Respirator					
• Demand mode		10	50
• Continuous flow mode		50	1,000	25/1,000	25
• Pressure-demand or other positive-pressure mode		50	1,000
4. Self-Contained Breathing Apparatus (SCBA)					
• Demand mode		10	50	50
• Pressure-demand or other positive-pressure mode		10,000	10,000

Note that only APFs indicated in **bold** are acceptable to OSHA for methylene chloride protection. Other respirators from the Respiratory Protection Standard that are not acceptable for methylene chloride protection are indicated in shaded cells.

Key Dermal Exposure Dose Models

Current EPA dermal models do not incorporate the evaporation of material from the dermis. The dermal potential dose rate, D_{exp} (mg/day), is calculated as ([EPA, 2013a](#)):

(Eq. 2-6)

$$D_{exp} = S \times Q_u \times Y_{derm} \times FT$$

Where:

S is the surface area of contact (cm^2 ; defaults: 535 cm^2 (central tendency); $1,070 \text{ cm}^2$ (high end) = full area of one hand (central tendency) or two hands (high end), a 50th percentile value for men > 21 yr ([EPA, 2011a](#)), the highest exposed population); note: EPA has no data on actual surface area of contact with liquid and that the value is assumed to represent an adequate proxy for a high-end surface area of contact with liquid that may sometimes include exposures to much of the hands and also beyond the hands, such as wrists, forearms, neck, or other parts of the body, for some scenarios.

Q_u is the quantity remaining on the skin (mg/cm^2 -event; defaults: $1.4 \text{ mg}/\text{cm}^2$ -event (central tendency); $2.1 \text{ mg}/\text{cm}^2$ -event (high end))

Y_{derm} is the weight fraction of the chemical of interest in the liquid ($0 \leq Y_{derm} \leq 1$)

FT is the frequency of events (integer number per day; default: 1 event/day); note: EPA has described events per day (FT) as a primary uncertainty for dermal modeling in the discussion of occupational dermal uncertainties in section 4.4.2.4. This discussion also notes that this assumption likely underestimates exposure as workers often come into repeat contact with the chemical throughout their workday.

Here Q_u does not represent the quantity remaining after evaporation, but represents the quantity remaining after the bulk liquid has fallen from the hand that cannot be removed by wiping the skin (e.g., the film that remains on the skin).

One way to account for evaporation of a volatile solvent would be to add a multiplicative factor to the EPA model to represent the proportion of chemical that remains on the skin after evaporation, f_{abs} (default: 0.08 for methylene chloride during industrial use; 0.13 for methylene chloride during commercial use) ([Miller et al., 2005](#)):

(Eq. 2-7)

$$D_{exp} = S \times \frac{(Q_u \times f_{abs})}{PF} \times Y_{derm} \times FT$$

This approach simply removes the evaporated mass from the calculation of dermal uptake. Evaporation is not instantaneous, but the EPA model already has a simplified representation of the kinetics of dermal uptake. The model assumes a fixed fractional absorption of the applied dose; however, fractional absorption may vary and is dependent on various factors including physical-chemical properties and wind speed. More information about this approach is presented in Appendix E of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

The occupational and consumer dermal exposure assessment approaches have a common underlying methodology but use different parametric approaches for dermal exposures due to different data availability and assessment needs. For example, the occupational approach accounts for glove use using protection factors, while the consumer approach does not consider glove use since consumers are not expected to use gloves constructed with appropriate materials. The consumer approach (see Dermal section of Section 2.4.2.3.1) factors in time because consumer activities as a function of exposure times to products are much better defined and characterized, while duration of dermal exposure times for different occupational activities across various workplaces are often not known.

Regarding glove use, data about the frequency of effective glove use – that is, the proper use of effective gloves – is very limited in industrial settings. Initial literature review suggests that there is unlikely to be sufficient data to justify a specific probability distribution for effective glove use for a chemical or industry. Instead, the impact of effective glove use is explored by considering different percentages of effectiveness.

EPA also made assumptions about glove use and associated protection factors (PF). Where workers wear gloves, workers are exposed to methylene chloride-based product that may penetrate the gloves, such as seepage through the cuff from improper donning of the gloves, and if the gloves occlude the evaporation of methylene chloride from the skin. Where workers do not wear gloves, workers are exposed through direct contact with methylene chloride.

Gloves only offer barrier protection until the chemical breaks through the glove material. Using a conceptual model, Cherrie ([2004](#)) proposed a glove workplace protection factor – the ratio of estimated uptake through the hands without gloves to the estimated uptake through the hands while wearing gloves: this protection factor is driven by flux, and thus varies with time. The European Centre For Ecotoxicology and Toxicology of Chemicals Targeted Risk Assessment

(ECETOC TRA) model represents the protection factor of gloves as a fixed, assigned protection factor equal to 5, 10, or 20 ([Marquart et al., 2017](#)), where, similar to the APR for respiratory protection, the inverse of the protection factor is the fraction of the chemical that penetrates the glove. Dermal doses without properly trained glove use are estimated in the occupational exposure sections below and summarized in Table 2-26. Potential impacts of these protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-83. As indicated in Table 2-26, use of protection factors above 1 is recommended only for glove materials that have been tested for permeation against the methylene chloride-containing liquids associated with the condition of use. EPA has not found information that would indicate specific activity training (e.g., procedure for glove removal and disposal) for tasks where dermal exposure can be expected to occur in a majority of sites in industrial only OESs, so the PF of 20 would usually not be expected to be achieved.

Table 2-26. Glove Protection Factors for Different Dermal Protection Strategies from ECETOC TRA v3

Dermal Protection Characteristics	Setting	Protection Factor, PF
a. No gloves used, or any glove / gauntlet without permeation data and without employee training	Industrial and Commercial Uses	1
b. Gloves with available permeation data indicating that the material of construction offers good protection for the substance		5
c. Chemically resistant gloves (i.e., as <i>b</i> above) with “basic” employee training		10
d. Chemically resistant gloves in combination with specific activity training (e.g., procedure for glove removal and disposal) for tasks where dermal exposure can be expected to occur	Industrial Uses Only	20

EPA also considered potential dermal exposure in cases where exposure is occluded. See further discussion on occlusion in Appendix E of the Supplemental Information on Releases and Occupational Exposure Assessment document ([EPA, 2019b](#)).

It is important to note that the occupational dermal exposure approach and modeling differs from that for consumer exposure approach outlined in Section 2.4.2.3.1 due to different data availability and assessment needs and may result in different exposure values for similar conditions of use.

Appendix F contains information gathered by EPA in support of understanding glove use for pure methylene chloride and for paint and coatings removal using methylene chloride formulations. This information may be generally useful for a broader range of uses of methylene chloride and is presented for illustrative purposes. This appendix also contains a summary of information on gloves from Safety Data Sheets (SDS) for methylene chloride and formulations containing methylene chloride.

Risk Evaluation Definition of Central Tendency and High End

For most scenarios, EPA did not find enough data to determine statistical distributions of the actual exposure parameters and concentration inputs to the inhalation and dermal models described above. Within the distributions, central tendencies describe 50th percentile or the substitute that most closely represents the 50th percentile. The high-end of a distribution describes the range of the distribution above 90th percentile ([U.S. EPA, 1992](#)). Ideally, EPA would use the 50th and 95th percentiles for each parameter. Where these statistics were unknown, the mean or median (mean is preferable to median) served as substitutes for 50th percentile and the high-end of ranges served as a substitute for 95th percentile. However, these substitutes were highly uncertain and not ideal substitutes for the percentiles. EPA could not determine whether these substitutes were suitable to represent statistical distributions of real-world scenarios.

Exposures are calculated from the datasets provided in the sources depending on the size of the dataset. For datasets with six or more data points, central tendency and high-end exposures were estimated using the 50th percentile and 95th percentile. For datasets with three to five data points, central tendency exposure was calculated using the 50th percentile and the maximum was presented as the high-end exposure estimate. For datasets with two data points, the midpoint was presented as a midpoint value and the higher of the two values was presented as a higher value. Finally, data sets with only one data point presented the value as a what-if exposure. For datasets including exposure data that were reported as below the limit of detection (LOD), EPA estimated the exposure concentrations for these data, following EPA/OPPT's *Guidelines for Statistical Analysis of Occupational Exposure Data* (1994) which recommends using the $LOD / 2^{0.5}$ if the geometric standard deviation of the data is less than 3.0 and $LOD / 2$ if the geometric standard deviation is 3.0 or greater ([EPA, 1994](#)).

2.4.1.2 Occupational Exposure Estimates by Scenario

Details of the occupational exposure assessments for each of the Occupational Exposure Scenarios (OES) listed in Table 2-24, with one exception, are available in the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). The exception is for Paint and Coating Removers, which are covered in Appendix L.

The following subsections contain a summary of inhalation and dermal estimates for each OES, assuming no PPE use. Details on the inhalation and dermal estimates as well as process descriptions, numbers of sites and potentially exposed workers, and worker activities for each OES are available in the supplemental document ([EPA, 2019b](#)). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of this supplemental document. EPA could not determine whether PPE or engineering controls were used for some settings where monitoring was conducted.

Key uncertainties toward exposure estimates in these scenarios are summarized in Section 4.4.2.

Table 2-27 presents estimated numbers of workers in the OESs assessed for methylene chloride. Where available, EPA used publicly available data (typically CDR) to provide a basis to estimate

the number of sites, workers and ONUs. EPA supplemented the available CDR data with U.S. economic data using the following method:

1. Identify the North American Industry Classification System (NAICS) codes for the industry sectors associated with these uses.
2. Estimate total employment by industry/occupation combination using the Bureau of Labor Statistics' Occupational Employment Statistics data (BLS Data).
3. Refine the OES estimates where they are not sufficiently granular by using the U.S. Census' Statistics of US Businesses (SUSB) (SUSB Data) data on total employment by 6-digit NAICS.
4. Use market penetration data to estimate the percentage of employees likely to be using methylene chloride instead of other chemicals.
5. Where market penetration data are not available, use the estimated workers/ONUs per site in the 6-digit NAICS code and multiply by the number of sites estimated from CDR, TRI, or National Emissions Inventory (NEI).

EPA combined the data generated in Steps 1 through 5 to produce an estimate of the number of employees using methylene chloride in each industry/occupation combination (if available), and then summed these to arrive at a total estimate of the number of employees with exposure within the occupational exposure scenario. More details on the data are provided in the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Table 2-27. Estimated Numbers of Workers in the Assessed Industry Scenarios for Methylene Chloride

Occupational Exposure Scenario	Number of Workers	Number of ONUs
Manufacturing	1,200	*
Processing as a Reactant	460	120^
Processing - Incorporation into Formulation	4,500	*
Repackaging	2,300	*
Batch Open-Top Vapor Degreasing	270	*
Conveyorized Vapor Degreasing	180	*
Cold Cleaning	95,000	*
Aerosol Degreasing/Lubricants	250,000	29,000
Adhesives	2,700,000	810,000
Paints and Coatings	1,800,000	340,000
Adhesive and Caulk Removers	190,000	18,000
Fabric Finishing	19,000	12,000

Occupational Exposure Scenario	Number of Workers	Number of ONUs
Spot Cleaning	76,000	7,900
CTA Manufacturing	700	*
Flexible PU Foam Manufacturing	9,600	2,700
Laboratory Use	17,000	150,000
Plastic Product Manufacturing	210,000	90,000
Lithographic Printing Cleaner	40,000	19,000
Miscellaneous Non-Aerosol Industrial and Commercial Use (Cleaning Solvent)	<1,400,000	*
Waste Handling, Disposal, Treatment, and Recycling	12,000	7,600

* - Based on EPA's analysis, the data for worker and ONUs and could not be distinguished.

^ - One data source distinguished ONUs from workers and the other source did not.

2.4.1.2.1 Manufacturing

The Halogenated Solvents Industry Alliance (HSIA) provided personal monitoring data from 2005 through 2018 at two manufacturing facilities for a variety of worker activities ([Halogenated Solvents Industry Alliance, 2018](#)). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Overall, 136 8-hr TWA and 149 12-hr TWA personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8- and 12-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. Both the central tendency and high-end 8- and 12-hr TWA exposure concentrations for this scenario are approximately one order of magnitude below the OSHA Permissible Exposure Limit (PEL) value of 87 mg/m³ (25 ppm) as an 8-hr TWA. All data points were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and are summarized in Table 2-28.

Table 2-28. Worker Exposure to Methylene Chloride During Manufacturing^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
<i>8-hr TWA Results</i>				
8-hr TWA Exposure Concentration	136	0.36	4.6	High
Average Daily Concentration (ADC)		0.08	1.1	
Lifetime Average Daily Concentration (LADC)		0.14	2.4	
<i>12-hr TWA Results</i>				
12-hr TWA Exposure Concentration	149	0.45	12	High
Average Daily Concentration (ADC)		0.15	4.1	
Lifetime Average Daily Concentration (LADC)		0.27	9.3	

Sources: [Halogenated Solvents Industry Alliance \(2018\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-29 summarizes available short-term exposure data for workers provided by HSIA ([Halogenated Solvents Industry Alliance, 2018](#)).

Table 2-29. Short-Term Worker Exposure to Methylene Chloride During Manufacturing

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
15-min ^a	148	9.6	180	High
30-min ^b	1	2.6		
1-hr ^c	4	4.3	16	

Source: [Halogenated Solvents Industry Alliance \(2018\)](#).

a – EPA evaluated 148 samples, with durations ranging from 15 to 22 minutes, as 15-minute exposures.

b – EPA evaluated one sample, with a duration of 35 minutes, as a 30-minute exposure.

c – EPA evaluated four samples, with durations ranging from 50 to 55 minutes, as 1-hour exposures.

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA. One sample of 486 mg/m³ among the 148 15-min samples exceeded this limit, and the remaining 147 samples were below this limit.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures from methylene chloride manufacturing. Since ONUs do not directly handle methylene chloride (otherwise they would be considered workers), ONU inhalation exposures could be lower than worker inhalation exposures. Information on activities where ONUs may be

present are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-30 presents estimated dermal exposures during domestic manufacturing.

Table 2-30. Summary of Dermal Exposure Doses to Methylene Chloride for Manufacturing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Manufacturing	Industrial	1.0	60	180	0.08

a – EPA assumes methylene chloride manufactured at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has not identified additional uncertainties for this scenario beyond those discussed in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 136 8-hr and 149 12-hr data points from 1 source, and the data quality ratings from systematic review for these data were high. All of the data points were post-PEL rule. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.2 Processing as a Reactant

HSIA provided monitoring data (15 data points) from 2010 through 2017 from a fluorochemical manufacturing facility, where methylene chloride could be used as an intermediate for the production of fluorocarbon blends ([Halogenated Solvents Industry Alliance, 2018](#)). Finkel (2017) also submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes for Industrial Gas Manufacturing and Pesticide and Other Agricultural Chemical Manufacturing. For the set of 14 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 301 mg/m³. Worker activity information was not available; therefore, it was not possible to specifically attribute the exposures to the use of methylene chloride as a reactant, nor to distinguish workers from ONUs. While there may be additional activities at these sites, such as use of methylene chloride as a cleaning solvent that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker

exposure during processing as a reactant. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Overall, 29 8-hr TWA personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and worst-case estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration is more than an order of magnitude lower than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end 8-hr TWA exposure concentrations for this scenario is higher than the OSHA PEL. Of the 29 data points, 12 of the data points were pre-PEL rule, 2 data points were during the transition period, while 15 data points were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods). Based on available short-term exposure data, 10-minute TWAs could be up to 350 mg/m³ during specific operations such as filter changing, charging and discharging, etc.

Table 2-31 presents the calculated the AEC, ADC, and LADC for these 8-hr TWA exposure concentrations, as described in Section 2.4.1.1.

Table 2-31. Worker Exposure to Methylene Chloride During Processing as a Reactant During Fluorochemicals Manufacturing^a

	Number of Samples	Central Tendency (mg/m ³)	High End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	29	1.6	110	High and Medium
Average Daily Concentration (ADC)		0.37	25	
Lifetime Average Daily Concentration (LADC)		0.65	55	

Sources: [Halogenated Solvents Industry Alliance \(2018\)](#); [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-32 summarizes available short-term exposure data available for “other chemical industry” and during drumming at a pesticide manufacturing site.

Table 2-32. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Processing as a Reactant

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Other Chemical Industry	TNO (CIVO) (1999)	filter changing, charging and discharging, etc.	350 (max)	10 ^a	Low
Pesticides Mfg	Olin Corp (1979)	Drumming	1,700	25 ^b	Medium

a – EPA evaluated as a 15-minute exposure.

b – EPA evaluated as a 30-minute exposure

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal data on or parameters for modeling potential ONU inhalation exposures. Limited area monitoring data were identified (see Appendix A.2 of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#))). However, the representativeness of these data for ONU exposures is not clear because of uncertainty concerning the intended sample population and the selection of the specific monitoring location. EPA assumed that the area monitoring data were not appropriate surrogates for ONU exposure due to lack of necessary metadata, such as monitor location and distance from worker activities, to justify its use. ONUs are employees who work at the facilities that process and use methylene chloride, but who do not directly handle the material. ONUs may also be exposed to methylene chloride but are expected to have lower inhalation exposures and are not expected to have dermal exposures. ONUs for this condition of use may include supervisors, managers, engineers, and other personnel in nearby production areas. Since ONUs do not directly handle formulations containing methylene chloride (otherwise they would be considered workers), EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities is insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified using modeling.

Table 2-33 presents modeled dermal exposures during processing as a reactant.

Table 2-33. Summary of Dermal Exposure Doses to Methylene Chloride for Processing as a Reactant

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Processing as a Reactant	Industrial	1.0	60	180	0.08

a – EPA assumes methylene chloride is received at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 29 data points from 2 sources, and the data quality ratings from systematic review for these data were high and medium. The primary limitations of these data include the age of the data (12 of the data points were pre-PEL rule, 2 data points were during the transition period, while 15 data points were post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available to specifically attribute exposures to the use of methylene chloride as a reactant or to determine whether sampled activities were representative of full-shift exposures. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.3 Processing - Incorporation into Formulation, Mixture, or Reaction Product

Finkel (2017) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes for Paint and Coating Manufacturing and Adhesives Manufacturing. For the set of 45 data points, 8-hr TWA exposure concentrations ranged from 0.86 to 559 mg/m³. Worker activity information was not available; therefore, it was not possible to specifically attribute the exposures to formulation processes using methylene chloride, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use of methylene chloride as a reactant or as a cleaning solvent that contribute to

methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during processing methylene chloride into formulation. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Additional discussion of data treatment is included in Appendix H. U.S. EPA (1985) also provided exposure data for packing at paint/varnish and cleaning products sites, ranging from 52 mg/m³ (mixing) to 2,223 mg/m³ (valve dropper). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Overall, 55 personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is slightly higher than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is approximately six times higher. Of the 55 data points, 33 of the data points were pre-PEL rule, 7 data points were during the transition period, while 15 data points were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and are listed in Table 2-34.

Table 2-34. Worker Exposure to Methylene Chloride During Processing – Incorporation into Formulation, Mixture, or Reaction Product^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	55	100	540	High and Medium
Average Daily Concentration (ADC)		23	120	
Lifetime Average Daily Concentration (LADC)		40	280	

Sources: EPA (1985); Finkel (2017)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

TNO (CIVO) (1999) indicated that the peak exposure during filling may be up to 180 mg/m³ but did not provide exposure duration. Therefore, this exposure concentration was not used in the assessment.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, ONU inhalation exposures could be lower than worker inhalation exposures.

Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-35 presents modeled dermal exposures during processing – incorporation into formulation, mixture or reaction product.

Table 2-35. Summary of Dermal Exposure Doses to Methylene Chloride for Processing - Incorporation into Formulation, Mixture, or Reaction Product.

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Processing - Incorporation into Formulation, Mixture, or Reaction Product	Industrial	1.0	60	180	0.08

a – EPA assumes methylene chloride is received at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 55 data points from 2 sources, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the age of the data (33 of the data points were pre-PEL rule, 7 data points were during the transition period, while 15 data points were post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available to specifically attribute exposures to the formulation of methylene chloride-containing products or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 39.3% from pre- to post-rule. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.4 Repackaging

EPA found limited inhalation monitoring data for repackaging from published literature sources. A 1986 Industrial Hygiene (IH) study at Unocal Corporation found full-shift exposures during filling drums, loading trucks, and transfer loading to be between 6.0 and 137.8 mg/m³ (5 data points) ([Unocal Corporation, 1986](#)). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Because only five 8-hr TWA data points were available, EPA assessed the median value of 8.8 mg/m³ as the central tendency, and the maximum reported value of 137.8 mg/m³ as the high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is approximately 10 times lower the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is approximately 1.5 times higher. All data points were pre-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC. The results of these calculations are shown in Table 2-36.

Table 2-36. Worker Exposure to Methylene Chloride During Repackaging^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	5	8.8	140	Medium
Average Daily Concentration (ADC)		2.0	31	
Lifetime Average Daily Concentration (LADC)		3.5	71	

Source: [Unocal Corporation \(1986\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-37 summarizes available short-term exposure data available from the same OSHA source identified above for the 8-hr TWA data.

Table 2-37. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Repackaging

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Distribution		Transfer loading from truck to	0.35	30 ^a	Medium

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
	Unocal Corporation (1986)	storage tank (4,100 gallons)			
		Truck loading (2,000 gallons)	330	50 ^b	
		Truck loading (800 gallons)	35	30 ^a	
		Truck loading (250 gallons)	30	47 ^b	

a – EPA evaluated two samples with durations of 30 minutes each, as 30-minute exposures.

b – EPA evaluated two samples with durations of 47 and 50 minutes, as a 1-hr exposures.

Note: The OSHA STEL is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. ONUs are employees who work at the site where methylene chloride is repackaged, but who do not directly perform the repackaging activity. ONUs for repackaging include supervisors, managers, and tradesmen that may be in the repackaging area but do not perform tasks that result in the same level of exposures as repackaging workers.

Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-38 presents modeled dermal exposures during repackaging.

Table 2-38. Summary of Dermal Exposure Doses to Methylene Chloride for Repackaging

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Repackaging	Industrial	1.0	60	180	0.08

a – EPA assumes repackaging of methylene chloride at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 5 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the age of the data (pre-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. No data were available to compare pre- and post-PEL rule exposures in Section 2.4.1.1. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.5 Batch Open-Top Vapor Degreasing

EPA found no monitoring data for methylene chloride in this use. To fill this data gap, EPA performed modeling of near-field and far-field exposure concentrations in the OTVD scenario for both workers and ONUs. Modeling details are in Appendix F of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). The central tendency and high-end 8-hr TWA exposure concentrations for this scenario exceed the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA.

Estimates of ADC and LADC for use in assessing risk were made using the approach and equations described in Section 2.4.1.1 and are presented in Table 2-39.

Table 2-39. Statistical Summary of Methylene Chloride 8-hr TWA Exposures (ADC and LADC) for Workers and ONUs for Batch Open-Top Vapor Degreasing

	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
Workers (Near-Field)			
8-hr TWA Exposure Concentration	170	740	N/A – Modeled Data
Average Daily Concentration (ADC)	38	170	
Lifetime Average Daily Concentration (LADC)	67	380	
ONUs (Far-Field)			
8-hr TWA Exposure Concentration	86	460	N/A – Modeled Data
Average Daily Concentration (ADC)	20	100	
Lifetime Average Daily Concentration (LADC)	34	230	

Table 2-40 presents modeled dermal exposures during batch open-top vapor degreasing use.

Table 2-40. Summary of Dermal Exposure Doses to Methylene Chloride for Batch Open-Top Vapor Degreasing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Batch Open-Top Vapor Degreasing	Industrial	1.0	60	180	0.08

a - EPA assumes that 100% methylene chloride is used for vapor degreasing operations.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation using the Latin hypercube sampling method with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from methylene chloride unit emissions and operating hours reported in the 2014 NEI ([EPA, 2018a](#)). The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for eight total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.6 ConveyORIZED Vapor Degreasing

EPA found no monitoring data for methylene chloride in this use. To fill this data gap, EPA performed modeling of near-field and far-field exposure concentrations in the conveyORIZED vapor degreasing scenario for both workers and ONUs. Modeling details are in Appendix F of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). The central tendency 8-hr TWA worker exposure concentration for this scenario is approximately twice the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is approximately five times higher. Exposure concentrations for ONUs are also considerably higher than the OSHA PEL.

Estimates of ADC and LADC for use in assessing risk were made using the approach and equations described in Section 2.4.1.1 and are presented in Table 2-41.

Table 2-41. Statistical Summary of Methylene Chloride 8-hr TWA Exposures (ADC and LADC) for Workers and ONUs for Conveyorized Vapor Degreasing

	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
Workers (Near-Field)			
8-hr TWA Exposure Concentration	490	1,400	N/A – Modeled Data
Average Daily Concentration (ADC)	110	320	
Lifetime Average Daily Concentration (LADC)	190	720	
ONUs (Far-Field)			
8-hr TWA Exposure Concentration	250	900	N/A – Modeled Data
Average Daily Concentration (ADC)	58	210	
Lifetime Average Daily Concentration (LADC)	100	460	

Table 2-42 presents modeled dermal exposures during conveyorized vapor degreasing use.

Table 2-42. Summary of Dermal Exposure Doses to Methylene Chloride for Conveyorized Vapor Degreasing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Conveyorized Vapor Degreasing	Industrial	1.0	60	180	0.08

a - EPA assumes that 100% methylene chloride is used for vapor degreasing operations.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation using the Latin hypercube sampling method with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from methylene chloride unit emissions and operating hours reported in the 2014 NEI ([EPA, 2018a](#)). The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for two total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.7 Cold Cleaning

EPA found limited inhalation monitoring data for cold cleaning manufacturing from published literature sources. TNO (CIVO) ([1999](#)) indicated that mean exposure values for cold degreasing were found to be approximately 280 mg/m³ on average, ranging from 14 to over 1,000 mg/m³. The referenced data were from United Kingdom (U.K.) Health and Safety Executive (HSE) reports from 1998, but details, including specific worker activities and sampling times were not available. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Because the underlying data were not available, EPA assessed the average value of 280 mg/m³ as the central tendency, and the maximum reported value of 1,000 mg/m³ as the high-end estimate of potential occupational inhalation exposure for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is approximately three times the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is almost 12 times higher. All data points were pre-PEL rule or during the transition period (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC. The results of these calculations are shown in Table 2-43.

Table 2-43. Worker Exposure to Methylene Chloride During Cold Cleaning^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	unknown ^b	280	1,000	Low
Average Daily Concentration (ADC)		64	230	
Lifetime Average Daily Concentration (LADC)		110	510	

Source: [TNO \(CIVO\) \(1999\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

b – One source provided a range of values for an unknown number of samples.

EPA has not identified short-term exposure data from cold cleaning using methylene chloride, nor personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Note that EPA also performed a Monte Carlo simulation with 100,000 iterations using the Latin hypercube sampling method to model near-field and far-field exposure concentrations for the cold cleaning scenario. EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and worst-case estimate of potential occupational inhalation exposures, respectively, for this life cycle stage. For workers, the modeled 8-hr TWA exposures are 1 mg/m³ at the 50th percentile and 103.8 mg/m³ at the 95th percentile. For ONUs, the modeled 8-hr TWA exposures are 0.5 mg/m³ at the 50th percentile and 60 mg/m³ at the 95th percentile. For the risk evaluation, EPA used the available monitoring data for several reasons. The monitoring data have higher weight of evidence due to higher relevance than modeling results for this use for several reasons because the monitoring data are known to be relevant to this use, and the modeled results cannot be validated and do not capture the full range of possible exposure concentrations identified by the monitoring data for this use. For example, the 95th percentile modeling results appear equal to about the 25th percentile of monitoring data. Modeling details are in Appendix F of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Table 2-44 presents modeled dermal exposures during cold cleaning use.

Table 2-44. Summary of Dermal Exposure Doses to Methylene Chloride for Cold Cleaning

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Cold Cleaning	Industrial	1.0	60	180	0.08

a - EPA assumes that 100% methylene chloride is used for cold cleaning operations.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 3 data points from 1 source, and the data quality ratings from systematic review for these data were low. The primary limitations of these data include the age of the data (pre-PEL rule and transition period) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. Additionally, the source reported data from two studies, one of which was presented as a range, and the other presented as a high-end exposure if stringent controls are applied. No data were available to compare pre- and post-PEL rule exposures in Section 2.4.1.1. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.8 Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)

EPA found limited inhalation monitoring data from a published literature source and associated the data with commercial aerosol product applications. Finkel (2017) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with potentially relevant NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

For the set of 21 data points, 8-hr TWA exposure concentrations ranged from 0.1 to 396.5 mg/m³. Worker activity information was not available; therefore, it was not possible to specifically attribute the exposures to aerosol product applications, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as application of paints

and coatings, use of adhesives, and use of paint strippers that contributed to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during aerosol product application. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours.

The central tendency 8-hr TWA exposure concentration is more than an order of magnitude lower than the OSHA PEL value of 87 mg/m³ (25 ppm), while the high-end 8-hr TWA exposure concentrations for this scenario is approximately 3 times the OSHA PEL. Of the 21 data points, 7 of the data points were pre-PEL rule, while 13 data points were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC. The results of these calculations are shown in Table 2-47.

Table 2-45. Worker Exposure to Methylene Chloride During Aerosol Product Applications Based on Monitoring Data^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	21	6.0	230	Medium
Average Daily Concentration (ADC)		1.4	52	
Lifetime Average Daily Concentration (LADC)		2.4	120	

Source: [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

EPA has not identified short-term exposure data from aerosol degreasing using methylene chloride, nor personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

EPA also performed modeling for near-field and far-field exposure concentrations for the aerosol degreasing for both workers and ONUs. Modeling details are in Appendix F of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). Both the central tendency and high-end 8-hr TWA exposure concentrations for workers in this this scenario are lower than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA. ONUs include employees that work at the facility but do not directly apply the aerosol product to

the service item and are therefore expected to have lower inhalation exposures and are not expected to have dermal exposures. ONU exposures are an order of magnitude lower than the worker exposures.

Estimates of ADC and LADC for use in assessing risk were made using the approach and equations described in the Section 2.4.1.1 and are presented in Table 2-46. EPA also modeled maximum 1-hr TWA exposures, which are also shown in the table.

Table 2-46. Statistical Summary of Methylene Chloride 8-hr and 1-hr TWA Exposures (ADC and LADC) for Workers and ONUs for Aerosol Products Based on Modeling

	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
Workers (Near-Field)			
8-hr TWA Exposure Concentration	22	79	N/A – Modeled Data
Average Daily Concentration (ADC)	5.0	18	
Lifetime Average Daily Concentration (LADC)	8.7	40	
Maximum 1-hr TWA Exposures	68	230	
ONUs (Far-Field)			
8-hr TWA Exposure Concentration	0.40	3.3	N/A – Modeled Data
Average Daily Concentration (ADC)	0.09	0.74	
Lifetime Average Daily Concentration (LADC)	0.16	1.7	
Maximum 1-hr TWA Exposures	1.2	9.7	

Table 2-47 presents modeled dermal exposures during commercial aerosol use.

Table 2-47. Summary of Dermal Exposure Doses to Methylene Chloride for Commercial Aerosol Product Uses

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Commercial Aerosol Product Uses	Commercial	1.0	94	280	0.13

a - EPA assumes that 100% methylene chloride is used for commercial aerosol product uses.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of PFs are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data.

For the inhalation air monitoring concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 21 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the age of the data (7 data points pre-PEL rule and 13 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available to specifically attribute exposures to aerosol degreasing or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations increased by 129.7% from pre- to post-rule. Based on these strengths and limitations of the non-spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

For the modeling approach, the primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation using the Latin hypercube sampling method with 100,000 iterations was used to capture the range of potential input parameters. Various model parameters were derived from a California Air Resources Board (CARB) brake service study at 137 automotive maintenance and repair shops in California. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these brake servicing data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA model results in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.9 Adhesives and Sealants

EPA found inhalation exposure data for both spray and non-spray industrial adhesive application, as well as data for unknown application methods. 8-hr TWA data are primarily from Finkel (2017) who submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with potentially relevant NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). For the set of 468 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 2,280 mg/m³. Worker activity information was not available; therefore, it was not possible to specifically attribute the exposures to application of adhesives and sealants, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as application of paints and coatings and use of paint strippers that contribute to methylene

chloride exposures, EPA assumes that exposures are representative of worker exposures during use of adhesives and sealants. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Additional 8-hr TWA data for non-spray uses are primarily from a 1985 EPA Risk Assessment that compiled laminating and gluing activities in various industries, ranging from ND to 575 mg/m³ (97 samples) ([EPA, 1985](#)). A 1984 National Institute for Occupational Safety and Health (NIOSH) Health Hazard Evaluation (HHE) performed at a flexible circuit board manufacturing site encompassed various worker activities in adhesive mixing and laminating areas, ranging from 86.8 to 458.5 mg/m³ (12 samples) ([NIOSH, 1985](#)). 8-hr TWA data for spray uses are available from three sources [TNO \(CIVO\) \(1999\)](#); [WHO \(1996b\)](#); [EPA \(1985\)](#). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*"([EPA, 2019b](#)).

Considering 8-hr TWA samples, 100 personal monitoring samples were available for industrial non-spray adhesives use, 16 personal monitoring samples were available for industrial spray adhesives use, while 468 personal monitoring samples were available for unknown application methods. EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. Central tendency 8-hr TWA exposure concentrations for these scenarios are less than half of the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while high-end estimates are between three and eight times the OSHA PEL. For non-spray application, 98 of the data points were pre-PEL rule, while 2 data points were post-PEL rule. For spray application all 16 data points were from the pre-PEL or transition period (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods). For unknown application methods, 222 of the data points were pre-PEL rule, 49 were during the transition period, while 197 data points were post-PEL rule.

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1. The results of these calculations are shown in Table 2-48, Table 2-49, and Table 2-50 for industrial non-spray, industrial spray, and unknown adhesives application, respectively.

Table 2-48. Worker Exposure to Methylene Chloride During Industrial Non-Spray Adhesives Use^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	100	10	300	High
Average Daily Concentration (ADC)		2.4	67	
Lifetime Average Daily Concentration (LADC)		4.2	150	

Sources: [NIOSH \(1985\)](#); [EPA \(1985\)](#); [OSHA \(2019\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-49. Worker Exposure to Methylene Chloride During Industrial Spray Adhesives Use^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	16	39	560	Low to High
Average Daily Concentration (ADC)		8.9	130	
Lifetime Average Daily Concentration (LADC)		16	290	

Sources: [TNO \(CIVO\) \(1999\)](#); [WHO \(1996b\)](#); [EPA \(1985\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-50. Worker Exposure to Methylene Chloride During Adhesives and Sealants Use (Unknown Application Method)^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	468	27	690	Medium
Average Daily Concentration (ADC)		6.2	160	
Lifetime Average Daily Concentration (LADC)		11	350	

Sources: [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-51 summarizes available short-term exposure data available from the same references and industries identified above for the 8-hr TWA data, as well as OSHA inspection data. Data range from 12 mg/m³ to 720 mg/m³ during adhesive application.

Table 2-51. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Industrial Adhesives Use

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Unknown	OSHA (2019)	Adhesive Sprayer	720	15 ^a	High
			580		
			140		
			480		
			160		
			360		
			100		
			280		
			12		
Flexible Circuit Board Manufacturing	NIOSH (1985)	Operator, laminator #3 & #4, cleaning (Non-Spray)	420	10 ^a	High
		Employee mixing adhesives, Dept 12 (Non-Spray)	570	12 ^a	
Industrial Sign Manufacturing	OSHA (2019)	Laminator	63.4	71 ^b	High

a – EPA evaluated samples with durations ranging from 10 to 15 minutes, as 15-minute exposures.

b – EPA evaluated one sample with duration of 71 minutes as a 1-hr exposure.

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Limited area monitoring data were identified (see Appendix A.6 of the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#))). However, the representativeness of these data for ONU exposures is not clear because of uncertainty concerning the intended sample population and the selection of the specific monitoring location. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-52 presents modeled dermal exposures during adhesives and sealants uses.

Table 2-52. Summary of Dermal Exposure Doses to Methylene Chloride for Adhesives and Sealants Uses

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Adhesives and Sealants Uses	Industrial	1.0	60	180	0.08

a – The 2017 Preliminary Use Document ([U.S. EPA, 2017b](#)) and EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) list commercial products containing between 30 and 100% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the non-spray inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 100 data points from 3 sources, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the age of the data (98 data points pre-PEL rule and 2 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 45.5% from pre- to post-rule. Based on these strengths and limitations of the non-spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium.

For the spray inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the approach hierarchy. These monitoring data include 16 data points from 3 sources, and the data quality ratings from systematic review for these data were low to high. The primary limitations of these data include the age of the data (all data points were from the pre-PEL or transition period) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 45.5% from pre- to post-rule. Based on these strengths and limitations of the spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

For the unknown application inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the approach hierarchy.

These monitoring data include 468 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the age of the data (222 of the data points were pre-PEL rule, 49 were during the transition period, while 197 data points were post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available to specifically attribute exposures to use of adhesives and sealants or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 45.5% from pre- to post-rule. Based on these strengths and limitations of the spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.10 Paints and Coatings

Occupational exposures for use of paints and coatings containing methylene chloride are described in this section. Occupational exposures for methylene chloride-based paint and coating removers were assessed in EPA's TSCA Work Plan Chemical Risk Assessment Methylene Chloride: Paint Stripping Use ([U.S. EPA, 2014](#)), and those results are included in Appendix L. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

EPA found 8-hr TWA spray coating data primarily from monitoring data at various facility types, such as sporting goods stores, metal products, air conditioning equipment, etc., as compiled in the 1985 EPA assessment, ranging from ND to 439.7 mg/m³ (25 data points) ([EPA, 1985](#)). Two additional spray-painting data points were available from OSHA inspections between 2012 and 2016, one in the general automotive repair sector, and the other in the Wood Kitchen Cabinet and Countertop Manufacturing sector, of 14.2 and 222.3 mg/m³ ([OSHA, 2019](#)).

For unknown coating methods, Finkel ([2017](#)) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). For the set of 266 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 3,365 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to the use of paints and coatings, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use of paint strippers that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during use of paints and coatings. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Additional discussion of data treatment is included in Appendix H. The U.S. Department of Defense (DoD) provided five monitoring data points from painting operations during structural repair. The worker activities did not indicate the method of

paint application. The activities were also stated to have low durations (<15 minutes) but provided sampling data that occurred over 2-hr periods. EPA assumed that there was no exposure to methylene chloride over the remainder of the shift and calculated 8-hr TWA exposures; this assumption may not capture the entire exposure scenario, and the calculated result is the minimum exposure during the shift.

Because the method of paint application is unknown, EPA presents the spray application data and the unknown application data separately.

For spray painting/coating operations, 27 personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is below the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, but the high-end estimate is approximately four times higher. Of the 27 data points, 25 were pre-PEL rule, while 2 were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

For unknown application method operations, 271 data points were available. EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is approximately seven times below the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, and the high-end estimate is approximately three times higher. Of the 271 data points, 72 were pre-PEL rule, 49 during the transition period, and 150 were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in the Section 2.4.1.1. The results of these calculations are shown in Table 2-53 and Table 2-54 for spray coating and unknown paint/coating application, respectively.

Table 2-53. Worker Exposure to Methylene Chloride During Paint/Coating Spray Application^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	27	70	360	High
Average Daily Concentration (ADC)		16	83	
Lifetime Average Daily Concentration (LADC)		28	190	

Sources: [OSHA \(2019\)](#); [EPA \(1985\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-54. Worker Exposure to Methylene Chloride During Paint/Coating Application (Unknown Application Method)^a

	Number of Samples	Central Tendency (mg/m³)	High-End (mg/m³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	271	12	260	High and Medium
Average Daily Concentration (ADC)		2.8	60	
Lifetime Average Daily Concentration (LADC)		4.9	130	

Sources: [Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\) \(2018\)](#); [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-55 summarizes available short-term exposure data available from the DoD sampling identified above for the 8-hr TWA data, as well as short-term exposure data during painting at a Metro bus maintenance shop in 1981, and spray painting in a spray booth at a metal fabrication plant in 1973.

Table 2-55. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Paint/Coating Use

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Metro Bus Maintenance Shop	Love and Kern (1981)	Painting	ND (<0.01)	40 ^b	Medium
		Painting	ND (<0.01)	50 ^c	
Metal Fabrication Plant	Vandervort and Polakoff (1973)	Spray Painter in Aisle No. 2 (Front) Spray Booth	64	32 ^b	Medium
			54	32 ^b	
			63	27 ^b	
			36	20 ^a	
			74	29 ^b	
		Spray Painter in Aisle No. 1 (Rear) Spray Booth	1.0	18 ^a	
			3.0	23 ^b	
			4.0	22 ^b	
Department of Defense – Painting and Coating Operations	Defense Occupational and Environmental Health Readiness System - Industrial Hygiene (DOEHRS-IH) (2018)	Painting Operations	4.1	15 ^a	High
		Painting Operations	4.1		
		Painting Operations	4.1		
		Painting Operations	4.1		
		Priming Operations	5.2		
		IND-002-00 Chemical cleaning multi ops.	1.7		
		IND-006-00 Coating Operations, Multiple Operations	1.9		
		IND-006-00 Coating Operations, Multiple Operations	1.9		
		NPS ECE aerosol can painting	13.5		
Industrial Sign Manufacturing	OSHA (2019)	Floor Manager, Painter	133.9	72 ^c	High

ND – not detected

a – EPA evaluated 11 samples, with durations ranging from 15 to 20 minutes, as 15-minute exposures.

b – EPA evaluated seven samples, with durations ranging from of 22 to 32 minutes, as 30-minute exposures.

c – EPA evaluated one sample, with duration of 50 minutes, as 1-hr exposure.
 Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-56 presents modeled dermal exposures during paint and coatings uses.

Table 2-56. Summary of Dermal Exposure Doses to Methylene Chloride for Paint and Coatings Uses

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Paint and Coatings	Industrial	1.0	60	180	0.08

a – The 2016 CDR includes a submission that reports >90% concentration during commercial and consumer use (U.S. EPA, 2016). EPA assumes up to 100% concentration, and that similar concentrations will be used for industrial paints and coatings.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation data. For the spray inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 27 data points from 2 sources, and the data quality ratings from systematic review for these data were high and medium. The primary limitations of these data include the age of the data (25 data points pre-PEL rule and 2 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 47.8% from pre- to post-rule. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium.

For the unknown application method spray inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the approach hierarchy. These monitoring data include 271 data points from two sources, and the

data quality ratings from systematic review for these data were medium and high. The primary limitations of these data include the age of the data (72 data points pre-PEL rule, 49 data points from the transition period, and 150 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available to specifically attribute exposures to the use of paints and coatings or to determine whether sampled activities were representative of full-shift exposures. Based on these strengths and limitations of the spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.11 Adhesive and Caulk Removers

EPA did not find specific industry information exposure data for adhesive and caulk removers. Products listed in EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) indicate potential use in flooring adhesive removal. Based on expected worker activities, EPA assumes that the use of adhesive and caulk removers is similar to paint stripping by professional contractors, as discussed in the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). Therefore, EPA uses the air concentration data from the 2014 Risk Assessment on Paint Stripping Use for Methylene Chloride ([U.S. EPA, 2014](#)).

EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is approximately 17 times the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is almost 34 times higher. All of the data points were pre-PEL rule.

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and shown in Table 2-57.

Table 2-57. Worker Exposure to Methylene Chloride for During Use of Adhesive and Caulk Removers^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	unknown	1,500	3,000	High
Average Daily Concentration (ADC)		350	680	
Lifetime Average Daily Concentration (LADC)		600	1,500	

Source: [U.S. EPA \(2014\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-58 summarizes available short-term exposure data from paint stripping using methylene chloride, which is assumed to be similar to use of adhesive and caulk removers.

Table 2-58. Short-Term Exposure to Methylene Chloride During Use of Adhesive and Caulk Removers

	Number of Samples	Central Tendency (Midpoint) (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
Professional Contractors	unknown	7,100	14,000	High

Source: [U.S. EPA \(2014\)](#)

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA. Durations of the short-term samples in the summary data set are not known.

EPA did not identify personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-59 presents modeled dermal exposures during adhesive and caulk removal.

Table 2-59. Summary of Dermal Exposure Doses to Methylene Chloride for Adhesive and Caulk Removers

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Adhesive and Caulk Removers	Commercial	0.9	85	260	0.13

a – EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) lists commercial products containing up to 90% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include >4 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the age of the data (pre-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. Additional uncertainties are that the data available were compiled from a secondary source, which only presented the high, median, and low values. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.12 Fabric Finishing

Finkel ([2017](#)) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with potentially relevant NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)). For the set of 38 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 331.3 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to fabric finishing process, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use of spot cleaners or general cleaning solvents that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during fabric finishing.

Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Additional discussion of data treatment is included in Appendix H. An additional two data points were provided by OSHA for a presser (0.8 mg/m³ – used as worker exposure) and a finishing department supervisor (1.2 mg/m³ – used as ONU exposure) (OSHA, 2019). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Overall, 39 personal monitoring data samples were available for workers and one sample available for ONUs; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for workers is approximately one order of magnitude less than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate for workers is approximately twice the PEL value. Exposure concentrations for ONUs based on the single data point are an order of magnitude less than the PEL value. Of the 39 worker data points, 25 were pre-PEL rule, 10 were from the transition period, and 4 were post-PEL rule. The single ONU data point was post-PEL (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and shown in Table 2-60.

Table 2-60. Worker and ONU Exposure to Methylene Chloride During Fabric Finishing

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
<i>Workers</i>				
8-hr TWA Exposure Concentration	39	7.8	140	Medium and High
Average Daily Concentration (ADC)		1.8	31	
Lifetime Average Daily Concentration (LADC)		3.1	70	
<i>Occupational Non-Users</i>				
8-hr TWA Exposure Concentration	1	1.2		High
Average Daily Concentration (ADC)		0.27		
Lifetime Average Daily Concentration (LADC)		0.47	0.61	

Source: [Finkel \(2017\)](#); [OSHA \(2019\)](#).

Table 2-61 summarizes available short-term exposure data available from OSHA inspections

Table 2-61. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Fabric Finishing

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
All Other Leather Good and Allied Product Manufacturing	OSHA (2019)	Sprayer of Methylene Chloride	10	194 ^a	High

a – As there are no health comparisons for 2- or 3-hr samples, this data point is presented but not used to calculate risk.

Table 2-62 presents modeled dermal exposures during fabric finishing.

Table 2-62. Summary of Dermal Exposure Doses to Methylene Chloride for Fabric Finishing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Fabric Finishing	Commercial	0.95	90	270	0.13

a – EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) lists commercial products containing up to 95% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the worker inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 39 data points from 2 sources, and the data quality ratings from systematic review for these data were medium (38 data points) and high (1 data point). The primary limitations of these data include the age of the data (25 data points pre-PEL rule, 10 data points from the transition period, and 4 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by

this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to fabric finishing or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 93.4% from pre- to post-rule. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for the worker 8-hr TWA data in this scenario is low.

For the ONU inhalation air concentration data, the primary strength is the use of post-PEL monitoring data, the highest of the inhalation approach hierarchy. The primary limitation is that only one data point is available. The uncertainty of the representativeness of this data point toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the ONU inhalation air concentration data, the overall confidence for the ONU 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.13 Spot Cleaning

Finkel (2017) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes for Industrial Launderers and Drycleaning and Laundry Services (except Coin-Operated). For the set of 18 data points, 8-hr TWA exposure concentrations ranged from 0.1 to 410.4 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to spot cleaning, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use general cleaning solvents that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during spot cleaning. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency value was two orders of magnitude less than the OSHA PEL value of 87 mg/m³ (25 ppm), while the high end value was approximately two times the OSHA PEL. Of the 18 data points, 14 were pre-PEL rule, 1 was from the transition period, and 3 were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods). Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and shown in Table 2-63.

Table 2-63. Worker Exposure to Methylene Chloride for During Spot Cleaning^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	18	0.67	190	Medium
Average Daily Concentration (ADC)		0.15	42	
Lifetime Average Daily Concentration (LADC)		0.26	95	

Source: [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

EPA has not identified personal or area data on short term exposures or potential ONU inhalation exposures. EPA has developed a model to evaluate potential worker and ONU exposures during spot cleaning for various solvents; however, the specific methylene chloride use rate during spot cleaning was not reasonably available. This is a critical data gap and other solvent use rates may not be applicable. EPA classified retail sales workers (e.g., cashiers), sewers, tailors, and other textile workers as “occupational non-users” because they perform work at the dry cleaning shop, but do not directly handle dry cleaning solvents. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-64 presents modeled dermal exposures during spot cleaning.

Table 2-64. Summary of Dermal Exposure Doses to Methylene Chloride for Spot Cleaning

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Spot Cleaning	Commercial	0.9	85	260	0.13

a – EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) lists commercial products containing up to 90% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 18 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the age of some data (15 data points pre-PEL rule or transition period and 3 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to spot cleaning or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 94.5% from pre- to post-rule. Additionally, the data source did not specify specific worker activities; therefore, the representativeness of these data specifically for spot cleaning is also uncertain. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium to low (full discussion in Section 2.4.1.3).

2.4.1.2.14 Cellulose Triacetate Film Production

EPA found 8-hr TWA data primarily from six studies performed in the 1970s and 1980s. Worker activities encompassed various areas of CTA production, including preparation, extrusion, and coating, but each study compiled data into overall statistics for each worker type instead of presenting separate data points (Ott et al., 1983a); (Dell et al., 1999); (TNO (CIVO), 1999). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Because the individual data points were not available, EPA presents the average of the median, and average of maximum values as central tendency and high end, respectively, in Table 2-75. The central tendency and high end 8-hr TWA exposure concentrations for this scenario are approximately 12 to 16 times the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, respectively. All of the data points were pre-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and shown in Table 2-65 for CTA film manufacturing.

Table 2-65. Worker Exposure to Methylene Chloride During CTA Film Manufacturing^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	>166 ^b	1,000	1,400	Medium and Low
Average Daily Concentration (ADC)		240	320	
Lifetime Average Daily Concentration (LADC)		410	560	

Sources: [Dell et al. \(1999\)](#); [TNO \(CIVO\) \(1999\)](#); [Ott et al. \(1983a\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

b – Various studies were compiled to determine central tendency and high-end estimates; however, not all indicated the number of samples. Therefore, actual number of samples is unknown.

Specific short-term data or personal or area data on or parameters for modeling potential ONU inhalation exposures were not found. Since ONUs do not directly handle methylene chloride, ONU inhalation exposures could be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-66 presents estimated dermal exposures during CTA film manufacturing.

Table 2-66. Summary of Dermal Exposure Doses to Methylene Chloride for CTA Film Manufacturing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
CTA Film Manufacturing	Industrial	1	60	180	0.08

a – EPA assumes methylene chloride is received at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include >166 data points from 3 sources, and the data quality ratings from systematic review for these

data were medium and low. The primary limitations of these data include the age of the data (all data were pre-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. An additional uncertainty for these sources is that only concentration ranges were provided rather than discrete data points. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.15 Flexible Polyurethane Foam Manufacturing

EPA found 8-hr TWA data from various sources, and cover activities such as application of mold release, foam manufacturing (blowing), blending, and sawing in the foam or plastic industry and tractor trailer construction. Exposures varied from 0.3 mg/m³ from purge operations, to 2,200.9 mg/m³ during laboratory operations ([IARC, 2016](#); [TNO \(CIVO\), 1999](#); [WHO, 1996b](#); [Vulcan Chemicals, 1991](#); [Reh and Lushniak, 1990](#); [EPA, 1985](#); [Cone Mills Corp, 1981a, b](#); [Olin Chemicals, 1977](#)). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Overall, 84 8-hr TWA personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is approximately 2.5 times higher than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is almost 12 times higher. Of the 84 data points, 77 were pre-PEL rule, 4 were from the transition period, and 3 were post-PEL rule (see Section 2.4.1.12.4.1.1 for pre-PEL, transition, and post-PEL rule periods). There appear to be many diverse uses of methylene chloride in the PU foam manufacturing industry, which may contribute to the wide range of exposure concentrations.

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC. The results of these calculations are shown in Table 2-67.

Table 2-67. Worker Exposure to Methylene Chloride During Industrial Polyurethane Foam Manufacturing^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	84	190	1,000	High to Low
Average Daily Concentration (ADC)		44	230	
Lifetime Average Daily Concentration (LADC)		76	510	

Sources: [IARC \(2016\)](#); [TNO \(CIVO\) \(1999\)](#); [WHO \(1996b\)](#); [Vulcan Chemicals \(1991\)](#); [Reh and Lushniak \(1990\)](#); [Cone Mills Corp \(1981a\)](#); [Cone Mills Corp \(1981b\)](#); [EPA \(1985\)](#); [Olin Chemicals \(1977\)](#); [OSHA \(2019\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-68 summarizes available short-term exposure data available from the 1985 EPA assessment.

Table 2-68. Summary of Personal Short-Term Exposure Data for Methylene Chloride During Polyurethane Foam Manufacturing

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Polyurethane Foam Manufacturing	EPA (1985)	Foam Blowing	5.2	360 ^a	High
		Foam Blowing	13	360 ^a	
		Foam Blowing	19	360 ^a	
		Foam Blowing	17	360 ^a	
		Foam Blowing	5.2	360 ^a	
		Foam Blowing	38	360 ^a	
		Foam Blowing	11	360 ^a	
		Nozzle Cleaning	55	30 ^b	

a – As there are no health comparisons for 6-hr samples, these data points are presented but not used to calculate risk

b – EPA evaluated one sample, with a 30-minute duration, as a 30-minute exposure.

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-69 presents modeled dermal exposures during polyurethane foam blowing.

Table 2-69. Summary of Dermal Exposure Doses to Methylene Chloride for Polyurethane Foam Manufacturing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Polyurethane Foam Manufacturing	Industrial	1	60	180	0.08

a – EPA assumes workers may be exposed to 100% methylene chloride solvent during equipment cleaning.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. In addition to the uncertainties identified for this scenario discussed in Section 4.4.2, regulations have limited the use of methylene chloride in polyurethane foam production and fabrication. OAR's July 16, 2007 Final National Emissions Standards for Hazardous Air Pollutants (NESHAP) for Area Sources: Polyurethane Foam Production and Fabrication (72 FR 38864) prohibited the use of methylene chloride-based mold release agents at molded and rebond foam facilities, methylene chloride-based equipment cleaners at molded foam facilities, and the use of methylene chloride to clean mix heads and other equipment at slabstock facilities. Slabstock area source facilities are required to comply with emissions limitations for methylene chloride used as an auxiliary blowing agent, install controls on storage vessels, and comply with management practices for equipment leaks. The rule also prohibits methylene chloride-based adhesives for foam fabrication. The effect of these rules on current exposure levels is unclear.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation data. The primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 82 data points from 9 sources, and the data quality ratings from systematic review for these data were high to low. The primary limitations of these data include the age of the data (77 data points pre-PEL rule, 4 transition period, and 3 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by

this scenario. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. An additional uncertainty is that some sources provided only concentration ranges rather than discrete data points. Based on these strengths and limitations of the non-spray inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.16 Laboratory Use

Finkel (2017) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with potentially relevant NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). For the set of 65 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 371.4 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to laboratory activities, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use cleaning solvents that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during laboratory use. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. EPA also found 8-hr TWA data from a 1989 NIOSH inspection of an analytical laboratory at Texaco (Texaco Inc, 1993), and from the U.S. Department of Defense (DoD) (Defense Occupational and Environmental Health Readiness System - Industrial Hygiene (DOEHRS-IH), 2018). Worker descriptions include laboratory staff, and activities include sample preparation and transfer. Note that the NIOSH data were for various sample durations; EPA included samples that were more than 4 hrs long as full-shift exposures and adjusted the exposures to 8-hr TWAs, assuming that the exposure concentration for the remainder of the time was zero, because workers were not expected to perform the activities all day. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Overall, 76 8-hr TWA personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentration for this scenario is an order of magnitude lower than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is slightly above the PEL value. Of the 76 data points, 23 were pre-PEL rule, 15 were during the transition period and 38 were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and are summarized in Table 2-70.

Table 2-70. Worker Exposure to Methylene Chloride During Laboratory Use^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	76	6.0	100	High and Medium
Average Daily Concentration (ADC)		1.4	23	
Lifetime Average Daily Concentration (LADC)		2.4	52	

Sources: [Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\) \(2018\)](#); [Texaco Inc \(1993\)](#); [Mccammon \(1990\)](#); [OSHA \(2019\)](#); [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-71 summarizes short-term exposure data available from the same inspections identified above for the 8-hr TWA data, as well as OSHA inspection data.

Table 2-71. Worker Personal Short-Term Exposure Data for Methylene Chloride During Laboratory Use

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Analytical Laboratory	Mccammon (1990)	sample concentrating	2.7	233 ^d	Medium
		sample sonification	3.9	218 ^d	
		sample sonification	4.5	218 ^d	
		washing separatory funnels in sink near continuous liquid/liquid extraction	110	10 ^a	
		column cleaning	10	200 ^d	
		sample concentrating	30	210 ^d	
		sample concentrating	4.2	234 ^d	
		sample concentrating	6.8	198 ^e	
		transferring 100 mL methylene chloride into soil samples	9.8	115 ^d	
		collecting waste chemicals & dumping into waste chemical storage	1,000	24 ^b	
	Defense Occupational and Environmental Health Readiness System -	Miscellaneous lab operations	3.1	244 ^d	High
		Miscellaneous lab operations	3.1	238 ^d	
		Sample extraction and analysis (3809, OCD)	34.7	180 ^e	

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
	Industrial Hygiene (DOEHRS-IH) (2018)	(3)Gas Chromatography (GC) Extraction	0.7	154 ^c	
		134: Extraction of PCB in water samples (Rm 221 - Prep & Rm 227 - GC)	22.5	130 ^c	
		134: Extraction of total volatiles (Toxicity Characteristic Leaching Procedure (TCLP)) (Rm 227)	64.7	130 ^c	
		Analysis, chemical (Laboratory Operations)	1.7	59 ^c	
		Analysis, chemical (Laboratory Operations)	2.4	48 ^c	
		LAB ACTIVITIES	3.3	31 ^b	
		LAB ACTIVITIES	6.4	30 ^b	
		LAB ACTIVITIES	16.6	30 ^b	
		LAB ACTIVITIES	3.4	30 ^b	
		LAB ACTIVITIES	3.4	30 ^b	
		LAB ACTIVITIES	3.4	30 ^b	
		LAB ACTIVITIES	3.4	30 ^b	
		LAB ACTIVITIES	3.4	30 ^b	
		PRO-001-01 LABORATORY CHEMICAL ANALYSIS/SAMPLING	5.4	30 ^b	
		514A Using Solvents	1830.0	25 ^b	
		EXTRACTION OP	3.6	19 ^a	
		EXTRACTION OP	24.8	19 ^a	
		(3)GC Extraction	10.4	15 ^a	
		(3)GC Extraction	10.4	15 ^a	
		Sample extraction and analysis (3809, OCD)	62.5	15 ^a	
		Miscellaneous lab operations	6.7	15 ^a	
		EXTRACTION OP	4.6	15 ^a	
		EXTRACTION OP	4.6	15 ^a	
		134: Extraction of PCB in water samples (Rm 221 - Prep & Rm 227 - GC)	5.3	15 ^a	
		134: Extraction of total volatiles (TCLP) (Rm 227)	5.0	15 ^a	
		PRO-001-01 LABORATORY CHEMICAL ANALYSIS/SAMPLING	5.4	15 ^a	

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
		IND-025-10 HM/HW HANDLING CLEANUP, CONTAINER SAMPLE/OPEN	6.1	15 ^a	
		PRO-001-01 LABORATORY CHEMICAL ANALYSIS/SAMPLING	10.9	15 ^a	
		PRO-001-01 LABORATORY CHEMICAL ANALYSIS/SAMPLING	13.2	15 ^a	
Laboratory	OSHA (2019)	Organic Prep Lab Tech	ND	53 ^f	High
		Organic Prep Lab Tech	ND	49 ^f	

a – EPA evaluated 15 samples, with durations ranging from 10 to 19 minutes, as 15-minute exposures.

b – EPA evaluated 10 samples, with durations ranging from 24 to 31 minutes, as 30-minute exposures.

c – EPA evaluated two samples, with durations ranging from 48 to 59 minutes, as 1-hr exposures.

d – EPA evaluated six samples, with durations ranging from 218 to 244 minutes, as 4-hr exposures.

e – As there are no health comparisons for 2- or 3-hr samples, these data points are presented but not used to calculate risk.

f – Limit of detection was not provided for these samples, so they were not used to evaluate risk.

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle products containing methylene chloride, ONU inhalation exposures could be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-72 presents modeled dermal exposures during laboratory use.

Table 2-72. Summary of Dermal Exposure Doses to Methylene Chloride for Laboratory Use

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Laboratory Use	Commercial	1	94	280	0.13

a – EPA's Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)) lists commercial products containing up to 100% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 76 data points from 5 sources, and the data quality ratings from systematic review for these data were high and medium. The primary limitations of these data include the age of some of the data (23 were pre-PEL rule, 15 were during the transition period and 38 were post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to laboratory activities or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 38.9% from pre- to post-rule. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.17 Plastic Product Manufacturing

Finkel (2017) submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with potentially relevant NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). For the set of 32 data points, 8-hr TWA exposure concentrations ranged from 0.1 to 1,637.3 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to the plastic manufacturing process, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use of adhesives or cleaning solvents that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during plastics manufacturing. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. HSIA provided an additional 20 data points from 2005 through 2017, for production technicians during plastic product manufacturing. Exposure concentrations ranged from 3.9 to 134.1 mg/m³ (20 samples) (Halogenated Solvents Industry Alliance, 2018). Additional data were found for various other sources that ranged from 9 mg/m³ to 2,685.1 mg/m³ (for hop area operator) (Fairfax and Porter, 2006); (WHO, 1996b); (Halogenated Solvents Industry Alliance, 2018); (General Electric Co, 1989). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for*

Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment" ([EPA, 2019b](#)).

Overall for the 8-hr TWA, 62 personal monitoring data samples were available for workers, and two samples were available for ONUs (although one sample was for an OSHA inspector and may or may not be reflective of industry ONUs); ONUs are employees who work at the facilities that process and use methylene chloride, but who do not directly handle the material. ONUs may also be exposed to methylene chloride but are expected to have lower inhalation exposures and are not expected to have dermal exposures. ONUs for this condition of use may include supervisors, managers, engineers, and other personnel in nearby production areas. EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentrations for workers and ONUs is approximately ten times lower the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate for workers is approximately two times higher. Of the 62 worker data points, 18 were pre-PEL rule, 3 were transition period, and 41 were post-PEL rule. The ONU exposure values were post-PEL (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods)

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and are summarized in Table 2-73.

Table 2-73. Worker and ONU Exposure to Methylene Chloride During Plastic Product Manufacturing

Exposure	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
Workers				
8-hr TWA Exposure Concentration	62	8.5	210	High to Low
Average Daily Concentration (ADC)		1.9	47	
Lifetime Average Daily Concentration (LADC)		3.4	110	
ONUs				
8-hr TWA Exposure Concentration	2	9.7	10	High
Average Daily Concentration (ADC)		2.2	2.3	
Lifetime Average Daily Concentration (LADC)		3.9	5.3	

Sources: [OSHA \(2019\)](#); [Halogenated Solvents Industry Alliance \(2018\)](#); [Fairfax and Porter \(2006\)](#); [\(IPCS\) \(1996\)](#); [General Electric Co \(1989\)](#); [Finkel \(2017\)](#)

Table 2-74 summarizes available short-term exposure data for workers and ONUs from the same OSHA inspections identified above for the 8-hr TWA data, as well as short-term data provided by HSIA ([2018](#)). EPA has not identified area data on or parameters for modeling potential ONU inhalation exposures.

Table 2-74. Worker Short-Term Exposure Data for Methylene Chloride During Plastic Product Manufacturing

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Plastic Product Manufacturing	OSHA (2019)	Plastics Manufacturer	ND	15 ^a	High
			28	15 ^a	
			21	20 ^a	
Plastics Material and Resin Manufacturing	Halogenated Solvents Industry Alliance (2018)	Operator	100	13 ^a	High
		Operator	74	18 ^a	
		Operator	94	14 ^a	
		Operator	66	20 ^a	
		Operator	66	20 ^a	
		Operator	60	22 ^b	
		Operator	130	10 ^a	
		Operator	66	20 ^a	
		Operator	100	13 ^a	
		Operator	170	8 ^a	
		Operator	110	12 ^a	
		Operator	83	15 ^a	
		Product technician	120	11 ^a	
		Product technician	69	19 ^a	
		Product technician	83	16 ^a	
		Product technician	63	21 ^a	
		Product technician	88	15 ^a	
Product technician	83	16 ^a			
Product technician	100	13 ^a			
Product technician	110	12 ^a			
Product technician	51	26 ^b			
Plastics Material and Resin Manufacturing	OSHA (2019)	CSHO	ND	92 ^c	High
		Extruder Operator	20.4	313 ^d	

a – EPA evaluated 21 samples, with durations ranging from 8 to 21 minutes, as 15-minute exposures.

b – EPA evaluated 10 samples, with durations ranging from 22 to 26 minutes, as 30-minute exposures.

c – Limit of detection was not provided for this sample, so it was not used to evaluate risk.

d – As there are no health comparisons for ~5-hr samples, this data point is presented but not used to calculate risk.

Note: The OSHA STEL is 433 mg/m³ as a 15-min TWA.

Table 2-75 presents estimated dermal exposures during plastic product manufacturing.

Table 2-75. Summary of Dermal Exposure Doses to Methylene Chloride for Plastic Product Manufacturing

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Plastic Product Manufacturing	Industrial	1	60	180	0.08

a – EPA assumes methylene chloride is received at 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the worker inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 62 data points from 6 sources, and the data quality ratings from systematic review for these data were high to low. The primary limitations of these data include the age of some the data (18 data points pre-PEL rule, 3 data points transition period, and 41 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to plastics manufacturing or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations increased by 617% from pre- to post-rule. Based on these strengths and limitations of the worker inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low.

For the ONU inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 2 data points from 1 source, and the data quality ratings from systematic review for these data points was high. The primary limitations of these data points include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Both of the data points were post-PEL rule. Based on these strengths and limitations of the inhalation air

concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.18 Lithographic Printing Plate Cleaning

8-hr TWA data are primarily from Finkel (2017), who submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). For the set of 50 data points, 8-hr TWA exposure concentrations ranged from 0.01 to 167 mg/m³. Worker activity information was not available; therefore, it was not possible to specifically attribute the exposures to use as a lithographic printing plate cleaner, nor to distinguish workers from ONUs. While additional activities are possible at these sites, such as use of inks or coatings that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during lithographic printing plate cleaning. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. EPA found additional 8-hr TWA inhalation monitoring data from the 1985 EPA assessment covering various printers and activities, which ranged from ND (during printing) to 547.9 mg/m³ (during screen making for commercial letterpress) (44 data points) (EPA, 1985). Additional data were also obtained from a 1998 occupational exposure study and a 1980 NIOSH inspection of a printing facility (Ukai et al., 1998); (Ahrenholz, 1980). Exposure data were for workers involved in the printing plate/roll cleaning. The 1998 occupational exposure study only presented the min, mean, and max values for 61 samples, while the 1980 NIOSH inspection included two full-shift readings (ND to 17.0 mg/m³; ND was assessed as zero). Minimum and maximum values from reported ranges were used as discrete data points, while calculated statistics such as mean values were excluded. Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Overall, EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and worst-case estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentrations for this scenario is one order of magnitude lower than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate is approximately three times higher. Of the 130 worker data points, 98 were pre-PEL rule, 11 were from the transition period, and 21 were post-PEL rule.

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC. The results of these calculations are shown in Table 2-76 for workers during plastic product manufacturing.

Table 2-76. Worker Exposure to Methylene Chloride During Printing Plate Cleaning^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	>130 ^b	8.7	160	High and Medium
Average Daily Concentration (ADC)		2.0	37	
Lifetime Average Daily Concentration (LADC)		3.5	82	

Sources: [Ukai et al. \(1998\)](#); [EPA \(1985\)](#); [Ahrenholz \(1980\)](#); [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

b – One study indicated that statistics were based on 61 samples, but only provided the minimum, maximum, and mean values. Another study provided two exposure values, one of which was ND. ND was assessed as zero

Table 2-77 summarizes the available 4-hr TWA exposure data for workers from the same source identified above for the 8-hr TWA data. Data were taken in two 4-hr shifts.

Table 2-77. Worker Short-Term Exposure Data for Methylene Chloride During Printing Plate Cleaning

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min) ^a	Data Quality Rating of Associated Air Concentration Data
Lithographic Printing Plate Cleaning	Ukai et al. (1998)	Cleaning of printing rolls / solvent in production	3.5	240	Medium
			940		
			3.6		
			480		

a – EPA evaluated these samples as 4-hr exposures.

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-78 presents estimated dermal exposures during lithographic printing plate cleaning.

Table 2-78. Summary of Dermal Exposure Doses to Methylene Chloride for Lithographic Printing Plate Cleaner

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y_{derm}^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Lithographic Printing Plate Cleaner	Commercial	0.885	84	250	0.13

a – The 2017 Preliminary Use Document ([U.S. EPA, 2017b](#)) lists commercial/industrial products containing up to 88.5% methylene chloride.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include >130 data points from 4 sources, and the data quality ratings from systematic review for these data were high and medium. The primary limitations of these data include the age of the data (98 were pre-PEL rule, 11 were from the transition period, and 21 were post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to lithographic printing plate cleaning or to determine whether sampled activities were representative of full-shift exposures. A comparison of pre- and post-rule OSHA data (summarized in Table 2-26) shows that mean exposure concentrations decreased by 47.7% from pre- to post-rule. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.19 Miscellaneous Non-Aerosol Industrial and Commercial Uses

EPA compiled various monitoring data for miscellaneous non-aerosol industrial and commercial settings, including 8-hr TWA data. 8-hr TWA data are from various OSHA inspection at wholesalers and retail stores, and include generic worker activities, such as plant workers, service workers, laborers, etc. Exposure concentrations for various workers ranged from ND to 1,294.8 mg/m³ (EPA, 1985). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM)*"

CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment" ([EPA, 2019b](#)).

Overall, 108 personal monitoring data samples were available; EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively, for this scenario. The central tendency 8-hr TWA exposure concentrations for workers is approximately three times higher than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA, while the high-end estimate for workers is more than nine times higher. All 108 data points were pre-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1. The results of these calculations are shown in Table 2-79 for workers during plastic commercial non-aerosol use.

Table 2-79. Worker Exposure to Methylene Chloride During Miscellaneous Industrial and Commercial Non-Aerosol Use^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	108	57	930	High
Average Daily Concentration (ADC)		13	210	
Lifetime Average Daily Concentration (LADC)		23	480	

Sources: [EPA \(1985\)](#).

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

EPA has not identified short-term exposure data or personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-80 presents estimated dermal exposures during industrial and commercial non-aerosol use.

Table 2-80. Summary of Dermal Exposure Doses to Methylene Chloride for Miscellaneous Industrial and Commercial Non-Aerosol Use

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, $Y_{\text{derm}}^{\text{a}}$	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F_{abs}
			Central Tendency	High End	
Miscellaneous Industrial Non-Aerosol Use	Industrial	1	60	180	0.08
Miscellaneous Commercial Non-Aerosol Use	Commercial	1	94	280	0.13

a – EPA assumes exposure to methylene chloride at up to 100% concentration.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 108 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the age of the data (all data points were pre-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.2.20 Waste Handling, Disposal, Treatment, and Recycling

8-hr TWA data are primarily from Finkel (2017), who submitted workplace monitoring data obtained from a FOIA request of OSHA. EPA extracted relevant monitoring data by crosswalking the Standard Industrial Classification (SIC) codes in the dataset with the NAICS codes as discussed in the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b). For the set of 15 data points, 8-hr TWA exposure concentrations ranged from 0.11 to 107 mg/m³. Worker activity information was not available; therefore it was not possible to specifically attribute the exposures to waste handling activities, nor to distinguish workers from ONUs. While additional activities are possible at these

sites, such as use of cleaning solvents that contribute to methylene chloride exposures, EPA assumes that exposures are representative of worker exposures during waste handling. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. EPA's 1985 assessment included three full-shift data points for solvent reclaimers at solvent recovery sites, ranging from 10.5 to 19.2 mg/m³ (EPA, 1985). The U.S. Department of Defense (DoD) also provided four data points during waste disposal and sludge operations ranging from 0.4 to 2.3 mg/m³ (Defense Occupational and Environmental Health Readiness System - Industrial Hygiene (DOEHRS-IH), 2018). Lists of all inhalation monitoring data found in data sources and associated systematic review data quality ratings are available in Appendix A of the supplemental document "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" (EPA, 2019b).

Overall for the 8-hr TWA samples, 22 personal monitoring data samples were available; EPA assessed the 50th percentile value of 2.3 mg/m³ as the central tendency, and the 95% percentile value of 81 mg/m³ as the high-end estimate of potential occupational inhalation exposures for this life cycle stage. The central tendency exposure concentration for this scenario is an order of magnitude lower than the OSHA PEL value of 87 mg/m³ (25 ppm) as an 8-hr TWA and high-end 8-hr TWA exposure concentration is slightly lower than the PEL. Of the 22 data points, 18 were pre-PEL rule, while 4 were post-PEL rule (see Section 2.4.1.1 for pre-PEL, transition, and post-PEL rule periods).

Using these 8-hr TWA exposure concentrations, EPA calculated the ADC and LADC as described in Section 2.4.1.1 and are summarized in Table 2-81.

Table 2-81. Worker Exposure to Methylene Chloride During Waste Handling and Disposal^a

	Number of Samples	Central Tendency (mg/m ³)	High-End (mg/m ³)	Data Quality Rating of Associated Air Concentration Data
8-hr TWA Exposure Concentration	22	2.3	81	High and Medium
Average Daily Concentration (ADC)		0.54	18	
Lifetime Average Daily Concentration (LADC)		0.93	41	

Source: [Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\) \(2018\)](#); [EPA \(1985\)](#); [Finkel \(2017\)](#)

a – No data for ONUs were found; EPA assumes that ONU exposures are less than worker exposures.

Table 2-82 summarizes the available short-term exposure data for workers from the DoD data.

Table 2-82. Worker Short-Term Exposure Data for Methylene Chloride During Waste Handling and Disposal

Occupational Exposure Scenario	Source	Worker Activity	Methylene Chloride Short-Term Concentration (mg/m ³)	Exposure Duration (min)	Data Quality Rating of Associated Air Concentration Data
Waste Handling	Defense Occupational and Environmental Health Readiness System - Industrial Hygiene (DOEHRS-IH) (2018)	Transfer of solvent during waste disposal	2.9	30 ^a	High
			2.9	30 ^a	
			1.8	144 ^b	
			5.8	158 ^b	
			2.7	159 ^b	
			2.8	163 ^b	
			0.8	173 ^b	
		3.4	156 ^b		

a – EPA evaluated two 30-minute samples as 30-minute exposures.

b – As there are no health comparisons for 2- or 3-hr samples, these data points are presented but not used to calculate risk

Note: The OSHA Short-term exposure limit (STEL) is 433 mg/m³ as a 15-min TWA.

EPA has not identified personal or area data on or parameters for modeling potential ONU inhalation exposures. Since ONUs do not directly handle formulations containing methylene chloride, EPA expects ONU inhalation exposures to be lower than worker inhalation exposures. Information on processes and worker activities are insufficient to determine the proximity of ONUs to workers and sources of emissions, so relative exposure of ONUs to workers cannot be quantified.

Table 2-83 presents estimated dermal exposures during waste handling, disposal, treatment and recycling.

Table 2-83. Summary of Dermal Exposure Doses to Methylene Chloride for Waste Handling, Disposal, Treatment, and Recycling

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y _{derm} ^a	Dermal Exposure Dose (mg/day) ^b		Calculated Fraction Absorbed, F _{abs}
			Central Tendency	High End	
Waste Handling, Disposal, Treatment, and Recycling	Industrial	1	60	180	0.08

a – EPA assumes potential exposure to methylene chloride at 100% concentration for recovered solvent.

b – Conditions where no gloves are used, or for any glove / gauntlet use without permeation data and without employee training (PF = 1).

Potential impacts of protection factors are presented as what-if scenarios in the dermal exposure summary Table 2-85.

In summary, dermal and inhalation exposures are expected for this scenario. EPA has described uncertainties for this scenario in Section 4.4.2.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 22 data points from 3 sources, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the age of some of the data (18 data points pre-PEL rule and 4 data points post-PEL rule) and uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. As discussed earlier in this section, key metadata such as worker activity and sampling descriptions were not available in the Finkel (2017) dataset to specifically attribute exposures to waste handling or to determine whether sampled activities were representative of full-shift exposures. The analysis of pre- and post-rule OSHA data (summarized in Table 2-26) did not have enough data to compare pre- to post-rule mean exposure concentrations for this OES. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. The overall confidence of the dermal dose results is medium (full discussion in Section 2.4.1.3).

2.4.1.3 Summary of Occupational Exposure Assessment

The following tables summarize the exposures estimated for the inhalation (Table 2-84) and dermal (Table 2-85) routes for all occupational exposure scenarios, assuming no exposure reductions due to potential PPE use.

Table 2-84. Summary of Acute and Chronic Inhalation Exposures to Methylene Chloride for Central and Higher-End Scenarios by Occupational Exposure Scenario

Occupational Exposure Scenario	Category ^a	Acute Exposures		Chronic, Non-Cancer Exposures		Chronic, Cancer Exposures		Overall Confidence Rating of Acute Exposure Concentrations
		AEC, 8- or 12-hr TWA (mg/m ³)		ADC, 24-hr TWA (mg/m ³)		LADC, 24-hr TWA (mg/m ³)		
		Central Tendency	High End	Central Tendency	High End	Central Tendency	High End	
Manufacturing (8-hr TWA)	Worker	0.36	4.6	0.08	1.1	0.14	2.4	Medium to High
Manufacturing (12-hr TWA)	Worker	0.45	12	0.15	4.1	0.27	9.3	Medium to High
Processing as a Reactant	Worker	1.6	110	0.37	25	0.65	55	Low
Processing - Incorporation into Formulation	Worker	100	540	23	120	40	280	Low
Repackaging	Worker	8.8	140	2.0	31	3.50	71	Medium to Low

Occupational Exposure Scenario	Category ^a	Acute Exposures		Chronic, Non-Cancer Exposures		Chronic, Cancer Exposures		Overall Confidence Rating of Acute Exposure Concentrations
		AEC, 8- or 12-hr TWA (mg/m ³)		ADC, 24-hr TWA (mg/m ³)		LADC, 24-hr TWA (mg/m ³)		
		Central Tendency	High End	Central Tendency	High End	Central Tendency	High End	
Batch Open-Top Vapor Degreasing	Worker	170	740	38	170	67	380	Medium to Low
Batch Open-Top Vapor Degreasing	ONU	86	460	20	100	34	230	Medium to Low
Conveyorized Vapor Degreasing	Worker	490	1,400	110	320	190	720	Medium to Low
Conveyorized Vapor Degreasing	ONU	250	900	58	210	100	460	Medium to Low
Cold Cleaning	Worker	280	1,000	64	230	110	510	Medium to Low
Aerosol Degreasing/Lubricants (Monitoring)	Worker & ONU	6.0	230	1.4	52	2.4	120	Medium to Low
Aerosol Degreasing/Lubricants (Modeled)	Worker	22	79	5.0	18	8.7	40	Medium to Low
Aerosol Degreasing/Lubricants (Modeled)	ONU	0.40	3.3	0.09	0.74	0.16	1.7	Medium to Low
Adhesives (Spray)	Worker	39	560	8.9	130	16	290	Medium to Low
Adhesives (Non-Spray)	Worker	10	300	2.4	67	4.2	150	Medium
Adhesives/Sealants (Unknown Application)	Worker & ONU	27	690	6.2	160	11	350	Low
Paints and Coatings (Spray)	Worker	70	360	16	83	28	190	Medium
Paints and Coatings (Unknown Application Method)	Worker	12	260	2.8	60	4.9	130	Low
Adhesive and Caulk Removers	Worker	1,500	3,000	350	680	600	1,500	Medium to Low
Fabric Finishing	Worker	7.8	140	1.8	31	3.1	70	Low
Fabric Finishing	ONU	1.2		0.27		0.47	0.61	Low
Spot Cleaning	Worker	0.67	190	0.15	42	0.26	95	Low
CTA Manufacturing	Worker	1,000	1,400	240	320	410	560	Low
Flexible PU Foam Manufacturing	Worker	190	1,000	44	230	76	510	Medium

Occupational Exposure Scenario	Category ^a	Acute Exposures		Chronic, Non-Cancer Exposures		Chronic, Cancer Exposures		Overall Confidence Rating of Acute Exposure Concentrations
		AEC, 8- or 12-hr TWA (mg/m ³)		ADC, 24-hr TWA (mg/m ³)		LADC, 24-hr TWA (mg/m ³)		
		Central Tendency	High End	Central Tendency	High End	Central Tendency	High End	
Laboratory Use	Worker	6.0	100	1.4	23	2.4	52	Low
Plastic Product Manufacturing	Worker	8.5	210	1.9	47	3.4	110	Low
Plastic Product Manufacturing	ONU	9.7	10	2.2	2.3	3.9	5.3	Low
Lithographic Printing Cleaner	Worker	8.7	160	2.0	37	3.5	82	Low
Miscellaneous Non-Aerosol Industrial and Commercial Use (Cleaning Solvent)	Worker	57	930	13	210	23	480	Medium to Low
Waste Handling, Disposal, Treatment, and Recycling	Worker	2.3	81	0.54	18	0.93	41	Low

a – Where no ONU data or estimates are available, EPA assumes that ONU exposures are less than worker exposures in categories indicated as Worker.

Table 2-85. Summary of Dermal Exposure Doses to Methylene Chloride by Occupational Exposure Scenario and Potential Glove Use

Occupational Exposure Scenario	Maximum Weight Fraction, Y_{derm}	Dermal Exposure Dose (mg/day)			
		Central Tendency		High End	
		PF = 1	PF > 1	PF = 1	PF > 1
Manufacturing, Repackaging, Processing as a Reactant, Processing - Incorporation into Formulation, Mixture, or Reaction Product, Waste Handling, Disposal, Treatment, and Recycling	1	60	12 (PF = 5) 6 (PF = 10) 3 (PF = 20)	180	36 (PF = 5) 18 (PF = 10) 9 (PF = 20)
Industrial: Use of Adhesives, Use of Paints and Coatings, Flexible PU Foam Manufacturing, Batch Open-Top Vapor Degreasing, Conveyorized Vapor Degreasing, Cold Cleaning, CTA Film Production, Plastic Product Manufacturing, Miscellaneous Non-aerosol Industrial Uses	1	60	12 (PF = 5) 6 (PF = 10) 3 (PF = 20)	180	36 (PF = 5) 18 (PF = 10) 9 (PF = 20)
Commercial: Use of Adhesives, Use of Paints and Coatings, Laboratory Use, Miscellaneous Non-aerosol Commercial Uses, Commercial Aerosol Products	1	94	19 (PF = 5) 9 (PF = 10)	280	57 (PF = 5) 28 (PF = 10)
Commercial: Fabric Finishing	0.95	90	18 (PF = 5) 9 (PF = 10)	270	54 (PF = 5) 27 (PF = 10)
Commercial: Adhesive and Caulk Removers, Spot Cleaning	0.9	85	17 (PF = 5) 9 (PF = 10)	260	51 (PF = 5) 26 (PF = 10)
Commercial: Lithographic Printing Cleaner	0.885	84	17 (PF = 5) 8 (PF = 10)	250	50 (PF = 5) 25 (PF = 10)

Note on Protection Factors (PFs): All PF values are what-if type values where use of PF above 1 is recommended only for glove materials that have been tested for permeation against the methylene chloride-containing liquids associated with the condition of use. For scenarios with only industrial sites, EPA assumes that some workers wear protective gloves and have activity-specific training on the proper usage of these gloves, which assumes a PF of 20. For scenarios covering a broader variety of commercial and industrial sites, EPA assumes either the use of gloves with minimal to no employee training, which assumes a PF of 5, or the use of gloves with basic training, which assumes a PF of 10.

EPA identified primary strengths and limitations and assigned an overall confidence to the occupational dermal assessment, as discussed below. EPA considered the assessment approach, the quality of the data, and uncertainties to determine the level of confidence.

The *Dermal Exposure to Volatile Liquids Model* used for modeling occupational dermal exposures accounts for the effect of evaporation on dermal absorption for volatile chemicals and the potential exposure reduction due to glove use. The model does not account for the transient exposure and exposure duration effect, which likely overestimates exposures. The model assumes one exposure event per day, which likely underestimates exposure as workers often come into repeat contact with the chemical throughout their work day. Surface areas of skin exposure are based on skin surface area of hands from EPA's Exposure Factors Handbook, but

actual surface areas with liquid contact are unknown and uncertain for all occupational scenarios OESs. For many OESs, the assumption of contact over the full area of two hands likely overestimates exposures. Weight fractions are usually reported to CDR and shown in other literature sources as ranges, and EPA assessed only upper ends of ranges. The glove protection factors are “what-if” assumptions and are uncertain. EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces of the OESs. Except where specified above, it is unknown whether most of these uncertainties overestimate or underestimate exposures. The representativeness of the modeling results toward the true distribution of dermal doses for the OESs is uncertain. These and other limitations are more fully discussed in Section 4.4.2.4.

Considering these primary strengths and limitations, the overall confidence of the dermal dose results is medium.

2.4.2 Consumer Exposures

Methylene chloride is found in a variety of consumer products and/or commercial products that are readily available for public purchase at common retailers. These products are found across a suite of categories and uses as outlined in the Use and Market Profile for Methylene Chloride ([U.S. EPA, 2017g](#)). Based on a combination of information gained from individual products containing methylene chloride and product use scenarios, consumer exposures due to inhalation or dermal contact were modeled across a suite of identified conditions of use.

2.4.2.1 Consumer Exposures Approach and Methodology

Following problem formulation, EPA compiled a comprehensive list of current products available for consumer household use. As noted in Section 1.4.1, while the Problem Formulation included uses such as metal products not covered elsewhere, apparel and footwear care products, and laundry and dishwashing products without distinguishing between industrial, commercial, and consumer uses, after additional review, no applicable consumer products were found for these uses. EPA has determined that there is no known, intended, or reasonably foreseen consumer use of these products. There are only industrial and commercial uses of methylene chloride for these conditions of use, and these conditions of use were therefore not further assessed as consumer uses. Products were grouped into 15 subcategories ranging from 1-10 identified products in each category, but with most characterized by 4 or less (Table 2-86). Additionally, these products are primarily aerosol in nature, but are found in liquid form as well for subcategories Adhesives, Adhesives Removers, and Brush Cleaners.

Table 2-86. Evaluated Consumer Uses for Products Containing Methylene Chloride

Consumer Use Subcategory	Form	Number of Products Identified
Adhesives	Liquid	4
Adhesives Remover	Liquid	1
Auto AC Leak Sealer	Aerosol	1
Auto AC Refrigerant Fill	Aerosol	10
Brake Cleaner	Aerosol	3
Brush Cleaner	Liquid	2

Carbon Remover	Aerosol	1
Carburetor Cleaner	Aerosol	3
Coil Cleaner	Aerosol	1
Cold Pipe Insulation Spray	Aerosol	2
Electronics Cleaner	Aerosol	1
Engine Cleaner/Degreaser	Aerosol	2
Gasket Remover	Aerosol	1
Sealants	Aerosol	1
Weld Spatter/Soldering Protectant	Aerosol	1

2.4.2.2 Exposure Routes

As described in Table 2-86, exposures were evaluated for 15 conditions of use for products containing methylene chloride. For each of the listed conditions of use, inhalation and dermal exposures were evaluated, with inhalation being the primary route of exposure.

Inhalation

Consumer and bystander inhalation exposure to methylene chloride is expected to be the most significant route of exposure through the direct inhalation of sprays, vapors and mists. EPA assumed mists are absorbed via inhalation, rather than ingestion, due to the deposition of vapors and mists in the upper respiratory tract. This principal exposure pathway is in line with EPA's 2014 risk assessment of methylene chloride paint stripping use, which assumed that inhalation was the main exposure pathway based on physical-chemical properties (e.g., high vapor pressure). All fifteen identified consumer use scenarios were evaluated for exposure via the inhalation pathway to both consumer users and bystanders. The majority of these uses were evaluated as sprays or aerosol products, but several products (adhesives, adhesive removers, and brush cleaners) were evaluated as liquids that have the expectation of inhalation of vapors emitted from the product due to methylene chloride's high vapor pressure.

Dermal

Dermal exposure to consumer uses of methylene chloride was also evaluated. Dermal exposure may occur via contact with vapor or mist deposition on the skin or via direct liquid contact during use. Exposures to skin would be expected to evaporate rapidly (0.06 mol/s) based on physical chemical properties including vapor pressure, water solubility and log Kow, but some methylene chloride would also dermally absorb. When evaporation of methylene chloride is reduced or impeded (e.g., continued contact with a methylene chloride-soaked rag), dermal absorption would be higher due to the longer duration of exposure. These dermal exposures would be concurrent with inhalation exposures and the overall contribution of dermal exposure to total exposure is expected to be smaller than via inhalation. Dermal exposures were evaluated for all 15 consumer use scenarios across a range of user age groups including adults (≥ 21 years), youths aged 16-20 years and youths aged 11-15 years due to the possible consumer uses of these products by younger age groups. Bystander dermal exposure was not evaluated as the incidence of those exposures are expected to be low and not contribute significantly to overall exposure.

Ingestion

Consumers may be exposed to methylene chloride via transfer from hand to mouth, but this exposure pathway is expected to be limited due to physical chemical properties including dermal absorption and volatilization from skin. Due to the limited expected exposure to consumers via this route, EPA did not further assess this pathway.

From Disposal

EPA does not expect exposure to consumers from disposal of consumer products. It is anticipated that most products will be disposed of in original containers, particularly those products that are purchased as aerosol cans.

2.4.2.3 Modeling Approach

EPA estimated consumer exposures for all currently known, intended or reasonably foreseen use scenarios for products containing methylene chloride. A variety of sources were reviewed during the Systematic Review process to identify these products and/or articles, including:

- Safety Data Sheets (SDS)
- NIH Household Products Database
- The Chemical and Products (CPDat) Database
- Peer-reviewed and gray literature
- Kirk-Othmer Encyclopedia of Chemical Technology

Consumer exposures were assessed for all methylene chloride containing products identified, as described in Section 2.4.2.1. As no chemical-specific personal monitoring data was identified during Systematic Review, a modeling approach was used to estimate the potential consumer exposures. All consumer use scenarios were assessed using EPA's Consumer Exposure Model Version 2.1.7 (CEM), as described in Section 2.4.2.3.1, for both inhalation and dermal routes.

To characterize consumer exposures, inhalation modeling for each scenario was conducted by varying one to three key parameters, while keeping all other input parameters constant. The key varied parameters included:

- 1) duration of use per event (minutes/use);
- 2) amount of chemical in the product/article (weight fraction); and/or
- 3) mass of product/article used per event (grams/use).

Duration of use and amount of chemical used were varied to correspond to the 10th percentile, 50th percentile and 95th percentile values as reported in U.S. EPA (1987) to encompass a range of possible exposure conditions. Weight fractions were varied based on reported values of methylene chloride in Material Safety Data Sheet (MSDS) sheets for evaluated products in individual consumer use scenarios. At times, the given weight fraction was reported as a single value whereby weight fraction was not varied in the modeling framework. However, oftentimes the weight fraction for a single product was reported as a range of possible weight fractions within that product, or if multiple products were identified for a consumer use scenario, the available weight fractions making up that scenario resulted in a range. In instances, where the range in weight fractions was <40% of the product, the maximum and minimum values of the range were evaluated. In instances where the range of possible weight fractions was >40%, the minimum, maximum, and midpoint weight fractions were used to better evaluate the wider range

of possible exposure conditions. The variation of modeling inputs for the three parameters resulted in up to 27 different exposure cases per scenario.

For dermal modeling, the varying parameters were limited to duration of use and weight fraction, since mass of product is not an input for the dermal models used. Therefore, there were up to 9 different exposure cases per scenario for dermal exposure estimates. The model inputs are described in Section 2.4.2.3.1 for CEM and shown in Tables 2-87, 2-88, and 2-89.

For all product scenarios, both acute and chronic exposures were expected to occur, but only acute exposures are evaluated here. Acute exposures were defined as those occurring within a single day; whereas chronic exposures were defined as exposures comprising 10% or more of a lifetime ([EPA, 2011a](#)). The acute exposure metric selected was a 1-hr TWA.

2.4.2.3.1 CEM Model and Scenarios (e.g., table of scenarios),

Consumer exposures have been assessed using CEM for fifteen consumer use scenarios as described in Section 2.4.2.1.

CEM Version 2.1.7 ([EPA, 2017](#)) was selected for the consumer exposure modeling as the most appropriate model to estimate consumer exposures to methylene chloride, primarily due to the lack of chemical-specific emission data and other required input parameter data that are needed to run more complex indoor air models. CEM predicts indoor air concentrations from consumer product use by implementing a deterministic, mass-balance calculation utilizing an emission profile determined by implementing appropriate emission scenarios. The advantages of CEM are the following:

- CEM has been peer-reviewed.
- CEM includes several distinct models (see ([EPA, 2017](#))) appropriate for evaluating specific product and article types and use scenarios.
- CEM includes pre-populated scenarios for a variety of products and articles, which have been pre-parameterized with default use patterns, human exposure factors, environmental conditions, and product-specific properties.
- CEM has flexibility to alter default parameters, with the exception of user and bystander activity patterns.
- CEM can accommodate chemical-specific inputs.
- CEM uses the same calculation engine to compute indoor air concentrations from a source as the higher-tier Multi-Chamber Concentration and Exposure Model (MCCEM), but does not require emission rates and emission factors derived from chamber studies.

2.4.2.3.1.1 Inhalation

CEM predicts indoor air concentrations from product use by implementing a deterministic, mass-balance calculation selected by the user depending on the relevant submodel (E1 through E5; see ([EPA, 2017](#))). The model uses a two-zone representation of the building of use, with Zone 1 representing the room where the consumer product is used and Zone 2 being the remainder of the building. The product user is placed within Zone 1 for the hour(s) encompassing the duration of use, while the bystander population remained in Zone 2 during this time period. A bystander

entering the room of use during the period of product use was not modeled since the inhalable air concentrations they would be exposed to would be similar to the evaluated user scenario. Following the time period of product use, product users and bystanders follow prescribed activity patterns and inhale airborne concentrations of those zones.

The general steps of the calculation engine within CEM include:

1. Introduction of the chemical (i.e., methylene chloride) into the room of use (Zone 1),
2. Transfer of the chemical to the rest of the house (Zone 2) due to exchange of air between the different rooms,
3. Exchange of the house air with outdoor air and,
4. Summation of the exposure doses as the modeled occupant moves about the house.

EPA applied the default activity pattern in CEM based on the occupant being present in the home for most of the day. As the occupants move between zones in the model, the associated zonal air concentrations at each 30-second time step were compiled to reflect the air concentrations a user and bystanders would be exposed to throughout the simulation period. Depending on the modeled room of use, it is possible that a user or bystander may enter into that room following the product use period according to the prescribed activity pattern. For the E1 and E3 submodels, the near-field option that captures the higher concentration in the breathing zone of the product user during use was selected. TWAs were then computed based on these user and bystander concentration time series per available human health hazard data. For methylene chloride, 1-hr and 8-hr TWAs were calculated for use in this risk evaluation (see Section 2.4.2.4 “Consumer Use Scenario Specific Results”).

The emissions models used for evaluating methylene airborne concentrations were either the E1, E2, or E3 emissions model depending on the given consumer use scenario (see Table 2-88). The E1 model estimates emission and inhalation exposures from a product applied to an indoor surface (incremental source model) and is mostly applicable to liquid products that are applied to a surface and evaporate from that surface (e.g., a cleaner). The E2 model estimates emission and inhalation exposures from a product applied to an indoor surface (double exponential model) and is applicable to liquid products that are applied to a surface and dry or cure over time (e.g., paints). Finally, the E3 model estimates emission and exposure from a sprayed product. For specifics on the varied emission models utilized, their assumptions, and underlying algorithms, EPA refers you to the user’s guide for CEM ([EPA, 2017](#)).

2.4.2.3.1.2 Dermal

For methylene chloride, dermal exposures to products directly contacting skin were evaluated using either the fraction absorbed submodel (P_DER2a) or the permeability submodel (P_DER2b) within CEM. The selection of the appropriate submodel was based on whether the evaluated condition of use was expected to involve dermal contact with impeded or unimpeded evaporation.

For situations where dermal contact with impeded evaporation was possible (e.g., wiping with a chemical soaked rag or immersion of dermal surface into the chemical product), the permeability submodel was utilized. P_DER2b estimates dermal flux based on a permeability coefficient (K_p) and is based on the ability of a chemical to penetrate the skin layer once contact occurs. It assumes a constant supply of chemical directly in contact with the skin throughout the exposure

duration. Note the permeability model does not inherently account for evaporative losses (unless the available flux or K_p values are based on non-occluded, evaporative conditions), which can be considerable for volatile chemicals in scenarios where evaporation is not impeded. For methylene chloride, a measured neat dermal permeability coefficient ($K_p = 8.66E-03$ cm/hr) is applied based on Schenk et al. (2018). While the permeability model does not explicitly represent exposures involving such impeded evaporation, the model assumptions make it the preferred model for an such a scenario. For complete description of this submodel, see the CEM User's Guide (EPA, 2017).

In contrast, in situations where dermal contact would be expected to result in unimpeded evaporation, the fraction absorbed submodel (P_DER2a) was utilized. Within this model, the potential dose is the amount of the chemical contained in bulk material that is applied to the skin and the absorbed dose is the amount of the substance that penetrates across the dermal barrier. The model is essentially the measure of two competing processes, evaporation of the chemical from the skin surface and penetration deeper into the skin. The fraction absorbed is estimated for methylene chloride based on Frasch and Bunge (2015) and described in full within the CEM User's Guide (EPA, 2017). This model assumes the skin surface layer is "filled" once during product use to an input thickness with subsequent absorption over an estimated absorption time. Due to the submodel's ability to incorporate evaporative processes, it was considered to be more representative of dermal exposure under unimpeded situations.

As first outlined in Section 2.4.1.1, it is important to note that while occupational and certain consumer dermal exposure assessments have a common underlying methodology using dermal fractional absorption, they use different parametric approaches for dermal exposures due to different data availability and assessment needs. For example, the occupational approach accounts for glove use using protection factors, while the consumer approach does not consider glove use since consumers are not expected to always use gloves constructed with appropriate materials. The consumer approach factors in duration of use because consumer activities as a function of product duration of use are much better defined and characterized, while duration of dermal exposure times for different occupational activities across various workplaces are often not known. Additionally, the consumer dermal exposure assessments include scenario specific inputs for fractional surface area of the body exposed in certain consumer activities and offers different default values for film thickness (ranging from $1.88E-03$ to 0.01 cm), and skin surface area (ranging from 10% of both hands to inside of both hands) for different product users across different life stages (youth to adult) (Table 2-88 and Section 2.4.2.3.2). While these approaches both represent fractional absorption methodologies, the different models may result in different exposure values for similar conditions of use.

2.4.2.3.2 CEM Scenario Inputs

The complete CEM model inputs are provided in *Supplemental Information on Consumer Exposure Assessment*. A discussion of the key inputs is provided below. The inputs are categorized into three types: 1) parameters which are the same among all scenarios (Table 2-87); 2) Scenario-specific parameters which were not varied (Table 2-88); and 3) Scenario-specific

scenarios which were varied to obtain the range of exposure estimates (Table 2-89). A discussion of key inputs is provided below.

2.4.2.3.2.1 Fixed Scenario Inputs

Parameters used that were the same across all consumer use modeling scenarios parameters are shown in Table 2-87 and described briefly below. They include populations modeled for both inhalation and dermal exposure, receptor exposure factors and product properties, activity patterns, and environmental inputs.

Population

For all methylene chloride scenarios, the consumer user was assumed to be an adult (age 21+) and two youth age groups (16-20 years and 11-15 years), while a non-user bystander can include individuals of any age. Results are presented for users and non-user bystanders for inhalation exposures and users only for dermal exposures. Inhalation exposure results are presented as concentrations encountered by users and non-user bystanders and are independent of age group. EPA presents all three evaluated user age groups for dermal exposures as reported doses are age group specific. More information about how generated exposure estimates are used to evaluate consumer risk for specific age groups can be found in Section 4.2

Receptor Exposure Factors and Product Properties

Default receptor exposure factors in CEM, as determined from the Exposure Factors Handbook ([EPA, 2011a](#)) were used for body weight and inhalation rate during and after use. Aerosol fraction was set at the CEM default of 0.06. Exposure duration remained a value of 1 for acute exposures. For calculation of dermal exposure, the skin permeability coefficient was based on a neat value of 8.66E-03 ([Schenk et al., 2018](#)).

Activity Patterns and Product Use Start Time

The activity pattern selected for the user (i.e., room/building location throughout the exposure period on an hourly basis) was the default “stay-at-home” resident which places the user primarily in the home during and after use of the product. The activity patterns were developed based on Consolidated Human Activity Database (CHAD) ([Isaacs, 2014](#)) data of activity patterns.

The use environment (room of product use) was the default in CEM for pre-populated scenarios, unless professional judgement was used based on review of specific product information and/or consumer behavior pattern data in the U.S. EPA ([1987](#)) survey of product users for various consumer product categories. In all cases, the product use was assumed to start at 9:00 AM in the morning.

Environmental Inputs

All environmental inputs (building volume, air exchange, interzonal air flow) were based on a residence environment and used CEM default values obtained from Exposure Factors Handbook ([EPA, 2011a](#)). Building volume (492 m³) is used to calculate air concentrations in Zone 2 and room volume is used to calculate air concentrations in Zone 1 (see below). The volume of the near-field bubble in Zone 1 was assumed to be 1 m³ in all cases, with the remaining as the far-field volume. The default interzonal air flows are a function of the overall air exchange rate and volume of the building, as well as the “openness” of the room itself. Kitchens, living rooms,

garages, schools, and offices are considered to be more open to the rest of the home or building of use; bedrooms, bathrooms, laundry rooms, and utility rooms are usually accessed through one door and are considered more closed. Background concentration was set to a CEM default value of 0 mg/m³.

Table 2-87. Fixed Consumer Use Scenario Modeling Parameters

Parameter	Units	Value / Description
MODEL SELECTION / SCENARIO INPUTS		
Pathways Selected	n/a	Inhalation and Dermal
Inhalation Model	n/a	Inhalation of Product Used in Environment (Near-Field / Far-Field) (P_INH2)
Emission Rate	n/a	Let CEM Estimate Emission Rate
Product User (s)	n/a	Adult (≥21 years) and Youth (Age 11-20 years)
Activity Pattern	n/a	User Stays at home entire day
Product Use Start Time	n/a	9:00 AM
Background Concentration	mg/m ³	0
PRODUCT/ARTICLE PROPERTIES		
Frequency of Use (Acute)	events/day	Fixed at 1 event/day (CEM default)
Aerosol Fraction	-	CEM default (0.06)
Fraction Product Ingested	n/a	0
Skin Permeability Coefficient	cm/hr	8.66E-03 (Schenk et al., 2018)
Product Dilution Factor	unitless	Fixed at 1 (i.e., no dilution)
ENVIRONMENT INPUTS		
Building Volume (Residence)	m ³	492
Air Exchange Rate, Zone 1 (Residence)	hr ⁻¹	CEM default (0.45)
Air Exchange Rate, Zone 2 (Residence)	hr ⁻¹	CEM default (0.45)
Air Exchange Rate, Near-Field Boundary	hr ⁻¹	CEM default (402)

2.4.2.3.2.2. Non-varying Scenario Specific Inputs

Consumer use non-varying scenario specific inputs for evaluation of inhalation and dermal exposure are shown in Table 2-88 and described in more detail below.

Product Density

Product density was derived for each consumer use scenario from individual product derived information found on company websites and/or available SDSs. As multiple products with

varying densities may be found within the same use scenario, the highest reported density was used in the CEM modeling.

Dermal Exposure Inputs

For the evaluation of dermal exposures from the use of methylene chloride, multiple scenario specific inputs were used. Surface area to body weight ratio inputs were based on whether the evaluated COU was run with the CEM Absorption or CEM Permeability submodel. For those condition of use scenarios run with the CEM Absorption submodel (P_DER2a) a 10% of both hands SA/BW ratio was selected since product contact with dermal surfaces would likely be limited. For those scenarios run with the CEM Permeability submodel (P_DER2b) an inside of one hand or both hands SA/BW ratio was selected based on whether the evaluated COU was expected to have a situation where product use would involve wiping (e.g., a methylene chloride soaked rag) or full immersion of both hands respectively (e.g., cleaning a brush). Film thickness was input based on CEM scenario specific default inputs or set to a default value of 0.01 cm. Amount of chemical retained on skin is a calculated parameter dependent on film thickness and methylene chloride density for the given use scenario. Absorption fraction was input based on neat value given in Schenk et al. ([2018](#))

Room of use

The input room of use is based on information derived from U.S. EPA ([1987](#)) for developed use scenarios, CEM scenario default inputs, or information on chemical use from product labeling or company websites.

2.4.2.3.2.3. Scenario specific varied inputs

Consumer use non-varying scenario specific inputs for evaluation of inhalation and dermal exposure are shown in Table 2-89 and described in more detail below.

Duration of Use

The amount of time that a product is used per event was based on the U.S. EPA ([1987](#)) survey of consumer behavior patterns. The most representative product use category in the survey was selected for each scenario assessed. This input parameter was varied using the 10th, 50th, and 95th values.

Product Weight Fractions

Product weight fractions were determined from review of product SDSs and any other information identified during Systematic Review. This input parameter was varied using the 10th, 50th, and 95th values, unless only single products were identified. Different weight fractions could potentially make a product more or less efficient in time used or amount used however, EPA is not able to quantify that change.

Mass of Product Used

The amount of product used per event was based on the U.S. EPA ([1987](#)) survey of consumer behavior patterns. The most representative product use category in the survey was selected for each scenario assessed. This input parameter was varied using the 10th, 50th, and 95th values.

Table 2-88. Consumer Use Non-Varying Scenario Specific Inputs for Evaluation of Inhalation and Dermal Exposure

Consumer Conditions of Use	Form (# of Prod.) ¹	Selected CEM 2.1.6 Modeling Scenario ²	Product Density (g/cm ³) ³	Emission Model Applied ⁴	Dermal Exposure Model Applied ⁵	Dermal SA/BW ⁶	Dermal Film Thickness (cm)	Amount Retained on Skin (g/cm ²) ⁷	Absorption Fraction ⁸	Room of Use (m ³) ⁹
Adhesives	Liquid (4)	Glue and Adhesives (small scale)	1.375	E1	P_DER2a	10% of hand surface area	4.99E-03	0.012	0.017	Utility Room (20)
Adhesives Remover	Liquid (1)	Adhesive/Caulk Removers, 12 years	1.114	E2	P_DER2b	Inside of one hand	0.01	0.011	0.089	Utility Room (20)
Automotive AC Leak Sealer	Aerosol (1)	Generic Product	0.994	E3	P_DER2a	10% of hand surface area	0.01	0.010	0.134	Garage (90)
Automotive AC Refrigerant	Aerosol (10)	Generic Product	1.208	E3	P_DER2a	10% of hand surface area	0.01	0.012	0.134	Garage (90)
Brake Cleaner	Aerosol (3)	Degreasers	1.5322	E3	P_DER2b	Inside of one hand	0.01	0.007	0.033	Garage (90)
Brush Cleaner	Liquid (2)	Paint Strippers/Removers	0.9032	E2	P_DER2b	Inside of both hands	1.88E-03	0.011	0.134	Utility Room (20)
Carbon Remover	Aerosol (1)	Degreasers	1.17	E3	P_DER2b	Inside of one hand	0.01	0.012	0.062	Kitchen (24)
Carburetor Cleaner	Aerosol (3)	Degreasers	1.13	E3	P_DER2b	Inside of one hand	0.01	0.015	0.033	Garage (90)
Coil Cleaner	Aerosol (1)	Generic Product	1.34	E3	P_DER2b	Inside of one hand	0.01	0.013	0.062	Kitchen (24)
Cold Pipe Insulating Spray	Aerosol (2)	Generic Product	1.2	E3	P_DER2a	10% of hand surface area	0.01	0.002	0.017	Kitchen (24)
Electronics Cleaner	Aerosol (1)	Degreasers	1.27	E3	P_DER2a	10% of hand surface area	0.01	0.013	0.017	Living Room (50)

Consumer Conditions of Use	Form (# of Prod.) ¹	Selected CEM 2.1.6 Modeling Scenario ²	Product Density (g/cm ³) ³	Emission Model Applied ⁴	Dermal Exposure Model Applied ⁵	Dermal SA/BW ⁶	Dermal Film Thickness (cm)	Amount Retained on Skin (g/cm ²) ⁷	Absorption Fraction ⁸	Room of Use (m ³) ⁹
Engine Cleaner	Aerosol (2)	Degreasers	1.13	E3	P_DER2b	Inside of one hand	0.01	0.012	0.134	Garage (90)
Gasket Remover	Aerosol (1)	Degreasers	1.038	E3	P_DER2b	Inside of one hand	0.01	0.010	0.062	Garage (90)
Sealant	Aerosol (1)	Generic Product	1.05	E3	P_DER2a	10% of hand surface area	0.01	0.001	0.062	Garage (90)
Weld Spatter Protectant	Aerosol (1)	Generic Product	1.31	E3	P_DER2a	10% of hand surface area	0.01	0.009	0.017	Utility Room

1 Number of products identified for a condition of use scenario is based on product lists within EPA's 2017 Market and use Report.

2 The listed CEM 2.1.6 modeling scenario reflects the default product options within the model, which are prepopulated with certain default parameters. However, due to EPA choosing to select and vary many key inputs, the specific model scenario matters less than the associated emission and dermal exposure models (e.g., E1, E3, P_DER2a).

3 Selected product densities were primarily sourced from product SDSs and MSDSs unless otherwise noted. Where a range of densities was identified for a given condition of use, the highest reported product density was used.

4 Selected emissions model used is based on CEM scenario used or best professional judgement.

5 Selected dermal model is based on selection of absorption model for dermal exposure evaluation.

6 Selected dermal surface area to body weight (SA/BW) ratio used is based on CEM scenario used or best professional judgement for Generic Scenario.

7 The amount retained on the skin is an estimated parameter within CEM based on film thickness and chemical density.

8 Absorption fraction is an estimated parameter with CEM with values varying based on exposure time. Values shown here represent values derived from 10th percentile time used scenarios. Values would differ for 50th and 95th percentile time of use (see Table 2-91).

9 Room of use is either default scenario option within CEM, based on survey results from U.S. EPA (1987), or derived from product use information on product labels or websites.

Table 2-89. Consumer Use Scenario Specific Values of Duration of Use, Weight Fraction, and Mass of Product Used Derived from W.U.S. EPA (1987)

Consumer Conditions of Use	Form	Selected U.S. EPA (1987) Survey Scenario ¹	Duration of Use (min)			Weight Fraction (% methylene chloride) ³			Mass of Product Used (g, [oz]) ⁴		
			U.S. EPA (1987) Scenario Percentile			Min	Mid	Max	U.S. EPA (1987) Scenario Percentile		
			10% ²	50%	95%				10%	50%	95%
Adhesives	Liquid	Contact Cement, Super Glues, and Spray Adhesives	0.33	4.25	60	30	60	90	1.22 [0.03]	10.16 [0.25]	175.65 [4.32]
Adhesives Remover	Liquid	Adhesive Removers	3	60	480	50		75	22.07 [0.67]	263.53 [8]	2108.22 [64]
Automotive AC Leak Sealer	Aerosol	Engine Cleaners/Degreasers	5	15	120	1				88.18 [3]	
Automotive AC Refrigerant	Aerosol	Engine Cleaners/Degreasers	5	15	120	1		3	103.95 [2.91]	414.36 [11.6]	1714.59 [48]
Brake Cleaner	Aerosol	Brake Quieters/Cleaners	1	15	120	10	35	60	45.31 [1 oz]	181.23 [4]	724.91 [16]
Brush Cleaner	Liquid	Paint Removers/Strippers	5	60	420	1			71.31 [2.67]	427.32 [16]	3418.58 [128]
Carbon Remover	Aerosol	Solvent-type Cleaning Fluids or Degreasers	2	15	120	40		70	19.37 [0.56]	112.44 [3.25]	1107.10 [32]
Carburetor Cleaner	Aerosol	Carburetor Cleaner	1	7	45	20	45	70	41.77 [1.25]	167.07 [5]	644.89 [19.3]
Coil Cleaner	Aerosol	Solvent-type Cleaning Fluids or Degreasers	2	15	120	60		100	22.19 [0.56]	128.78 [3.25]	1267.96 [32]
Cold Pipe Insulating Spray	Aerosol	Rust Removers	0.25	5	60	30		60	15.97 [0.45]	77.00 [2.17]	521.61 [14.70]
Electronics Cleaner	Aerosol	Specialized Electronic Cleaners	0.17	2	30	5			1.50 [0.04]	18.78 [0.50]	281.65 [7.50]
Engine Cleaner	Aerosol	Engine Cleaners/Degreasers	5	15	120	20	45	70	97.24 [2.91]	387.60 [11.60]	1603.88 [48]
Gasket Remover	Aerosol	Gasket Remover	2	15	60	60		80	29.77 [0.97]	122.77 [4]	790.05 [25.74]

Consumer Conditions of Use	Form	Selected U.S. EPA (1987) Survey Scenario ¹	Duration of Use (min)			Weight Fraction (% methylene chloride) ³			Mass of Product Used (g, [oz]) ⁴		
			U.S. EPA (1987) Scenario Percentile			Min	Mid	Max	U.S. EPA (1987) Scenario Percentile		
			10% ²	50%	95%				10%	50%	95%
Sealant	Aerosol	Gasket Remover	2	15	60	10		30	30.12 [0.97]	124.19 [4]	799.19 [25.74]
Weld Spatter Protectant	Aerosol	Rust Removers	0.25	5	60	90			17.43 [0.45]	84.06 [2.17]	569.43 [14.70]

1 U.S. EPA (1987) was used to inform values used for duration of use and mass of product used. Where exact matches for conditions of use were not available, scenario selection was based on product categories that best met the description and usage patterns of the identified consumer conditions of use.

2 Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

3 The range in weight fractions is reflective of the identified products containing methylene chloride and not reflective of hypothetical functionality-based limits. Weight Fractions were primarily sourced from product SDSs and MSDSs unless otherwise noted. For information selection of weight fraction values, see Section 2.4.2.3.2.3.

4 Mass of product used within U.S. EPA (1987) for given scenarios is reported in ounces, but was converted to grams for use within CEM. Conversion to grams involved using reported density in SDSs and MSDSs for products within a condition of use. Therefore, mass of product used may vary for conditions of use where the same Westat (1987) scenario was used. See Table 2-90 for selected product densities.

2.4.2.3.3 Sensitivity Analysis

The CEM developers conducted a detailed sensitivity analysis for CEM version 1.5. A discussion of that sensitivity analysis is presented in Appendix G and is described in full within Appendix C of the CEM User Guide (EPA, 2017). In brief, the analysis was conducted on non-linear, continuous variables and categorical variables that were used in CEM models. A base run of different models using various product or article categories along with CEM defaults was used (see Table 1 of Appendix C in U.S. EPA (2017)). Individual variables were modified, one at a time, and the resulting Chronic Average Daily Dose (CADD) and Acute Dose Rate (ADR) were then compared to the corresponding results for the base run.

2.4.2.4 Consumer Use Scenario Specific Results

Consumer use scenarios for 15 different conditions of use for both possible inhalation and dermal exposures were evaluated across a range of user intensities based on differences in duration of use, weight fraction and mass of product used. While up to 27 different scenarios were evaluated for inhalation and 18 scenarios for dermal exposure, for the purposes of presenting the inhalation and dermal results, three combinations are presented to provide results across a range of use patterns modeled. EPA uses the following descriptors for these three use patterns: high intensity, moderate intensity, and low intensity use. These descriptors are based on three key input parameters varied during the modeling (duration of use, weight fraction, and mass of product used) which are summarized in Section 2.4.2.4.2.3 and Table 2-89 but included here for ease of reference.

For inhalation results, high intensity use refers to the model iteration that utilized the 95th percentile duration of use and mass of product used (as presented in U.S. EPA (1987)) and the maximum weight fraction derived from product specific SDS, when available. Moderate intensity use refers to the model iteration that utilized the median (50th percentile) duration of use and mass of product used (as presented U.S. EPA (1987)) and the midpoint weight fraction derived from product specific SDS, when available. In instances where only two weight fractions were modeled, the maximum weight fraction was used to represent the moderate intensity user. Low intensity use refers to the model iteration that utilized the 10th percentile duration of use and mass of product used (as presented in U.S. EPA (1987)) and the minimum weight fraction derived from product specific SDS, when available. For dermal results, only the duration of use and weight fraction inputs were varied across scenarios. Characterization of high intensity, moderate intensity uses and low intensity users following the same protocol as those described for the inhalation results, but only encompassing the two varied parameters. For certain situations, only a single value was identified for weight fraction in the product specific SDS. For those situations, that parameter is labeled single value and the same value is used in all three use patterns in the summary tables below.

2.4.2.4.1 Adhesives

Four consumer products used as an adhesive were found to contain methylene chloride in weight fractions between 30% - 90% (Table 2-90). Inhalation exposures were evaluated for users and bystanders for 27 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 4.2 – 1,576 mg/m³ for

users and from 0.38 – 200 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for nine scenarios using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 4.0E-02 – 2.5 mg/kg/day across all evaluated scenarios and age groups (Table 2-91).

Table 2-90. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as an Adhesive

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (60)	Max (90)	95% (175.65)	User	1,576	258
				Bystander	200	61
Moderate Intensity User	50% (4.25)	Midpoint (60)	50% (10.16)	User	71	10.9
				Bystander	6.5	1.9
Low Intensity User	10% (0.33) ¹	Min (30)	10% (1.22)	User	4.2	0.64
				Bystander	0.38	0.11

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

Table 2-91. Consumer Dermal Exposure to Methylene Chloride During Use as an Adhesive

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (60)	Max (90)	Adult (≥21 years)	2.5
			Youth (16-20 years)	2.4
			Youth (11-15 years)	2.6
Moderate Intensity User	50% (4.25)	Midpoint (60)	Adult (≥21 years)	0.60
			Youth (16-20 years)	0.56
			Youth (11-15 years)	0.62
Low Intensity User	10% (0.33) ¹	Min (30)	Adult (≥21 years)	4.3E-02
			Youth (16-20 years)	4.0E-02
			Youth (11-15 years)	4.4E-02

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

2.4.2.4.2 Adhesive Remover

A consumer product used as an adhesive remover were found to contain methylene chloride in weight fractions between 50% - 75% (Table 2-92). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 1.3 – 74 mg/m³ for users and from 0.29 – 20 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 0.70 – 183 mg/kg/day across all evaluated scenarios and age groups (Table 2-93).

Table 2-92. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as an Adhesives Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (480)	Max (75)	95% (2108.22)	User	74	68
				Bystander	62	18
Moderate Intensity User	50% (60)	Max (75)	50% (265.53)	User	49	8.1
				Bystander	6.3	1.9
Low Intensity User	10% (3)	Min (50)	10% (22.07)	User	3.3	0.50
				Bystander	0.29	8.9E-02

Table 2-93. Consumer Dermal Exposure to Methylene Chloride During Use as an Adhesive Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (480)	Max (75)	Adult (≥21 years)	179
			Youth (16-20 years)	168
			Youth (11-15 years)	183
Moderate Intensity User	50% (60)	Max (75)	Adult (≥21 years)	22
			Youth (16-20 years)	21
			Youth (11-15 years)	23
Low Intensity User	10% (3)	Min (50)	Adult (≥21 years)	0.75
			Youth (16-20 years)	0.70
			Youth (11-15 years)	0.76

2.4.2.4.3 Auto AC Leak Sealer

An automotive AC leak sealant containing methylene chloride was identified as available for consumer use with a weight fraction of <1% (Table 2-94). Inhalation exposures were evaluated for users and bystanders for three different scenarios of duration of use, weight fraction and mass of use. One-hour maximum TWA concentrations ranged from 4.0 – 7.0 mg/m³ for users and from 0.75 – 0.83 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for three scenarios using the CEM Fraction Absorbed submodel and ranged from 1.5E-02 – 4.2E-02 mg/kg/day across all evaluated scenarios and age groups (Table 2-95).

Table 2-94. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Auto Leak Sealer Use

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Single Value (1)	Single Value (88.18)	User	4.0	1.1
				Bystander	0.75	0.30
Moderate Intensity User	50% (15)	Single Value (1)	Single Value (88.18)	User	6.8	1.1
				Bystander	0.83	0.27
Low Intensity User	10% (5)	Single Value (1)	Single Value (88.18)	User	7.0	1.1
				Bystander	0.82	0.26

Table 2-95. Consumer Dermal Exposure to Methylene Chloride During Use as an Auto Leak Sealer

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Single Value (1)	Adult (≥21 years)	4.1E-02
			Youth (16-20 years)	3.8E-02
			Youth (11-15 years)	4.2E-02
Moderate Intensity User	50% (15)	Single Value (1)	Adult (≥21 years)	3.2E-02
			Youth (16-20 years)	3.0E-02
			Youth (11-15 years)	3.3E-02
Low Intensity User	10% (5)	Single Value (1)	Adult (≥21 years)	1.7 E-02
			Youth (16-20 years)	1.5 E-02
			Youth (11-15 years)	1.7 E-02

2.4.2.4.4 Auto AC Refrigerant

Ten consumer products used as an automotive AC refrigerant were found to contain methylene chloride in weight fractions of <1% - 3% (Table 2-96). Inhalation exposures were evaluated for

users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 8.3 – 233 mg/m³ for users and from 0.96 – 44 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 1.9E-02 – 0.15 mg/kg/day across all evaluated scenarios and age groups (Table 2-97).

Table 2-96. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Auto Air Conditioning Refrigerant Use

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Max (3)	95% (1714.59)	User	233	62
				Bystander	44	17
Moderate Intensity User	50% (15)	Max (3)	50% (414.36)	User	96	16
				Bystander	12	3.8
Low Intensity User	10% (5)	Min (1)	10% (103.95)	User	8.3	1.3
				Bystander	0.96	0.31

Table 2-97. Consumer Dermal Exposure to Methylene Chloride During Use as an Auto Air Conditioning Refrigerant

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Max (3)	Adult (≥21 years)	0.15
			Youth (16-20 years)	0.14
			Youth (11-15 years)	0.15
Moderate Intensity User	50% (15)	Max (3)	Adult (≥21 years)	0.12
			Youth (16-20 years)	0.11
			Youth (11-15 years)	0.12
Low Intensity User	10% (5)	Min (1)	Adult (≥21 years)	2.0E-02
			Youth (16-20 years)	1.9E-02
			Youth (11-15 years)	2.1E-02

2.4.2.4.5 Brake Cleaner

Three products used as a brake cleaner were found to contain methylene chloride in weight fractions between 10% - 60% (Table 2-98). Inhalation exposures were evaluated for users and bystanders for 27 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity

user scenarios, with 1-hr maximum TWA concentrations ranging from 36 – 1,974 mg/m³ for users and from 4.2 – 371 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for nine scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 6.4E-02 – 50 mg/kg/day across all evaluated scenarios and age groups (Table 2-99).

Table 2-98. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Brake Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Max (60)	95% (724.91)	User	1,974	522
				Bystander	371	146
Moderate Intensity User	50% (15)	Midpoint (35)	50% (181.23)	User	490	81
				Bystander	60	19
Low Intensity User	10% (1)	Min (10)	10% (45.31)	User	36	5.8
				Bystander	4.2	1.3

Table 2-99. Consumer Dermal Exposure to Methylene Chloride During Use as a Brake Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Max (65)	Adult (≥21 years)	49
			Youth (16-20 years)	46
			Youth (11-15 years)	50
Moderate Intensity User	50% (15)	Medium (35)	Adult (≥21 years)	3.6
			Youth (16-20 years)	3.4
			Youth (11-15 years)	3.7
Low Intensity User	10% (1)	Low (10)	Adult (≥21 years)	6.8E-02
			Youth (16-20 years)	6.4E-02
			Youth (11-15 years)	7.0E-02

2.4.2.4.6 Brush Cleaner

Two products used as a brush cleaner were found to contain methylene chloride in weight fractions <1% (Table 2-100). Inhalation exposures were evaluated for users and bystanders for nine different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 0.21 – 1.8 mg/m³ for users and from 1.9E-02 – 0.65 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for three scenarios using the CEM Permeability submodel. Selected scenarios representing low

intensity user, moderate intensity user and high intensity user scenarios ranged from 0.04 – 3.5 mg/kg/day across all evaluated scenarios and age groups (Table 2-101).

Table 2-100. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Brush Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (420)	Single Value (1)	95% (3418.58)	User	1.8	1.52
				Bystander	0.65	0.32
Moderate Intensity User	50% (60)	Single Value (1)	50% (427.32)	User	1.1	0.18
				Bystander	0.14	4.2E-02
Low Intensity User	10% (5)	Single Value (1)	10% (71.31)	User	0.21	3.2E-02
				Bystander	1.9E-02	5.8E-03

Table 2-101. Consumer Dermal Exposure to Methylene Chloride During Use as a Brush Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (420)	Single Value (1)	Adult (≥21 years)	3.4
			Youth (16-20 years)	3.2
			Youth (11-15 years)	3.5
Moderate Intensity User	50% (60)	Single Value (1)	Adult (≥21 years)	0.48
			Youth (16-20 years)	0.45
			Youth (11-15 years)	0.50
Low Intensity User	10% (5)	Single Value (1)	Adult (≥21 years)	0.04
			Youth (16-20 years)	0.04
			Youth (11-15 years)	0.04

2.4.2.4.7 Carbon Remover

One product used as a carbon remover (e.g., to clean appliances, pots and pans, etc.) was found to contain methylene chloride in weight fractions between 40-70% (Table 2-102). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 89– 4,751 mg/m³ for users and from 8.2 – 847 mg/m³ for bystanders across

scenarios. Dermal exposures were evaluated for six scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 0.39 – 45 mg/kg/day across all evaluated scenarios and age groups (Table 2-103).

Table 2-102. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Carbon Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Max (70)	95% (1107.10)	User	4,751	1,276
				Bystander	847	311
Moderate Intensity User	50% (15)	Max (70)	50% (112.44)	User	896	138
				Bystander	87	26
Low Intensity User	10% (2)	Min (40)	10% (19.37)	User	89	14
				Bystander	8.2	2.4

Table 2-103. Consumer Dermal Exposure to Methylene Chloride During Use as a Carbon Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Max (70)	Adult (≥21 years)	44
			Youth (16-20 years)	41
			Youth (11-15 years)	45
Moderate Intensity User	50% (15)	Max (70)	Adult (≥21 years)	5.5
			Youth (16-20 years)	5.1
			Youth (11-15 years)	5.6
Low Intensity User	10% (2)	Min (40)	Adult (≥21 years)	0.42
			Youth (16-20 years)	0.39
			Youth (11-15 years)	0.43

2.4.2.4.8 Carburetor Cleaner

Three products used as a carburetor cleaner were found to contain methylene chloride in weight fractions between 20-70% (Table 2-104). Inhalation exposures were evaluated for users and bystanders for 27 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 66 – 3,021 mg/m³ for users and from 7.7 – 428 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for nine scenarios using the CEM Permeability submodel. Selected scenarios

representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 9.5E-02 – 16 mg/kg/day across all evaluated scenarios and age groups (Table 2-105).

Table 2-104. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Carburetor Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (45)	Max (70)	95% (644.89)	User	3,021	525
				Bystander	428	148
Moderate Intensity User	50% (7)	Midpoint (45)	50% (167.07)	User	595	97
				Bystander	70	22
Low Intensity User	10% (1)	Min (20)	10% (41.77)	User	66	11
				Bystander	7.7	2.5

Table 2-105. Consumer Dermal Exposure to Methylene Chloride During Use as a Carburetor Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (45)	Max (70)	Adult (≥21 years)	16
			Youth (16-20 years)	15
			Youth (11-15 years)	16
Moderate Intensity User	50% (7)	Midpoint (45)	Adult (≥21 years)	1.6
			Youth (16-20 years)	1.5
			Youth (11-15 years)	1.6
Low Intensity User	10% (1)	Min (20)	Adult (≥21 years)	0.10
			Youth (16-20 years)	9.5E-02
			Youth (11-15 years)	0.10

2.4.2.4.9 Coil Cleaner

One product used as a coil cleaner (e.g., air conditioner condensing coils) was found to contain methylene chloride in weight fractions between 60-100% (Table 2-106). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 152 – 7,773 mg/m³ for users and from 14 – 1,387 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 0.67 – 74 mg/kg/day across all evaluated scenarios and age groups (Table 2-107).

Table 2-106. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During use as a Coil Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Max (100)	95% (1267.96)	User	7,773	2,088
				Bystander	1,387	509
Moderate Intensity User	50% (15)	Max (100)	50% (128.78)	User	1,465	225
				Bystander	142	42
Low Intensity User	10% (2)	Min (60)	10% (22.19)	User	152	23
				Bystander	14	4.2

Table 2-107. Consumer Dermal Exposure to Methylene Chloride During Use as a Coil Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Max (100)	Adult (≥21 years)	72
			Youth (16-20 years)	67
			Youth (11-15 years)	74
Moderate Intensity User	50% (15)	Max (100)	Adult (≥21 years)	9.0
			Youth (16-20 years)	8.4
			Youth (11-15 years)	9.2
Low Intensity User	10% (2)	Min (60)	Adult (≥21 years)	0.72
			Youth (16-20 years)	0.67
			Youth (11-15 years)	0.74

2.4.2.4.10 Cold Pipe Insulation Spray

Two products used as a cold pipe insulation spray were found to contain methylene chloride in weight fractions between 30-60% (Table 2-108). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 54 – 2,965 mg/m³ for users and from 5.0 – 390 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 7.0E-02 – 3.04 mg/kg/day across all evaluated scenarios and age groups (Table 2-109).

Table 2-108. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Cold Pipe Insulation Spray Use

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (60)	Max (60)	95% (521.61)	User	2,965	491
				Bystander	390	120
Moderate Intensity User	50% (5)	Max (60)	50% (77.00)	User	530	81
				Bystander	49	15
Low Intensity User	10% (0.25) ¹	Min (30)	10% (15.97)	User	54	8.2
				Bystander	5.0	1.5

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

Table 2-109. Consumer Dermal Exposure to Methylene Chloride During Use as a Cold Pipe Insulation Spray

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (60)	Max (60)	Adult (≥21 years)	2.97
			Youth (16-20 years)	2.78
			Youth (11-15 years)	3.04
Moderate Intensity User	50% (5)	Max (60)	Adult (≥21 years)	1.20
			Youth (16-20 years)	1.12
			Youth (11-15 years)	1.22
Low Intensity User	10% (0.25) ¹	Min (30)	Adult (≥21 years)	7.5E-02
			Youth (16-20 years)	7.0E-02
			Youth (11-15 years)	7.7E-02

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

2.4.2.4.11 Electronics Cleaner

One product used as an electronics cleaner was found to contain methylene chloride with a weight fraction of 5% (Table 2-110). Inhalation exposures were evaluated for users and bystanders for 9 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 0.72 – 130 mg/m³ for users and from 0.11 – 27 mg/m³ for bystanders across scenarios. Dermal exposures were

evaluated for three scenarios using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 1.2E-02 – 0.26 mg/kg/day across all evaluated scenarios and age groups (Table 2-111).

Table 2-110. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as an Electronics Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (30)	Single Value (5)	95% (281.65)	User	130	22
				Bystander	27	6.3
Moderate Intensity User	50% (2)	Single Value (5)	50% (18.78)	User	9.2	1.5
				Bystander	1.3	0.34
Low Intensity User	10% (0.17) ¹	Single Value (5)	10% (1.50)	User	0.72	0.12
				Bystander	0.11	2.7E-02

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

Table 2-111. Consumer Dermal Exposure to Methylene Chloride During Use as an Electronics Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (30)	Single Value (5)	Adult (≥21 years)	0.25
			Youth (16-20 years)	0.23
			Youth (11-15 years)	0.26
Moderate Intensity User	50% (2)	Single Value (5)	Adult (≥21 years)	4.9E-02
			Youth (16-20 years)	4.6E-02
			Youth (11-15 years)	5.0E-02
Low Intensity User	10% (0.17) ¹	Single Value (5)	Adult (≥21 years)	1.3E-02
			Youth (16-20 years)	1.2E-02
			Youth (11-15 years)	1.4E-02

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

2.4.2.4.12 Engine Cleaner

Two products used as an engine cleaner were found to contain methylene chloride in weight fractions between 20-70% (Table 2-112). Inhalation exposures were evaluated for users and

bystanders for 27 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 154 – 5,096 mg/m³ for users and from 18 – 958 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for nine scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 0.52 – 23 mg/kg/day across all evaluated scenarios and age groups (Table 2-113).

Table 2-112. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as an Engine Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (120)	Max (70)	95% (1603.88)	User	5,096	1,347
				Bystander	958	377
Moderate Intensity User	50% (15)	Midpoint (45)	50% (387.60)	User	1,347	221
				Bystander	164	53
Low Intensity User	10% (5)	Min (20)	10% (97.24)	User	154	25
				Bystander	18	5.8

Table 2-113. Consumer Dermal Exposure to Methylene Chloride During Use as an Engine Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (120)	Max (70)	Adult (≥21 years)	22
			Youth (16-20 years)	21
			Youth (11-15 years)	23
Moderate Intensity User	50% (15)	Midpoint (45)	Adult (≥21 years)	5.6
			Youth (16-20 years)	5.2
			Youth (11-15 years)	5.7
Low Intensity User	10% (5)	Min (20)	Adult (≥21 years)	0.56
			Youth (16-20 years)	0.52
			Youth (11-15 years)	0.57

2.4.2.4.13 Gasket Remover

One product used as a gasket remover was found to contain methylene chloride in weight fractions between 60-80% (Table 2-114). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 142 – 3,769 mg/m³ for

users and from 16 – 590 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Permeability submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 0.52 – 23 mg/kg/day across all evaluated scenarios and age groups (Table 2-115).

Table 2-114. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Gasket Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (60)	Max (80)	95% (790.05)	User	3,769	682
				Bystander	590	212
Moderate Intensity User	50% (15)	Max (80)	50% (122.77)	User	758	125
				Bystander	92	30
Low Intensity User	10% (2)	Min (60)	10% (29.77)	User	142	23
				Bystander	16	5.3

Table 2-115. Consumer Dermal Exposure to Methylene Chloride During Use as a Gasket Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (60)	Max (80)	Adult (≥21 years)	22
			Youth (16-20 years)	21
			Youth (11-15 years)	23
Moderate Intensity User	50% (15)	Max (80)	Adult (≥21 years)	5.6
			Youth (16-20 years)	5.2
			Youth (11-15 years)	5.7
Low Intensity User	10% (2)	Min (60)	Adult (≥21 years)	0.56
			Youth (16-20 years)	0.52
			Youth (11-15 years)	0.57

2.4.2.4.14 Sealants

One product used as a sealant was found to contain methylene chloride in weight fractions between 10-30% (Table 2-116). Inhalation exposures were evaluated for users and bystanders for 18 different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 24 – 1,430 mg/m³ for users and from 2.8 – 224 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios

using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user, moderate intensity user and high intensity user scenarios ranged from 7.6E-02 – 1.3 mg/kg/day across all evaluated scenarios and age groups (Table 2-117).

Table 2-116. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Sealant

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (60)	Max (30)	95% (799.19)	User	1,430	259
				Bystander	224	80
Moderate Intensity User	50% (15)	Max (30)	50% (124.19)	User	288	47
				Bystander	35	11
Low Intensity User	10% (2)	Min (10)	10% (30.12)	User	24	3.9
				Bystander	2.8	0.89

Table 2-117. Consumer Dermal Exposure to Methylene Chloride During Use as a Sealant

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (60)	Max (30)	Adult (≥21 years)	1.3
			Youth (16-20 years)	1.2
			Youth (11-15 years)	1.3
Moderate Intensity User	50% (15)	Max (30)	Adult (≥21 years)	1.0
			Youth (16-20 years)	0.96
			Youth (11-15 years)	1.0
Low Intensity User	10% (2)	Min (10)	Adult (≥21 years)	8.1E-02
			Youth (16-20 years)	7.6E-02
			Youth (11-15 years)	8.3E-02

2.4.2.4.15 Weld Spatter Protectant

One product used as a weld spatter protectant was found to contain methylene chloride in weight fractions >90% (Table 2-118). Inhalation exposures were evaluated for users and bystanders for nine different scenarios of duration of use, weight fraction and mass of use. Three scenarios are presented below as low intensity user, high intensity user and moderate intensity user scenarios, with 1-hr maximum TWA concentrations ranging from 181 – 5,111 mg/m³ for users and from 16 – 648 mg/m³ for bystanders across scenarios. Dermal exposures were evaluated for six scenarios using the CEM Fraction Absorbed submodel. Selected scenarios representing low intensity user,

moderate intensity user and high intensity user scenarios ranged from 0.23 – 5.0 mg/kg/day across all evaluated scenarios and age groups (Table 2-119).

Table 2-118. Consumer User and Bystander Inhalation Exposure to Methylene Chloride During Use as a Weld Spatter Protectant

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass of Use (g)	Product User or Bystander	1 hr Max TWA (mg/m ³)	8 hr Max TWA (mg/m ³)
High Intensity User	95% (60)	Single Value (90)	95% (569.43)	User	5111	836
				Bystander	648	198
Moderate Intensity User	50% (5)	Single Value (90)	50% (84.06)	User	897	136
				Bystander	81	24
Low Intensity User	10% (0.25) ¹	Single Value (90)	10% (17.43)	User	181	28
				Bystander	16	4.9

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

Table 2-119. Consumer Dermal Exposure to Methylene Chloride During Use as a Weld Spatter Protectant

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High Intensity User	95% (60)	Single Value (90)	Adult (≥21 years)	4.9
			Youth (16-20 years)	4.6
			Youth (11-15 years)	5.0
Moderate Intensity User	50% (5)	Single Value (90)	Adult (≥21 years)	2.0
			Youth (16-20 years)	1.8
			Youth (11-15 years)	2.0
Low Intensity User	10% (0.25) ¹	Single Value (90)	Adult (≥21 years)	0.25
			Youth (16-20 years)	0.23
			Youth (11-15 years)	0.25

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

2.4.2.5 Monitoring Data

2.4.2.5.1 Indoor Residential Air

Concentrations of methylene chloride in the indoor air of residential homes in the U.S. and Canada from 9 studies identified during Systematic Review are summarized in Table 2-120. Overall, more than 700 samples were collected between 1986 and 2010 in five U.S. states (CO, IL, MA, MI, and MN) and Canada (exact location not reported). Concentrations ranged from non-detect (limits varied) to 1,190 $\mu\text{g}/\text{m}^3$. The highest concentrations were from the Van Winkle et. al. (2001) study, which notes that the high methylene chloride concentrations are likely associated with analytical artifacts. Excluding this study, maximum concentrations of 147 and 176 $\mu\text{g}/\text{m}^3$ were observed in garages of residences in Boston, MA (Dodson et al., 2008) and in inner city homes in New York, NY (Sax et al., 2004), respectively. Maximum concentrations were much lower in other studies, generally less than 15 $\mu\text{g}/\text{m}^3$. Excluding the Van Winkle et. al. (2001) study, measures of central tendency (reported average or median) across all datasets were generally less than 10 $\mu\text{g}/\text{m}^3$, except for the Canadian study at 27 $\mu\text{g}/\text{m}^3$.

Data extracted for residential indoor air samples from studies conducted outside of North America, as well as studies conducted in schools and commercial establishments in the U.S. and other countries, is provided in *Systematic Review Supplemental File: Data Extraction Tables for Consumer and Environmental Exposure Studies*.

Table 2-120. Concentrations of Methylene Chloride in the Indoor Air of Residential Homes in the U.S. and Canada from Studies Identified During Systematic Review

Study Info	Site Description	Detect. Limit	Min.	Mean	Median	Max.	Variance	Data Eval. Score
(Chin et al., 2014); U.S., 2009-2010 (n=126; DFq = 0.06)	Detroit, MI area; Homes (n=126) with asthmatic children, sampled in living rooms and bedroom	0.71	ND	0.54	0.71	7.85	0.91 (SD)	High
(Dodson et al., 2008); U.S., 2004-2005 (n=16; DFq = 0.25)	Boston, MA; Garage of residences	0.39-1.25	ND	9.8	0.3	147 (95th)	36 (SD)	High
(Dodson et al., 2008); U.S., 2004-2005 (n=10; DFq = 0.2)	Boston, MA; Apartment hallway of residences	0.39-1.25	ND	2.6	0.4	15 (95th)	4.6 (SD)	High
(Dodson et al., 2008); U.S., 2004-2005 (n=52; DFq = 0.42)	Boston, MA; Basement of residences	0.39-1.25	ND	9.5	0.4	0.66 (95th)	28 (SD)	High
(Dodson et al., 2008); U.S., 2004-2005 (n=83; DFq = 0.4)	Boston, MA; Interior room of residences	0.39-1.25	ND	0.28	0.21	10 (95th)	8.7 (SD)	High
(Adgate et al., 2004); U.S., 2000 (n=113; DFq = 0.202)	Minneapolis, MN in spring; Child's primary residence	-- ^b	ND (0.2 10th)	--	0.3	1.2 (90th)	--	Medium
(Adgate et al., 2004); U.S., 2000 (n=113; DFq = 0.232)	Minneapolis, MN in winter; Child's primary residence.	-- ^b	ND (0.2 10th)	--	0.4	1.3 (90th)	--	Medium

Study Info	Site Description	Detect. Limit	Min.	Mean	Median	Max.	Variance	Data Eval. Score
(Sax et al., 2004); U.S., 2000 (n=32; DFq = 1)	Los Angeles, CA in fall; Homes in inner-city	0.22	0.2	1.4	1.1	4.3	1.2 (SD)	High
(Sax et al., 2004); U.S., 2000 (n=40; DFq = 0.95)	Los Angeles, CA in winter; Homes in inner-city	0.27	0.27	2.4	1.9	8.7	2 (SD)	High
(Sax et al., 2004); U.S., 1999 (n=30; DFq = 0.28)	New York, NY in summer; Homes in inner-city	1.63	1.63	10	1.4	176	32.9 (SD)	High
(Sax et al., 2004); U.S., 1999 (n=36; DFq = 0.97)	New York, NY in winter; Homes in inner-city	0.22	0.2	5.5	2.2	69	12.3 (SD)	High
(Van Winkle and Scheff, 2001); U.S., 1994-1995 (n=48; DFq = 1)	Southeast Chicago, IL; Urban homes (n=10) sampled over a 10-month period, from the kitchen in the breathing zone.	--	0.76 ^c	140 ^c	60.5 ^c	1190 ^c	235 (SD)	High
(Lindstrom et al., 1995); U.S., 1994 (n=9; DFq = 0.78)	Denver, CO; Homes, pre-occupancy (n=8)	0.14	0.14	2.64	1.57	--	2.63 (SD)	Medium
(Wallace et al., 1991); U.S., 1991 (n= 8; DFq = 1)	Los Angeles, CA in summer; Kitchens and living-area	--	--	5.6	--	14	1.4 (SE)	Medium
(Chan et al., 1990); Canada, 1986 (n=12; DFq = 0.92)	Homes (n=12), main floor	--	ND	9.1	--	--	--	Medium
(Chan et al., 1990); Canada, 1987 (n=6; DFq = 1)	Homes (n=6), main floor	--	4	26.9	--	--	--	Medium

Abbreviations: If a value was not reported, it is shown in this table as "--". ND = not detected at the reported detection limit. GM = geometric mean. GSD = geometric standard deviation. DFq = detection frequency. NR = Not reported. U.S.

Parameters: All statistics are shown as reported in the study. Some reported statistics may be less than the detection limit; the method of handling non-detects varied by study. All minimum values determined to be less than the detection limit are shown in this table as "ND". If a maximum value was not provided, the highest percentile available is shown (as indicated in parentheses); if a minimum value was not provided, the lowest percentile available is shown (as indicated in parentheses).

^a Samples from this study ([Dodson et al., 2008](#)) were collected as part of the BEAMS study.

^b No quantitative detection limit was provided in Adgate et al. (2004), however Chung et al (1999) was cited as the basis for the precision, accuracy, and suitability of the sampling methodology used. A detection limit of 0.9 µg/m³ was identified within Chung et al. (1999) and can be reasonably applied to Adgate et al. (2004) due to the similarities in their sampling and analytical methodologies.

^c Elevated methylene chloride concentrations likely associated with analytical artifact ([Van Winkle and Scheff, 2001](#)).

2.4.2.5.2 Personal Breathing Zone Data

Concentrations of methylene chloride in the personal breathing zones of residents in the U.S. from two studies identified during Systematic Review are summarized in Table 2-121. Overall,

more than 500 personal monitoring samples from 48-hr monitoring periods were collected between 1999 and 2000 in one U.S. state (MN). Reported concentrations ranged from non-detect (limits varied) to 13.6 $\mu\text{g}/\text{m}^3$; and central tendency values (reported mean or median) ranged from 0.3 to 6.7 $\mu\text{g}/\text{m}^3$. The maximum concentration of 13.6 $\mu\text{g}/\text{m}^3$ is a 90th percentile value based on an overall average of 70 non-smoking adults during spring, summer, and fall sampling and spending 89% of their time indoors (home, work, school), 6.4% outdoors, and 4.5% in transit ([Sexton et al., 2007](#)). The second study ([Adgate et al., 2004](#)) observed personal exposure to methylene chloride for 80 children while spending 66% of their time at home, 25.2% of their time at school, 1.5% of their time playing outdoors, and 3.8% of their time in transit during the spring and winter. There was a 10-fold difference between the maximum values reported in the two studies.

Data extracted for residential personal breathing zone samples from studies conducted outside of North America, as well as studies conducted in schools and commercial establishments in the U.S. and other countries, is provided in the *Supplemental Information on Consumer Exposure Assessment* ([EPA, 2019g](#)).

Table 2-121. Concentrations of Methylene Chloride in the Personal Breathing Zones of Residents in the U.S.

Study Info	Site Description	Detect. Limit	Min.	Mean	Median	Max.	Variance	Data Eval. Score
(Sexton et al., 2007) ; U.S., 1999 (n=333; DFq = 1)	Minneapolis-St. Paul, MN; Non-smoking adults (n=70); three neighborhoods: (inner-city/economically disadvantaged, blue-collar/near manufacturing plants, and affluent); indoors, outdoors, and in transit.	--	0.4 (10)	6.7	1.4	13.6 (90 th)	--	High
(Adgate et al., 2004) ; U.S., 2000 (n=113; DFq = 0.17)	Minneapolis, MN in spring; Child's primary residence, school, outside, and in transit	-- ^a	ND (0.2 10 th)	--	0.3	1.3 (90 th)	--	Medium
(Adgate et al., 2004) ; U.S., 2000 (n=113; DFq = 0.194)	Minneapolis, MN in winter; Child's primary residence, school, outside, and in transit.	-- ^a	ND (0.2 10 th)	--	0.4	1.3 (90 th)	--	Medium

Abbreviations: If a value was not reported, it is shown in this table as "--". ND = not detected at the reported detection limit.

Parameters: All statistics are shown as reported in the study. Some reported statistics may be less than the detection limit; the method of handling non-detects varied by study. All minimum values determined to be less than the detection limit are shown in this table as "ND". If a maximum value was not provided, the highest percentile available is shown (as indicated in parentheses); if a minimum value was not provided, the lowest percentile available is shown (as indicated in parentheses).

^a No quantitative detection limit was provided in Adgate et al. (2004), however Chung et al (1999) was cited as the basis for the precision, accuracy, and suitability of the sampling methodology used. A detection limit of 0.9 $\mu\text{g}/\text{m}^3$ was identified within Chung et al. (1999) and can be reasonably applied to Adgate et al. (2004) due to the similarities in their sampling and analytical methodologies.

2.4.2.6 Modeling Confidence in Consumer Exposure Results

Overall, there is medium to high or high confidence in the consumer inhalation exposure modeling approach and results (Table 2-122). This is based on the strength of the model employed, as well as the quality and relevance of the default, user-selected and varied modeling inputs. CEM 2.1.7 is a peer reviewed, publicly available model that was designed to estimate inhalation and dermal exposures from household products and articles. CEM uses central-tendency default values for sensitive inputs such as building and room volumes, interzonal ventilation rate, and air exchange rates. These parameters were not varied due to EPA having greater confidence in the central tendency inputs for such factors that are outside of a user's control (unlike, e.g., mass of product used or use duration). These central tendency defaults are sourced from EPA's Exposure Factors Handbook (EPA, 2011a). The confidence in the user-selected varied inputs (i.e., mass used, use duration, and weight fraction) are medium to high, depending on the condition of use. The sources of these data are U.S. EPA (1987) (high-quality) and company-generated SDSs. What reduces confidence for particular conditions of use is the relevance or similarity of the U.S. EPA (1987) survey product category for the modeled condition of use. For instance, the evaluated brake cleaner scenario had surveyed information directly about this condition of use within U.S. EPA (1987), resulting in a high confidence in model default values. In contrast, the coil cleaner scenario did not have an exact match within U.S. EPA (1987), resulting in use of a surrogate scenario selected by professional judgement that most closely approximates the use amount and duration associated with this condition of use. Additionally, in some cases, professional judgment or surveyed information from U.S. EPA (1987) was used in selection of room of use, which sets the volume for modeling zone 1.

Dermal exposure modeling results overall were rated as low to medium (Table 2-123). The processes and inputs described for the inhalation scenarios above are also valid for the dermal exposure scenarios. While the model used for dermal exposure estimates was the same as used for the inhalation exposure estimates, there is overall low to medium (vs. high for inhalation) confidence in the model used due to the used dermal submodels. As described in Section 2.4.2.3.1.2, the evaluation of dermal exposures used a fraction absorbed or permeability submodel depending on condition of use. Both of these models have inherent assumptions included in their calculations which may over or underestimate calculated dermal exposures. For instance, the fraction absorbed submodel assumes that the entire mass of the chemical found in the film thickness enters the skin. This may overestimate exposure as some surface evaporation would be expected. Conversely, the model may underestimate exposures since it assumes the given thin film is only applied once and does not account for situations where multiple application events may be possible, particularly during high duration conditions of use. The permeability submodel also may overestimate exposures since it assumes a constant supply of chemical over the length of the exposure duration. While indicative of impeded exposure conditions, such a scenario is unlikely as impeded use conditions would be likely to be intermittent and not constant in nature. These and other assumptions and uncertainties are further discussed in Section 4.3.3.

Table 2-122. Confidence in Individual Consumer Conditions of Use Inhalation Exposure Evaluations

Consumer Condition of Use	Form	Confidence in Model Used ¹	Confidence in Model Default Values ²	Confidence in User-Selected Varied Inputs ³				Overall Confidence
				Mass Used ⁴	Use Duration ⁵	Weight Fraction ⁶	Room of Use ⁷	
Automotive AC Leak Sealer	Aerosol	High	High	Medium	Medium	High	High	Medium to High
Automotive AC Refrigerant	Aerosol	High	High	Medium	Medium	High	High	Medium to High
Adhesives	Liquid	High	High	High	High	High	Medium	High
Adhesives Remover	Liquid	High	High	High	High	High	Medium	High
Brake Cleaner	Aerosol	High	High	High	High	High	High	High
Brush Cleaner	Liquid	High	High	Medium	Medium	High	Medium	Medium to High
Carbon Remover	Aerosol	High	High	High	High	High	High	High
Carburetor Cleaner	Aerosol	High	High	High	High	High	High	High
Coil Cleaner	Aerosol	High	High	Medium	Medium	High	High	Medium to High
Cold Pipe Insulating Spray	Aerosol	High	High	Medium	Medium	High	High	Medium to High
Electronics Cleaner	Aerosol	High	High	High	High	High	High	High
Engine Cleaner	Aerosol	High	High	High	High	High	High	High
Gasket Remover	Aerosol	High	High	High	High	High	High	High
Sealant	Aerosol	High	High	High	High	High	High	High
Weld Spatter Protectant	Aerosol	High	High	Medium	Medium	High	High	Medium to High

¹Confidence in Model Used considers whether model has been peer reviewed and whether model is applied in a manner appropriate to its design and objective. The model used (CEM 2.1) has been peer reviewed, is publicly available, and has been applied in a manner intended.

²Confidence in Model Default Values considers default value data source(s) such as building and room volumes, interzonal ventilation rates, and air exchange rates. These default values are all central tendency values (i.e., mean or median values) sourced from EPA's Exposure Factors Handbook ([EPA, 2011a](#)). The one default value with a high-end input is the overspray fraction, which is used in the aerosol or spray scenarios and assumes a certain percentage is immediately available for inhalation.

Consumer Condition of Use	Form	Confidence in Model Used ¹	Confidence in Model Default Values ²	Confidence in User-Selected Varied Inputs ³			Overall Confidence
				Mass Used ⁴	Use Duration ⁵	Weight Fraction ⁶	
<p>³Confidence in User-Selected Varied Inputs considers the quality of their data sources, as well as relevance of the inputs for the selected consumer condition of use.</p> <p>⁴Mass Used is primarily sourced from the U.S. EPA (1987), which received a high-quality rating during data evaluation and has been applied in previous agency assessments. Automotive AC Leak Sealer mass used was derived by directions on product.</p> <p>⁵Use Duration is primarily sourced from U.S. EPA (1987), which received a high-quality rating during data evaluation and has been applied in previous agency assessments.</p> <p>⁶Weight fraction of methylene chloride in products is sourced from product SDSs, which were not reviewed as part of systematic review but were taken as authoritative sources on a product's ingredients.</p> <p>⁷Room of use (zone 1 in modeling) is informed by responses in U.S. EPA (1987) which received a high-quality rating during data evaluation, although professional judgment is also applied for some scenarios.</p>							

Table 2-123. Confidence in individual consumer conditions of use for dermal exposure evaluations

Consumer Condition of Use	Form	Confidence in Model Used ¹	Confidence in Model Default Values ²	Confidence in User-Selected Varied Inputs ³			Overall Confidence
				Use Duration ⁴	Weight Fraction ⁵	Room of Use ⁶	
Adhesives	Liquid	Low to Medium	High	High	High	Medium	Low to Medium
Adhesives Remover	Liquid	Low to Medium	High	High	High	Medium	Low to Medium
Automotive AC Leak Sealer	Aerosol	Low to Medium	High	Medium	High	High	Low to Medium
Automotive AC Refrigerant	Aerosol	Low to Medium	High	Medium	High	High	Low to Medium
Brake Cleaner	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Brush Cleaner	Liquid	Low to Medium	High	Medium	High	Medium	Low to Medium
Carbon Remover	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Carburetor Cleaner	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Coil Cleaner	Aerosol	Low to Medium	High	Medium	High	High	Low to Medium

Table 2-123. Confidence in individual consumer conditions of use for dermal exposure evaluations

Cold Pipe Insulating Spray	Aerosol	Low to Medium	High	Medium	High	High	Low to Medium
Electronics Cleaner	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Engine Cleaner	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Gasket Remover	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Sealant	Aerosol	Low to Medium	High	High	High	High	Low to Medium
Weld Spatter Protectant	Aerosol	Low to Medium	High	Medium	High	High	Low to Medium

¹Confidence in Model Used considers whether model has been peer reviewed and whether model is applied in a manner appropriate to its design and objective. The model used (CEM 2.1) has been peer reviewed, is publicly available, and has been applied in a manner intended.

²Confidence in Model Default Values considers default value data source(s) such as surface area to body weight ratios for the dermal contact area. These default values are all central tendency values (i.e., mean or median values) sourced from EPA's Exposure Factors Handbook ([EPA, 2011a](#)).

³Confidence in User-Selected Varied Inputs considers the quality of their data sources, as well as relevance of the inputs for the selected consumer condition of use.

⁴Use Duration is primarily sourced from U.S. EPA ([1987](#)), which received a high-quality rating during data evaluation and has been applied in previous agency assessments.

⁵Weight fraction of methylene chloride in products is sourced from product SDSs, which were not reviewed as part of systematic review but were taken as authoritative sources on a product's ingredients.

⁶Room of use (zone 1 in modeling) is informed by responses in U.S. EPA ([1987](#)) which received a high-quality rating during data evaluation, although professional judgment is also applied for some scenarios.

3 HAZARDS

3.1 Environmental Hazards

3.1.1 Approach and Methodology

During scoping and problem formulation, EPA reviewed potential environmental health hazards associated with methylene chloride. EPA identified the following sources of environmental hazard data: *TSCA Work Plan Chemical Risk Assessment Methylene Chloride: Paint Stripping Use CASRN 75-09-2* ([U.S. EPA, 2014](#)), *Dichloromethane: Screening Information DataSet (SIDS) Initial Assessment Profile* ([OECD, 2011](#)), *Environmental Health Criteria 164 Methylene Chloride* ([WHO, 1996a](#)), *Canadian Environmental Protection Act Priority Substances List Assessment Report: Dichloromethane* ([Health Canada, 1993](#)), and *Ecological Hazard Literature Search Results in Methylene Chloride (CASRN 75-09-2) Bibliography: Supplemental File for the TSCA Scope Document (EPA-HQ-OPPT-2016-0742-0059)* ([U.S. EPA, 2017a](#)).

EPA completed the review of environmental hazard data/information sources during risk evaluation using the data quality review evaluation metrics and the rating criteria described in the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). Studies were assigned an overall quality level of high, medium, or low. The data quality evaluation results are outlined in *Supplemental File: Data Quality Evaluation of Environmental Hazard Studies* ([EPA, 2019r](#)). With the data available, EPA only used studies with an overall quality level of high or medium for quantitative analysis during data integration. Studies assigned an overall quality level of low were used qualitatively to characterize the environmental hazards of methylene chloride. Any study assigned an overall quality level of unacceptable was not used for data integration.

3.1.2 Hazard Identification

Toxicity to Aquatic Organisms

EPA assigned an overall quality level of high, medium, or low to 14 acceptable studies, including two studies submitted as “substantial risk” notifications under Section 8(e). These studies contained relevant aquatic toxicity data for amphibians, fish, aquatic invertebrates, and aquatic plants. EPA identified 11 aquatic toxicity studies, displayed in Table 3-1, as the most relevant for quantitative assessment. The rationale for selecting these studies is provided in Section 3.1.3 Weight of Scientific Evidence.

Aquatic Environmental Hazards from Acute Exposures to Methylene Chloride

Amphibians: Seven amphibian species were exposed to methylene chloride for up to five and a half days in two flow-through studies, which EPA assigned an overall quality level of high ([Black et al., 1982](#); [Birge et al., 1980](#)). Birge (1980) exposed embryos and larvae of *Anaxyrus fowleri* (Fowler’s toad, hatches in 3 days), *Lithobates palustris* (pickerel frog, hatches in 4 days), and *Rana catesbeiana* (American bullfrog, hatches in 4 days) to methylene chloride through 4

days post-hatch. Black (1982) tested *Rana temporaria* (common European frog, hatches in 5 days), *Xenopus laevis* (African clawed frog, hatches in 2 days), *Lithobates pipiens* (leopard frog, hatches in 5 days), and *Ambystoma gracile* (Northwestern salamander) through 4 days post-hatch. The concentration of methylene chloride lethal to half the population (median lethal concentration, or LC₅₀) of *R. catesbeiana* embryos, exposed for 4 days, was 30.6 mg/L, and for *R. temporaria* embryos exposed for 5 days was 23 mg/L (Birge et al., 1980). Definitive LC₅₀s were not established for embryos of *A. fowleri* (> 32 mg/L), *L. palustris* (> 32 mg/L), *X. laevis* (> 29 mg/L), and *L. pipiens* (> 48 mg/L), which were exposed from 2 to 5 days to the highest concentrations tested. The embryos of the Northwestern salamander, *A. gracile*, had an LC₅₀ of 23.9 mg/L after 5.5 days of exposure, similar to *R. temporaria* and *R. catesbeiana* (Black et al., 1982). However, because the exposure duration was a borderline sub-chronic value, and because salamanders have a different biology (i.e., gill structure) from the frogs tested, EPA did not integrate this hazard value with the frog results. The two amphibian studies demonstrate the variation in amphibian species sensitivity to methylene chloride, with the bullfrog, *R. catesbeiana* having the greatest sensitivity to the chemical substance. Both study authors included embryo teratogenesis, which they defined as the percent of survivors with gross and debilitating abnormalities likely to result in eventual mortality, into the LC₅₀ values and adjusted for controls. EPA integrated the definitive LC₅₀ values for *R. temporaria* (common European frog) and *R. catesbeiana* (American bullfrog) into a geometric mean of 26.3 mg/L (Black et al., 1982; Birge et al., 1980).

Fish: EPA assigned an overall quality level of high to three acute (96-hr; flow-through) fish toxicity studies, which evaluated the median lethal concentrations (LC₅₀s) of methylene chloride to *Pimephales promelas* (fathead minnow) or *Oncorhynchus mykiss* (rainbow trout) (Dill et al., 1987; E I Dupont Denemours & Co Inc, 1987b; Geiger et al., 1986). EPA assigned one study that used adult *P. promelas* obtained from a bait company with an overall quality level of medium (Alexander et al., 1978). Dill (1987) noted loss of equilibrium, a sub-lethal effect, in juvenile *P. promelas* exposed to methylene chloride at concentrations > 357 mg/L for exposures from 24 hours to test termination at 192 hours. The 96-hour LC₅₀ for fathead minnows was 502 mg/L. Alexander (1978) established an LC₅₀ of 193 mg/L for adult *P. promelas* exposed to methylene chloride for 96 hours. The authors also reported an EC₅₀ of 99 mg/L for immobilization in fathead minnows exposed to methylene chloride. The authors defined immobilization as fish with loss of equilibrium, melanization, narcosis, and swollen, hemorrhaging gills. E I Dupont Denemours & Co Inc (1987b) established a 96-hour LC₅₀ of 108 mg/L in *O. mykiss*. The authors observed rainbow trout exposed to methylene chloride concentrations ≥ 39 mg/L swimming at the surface, swimming erratically, and/or exhibiting melanization. The 96-hr LC₅₀s from the high and medium quality-level studies ranged from 108 mg/L to 502 mg/L. EPA integrated the acute 96-hour LC₅₀ values for hazard evaluation into a geometric mean of 242.4 mg/L.

Aquatic Invertebrates: For freshwater aquatic invertebrates, EPA assigned two studies with *Daphnia magna* (water flea) acute (48-hr EC₅₀; static) exposures to methylene chloride with an overall quality level of high (E I Dupont Denemours & Co Inc, 1987a; Leblanc, 1980). EPA assigned one study on *D. magna* an overall quality level of medium (Abernethy et al., 1986), and one study an overall quality level of low (Kuhn et al., 1989). The EC₅₀ values for the studies that EPA assigned medium or high overall quality levels ranged from 135.8 mg/L to 177 mg/L for

48-hour exposures to methylene chloride. LeBlanc (1980) established a 48-hour LC₅₀ of 176 mg/L. For aquatic invertebrates, EC₅₀s and LC₅₀s are calculated using the same methodologies and integrated together, because mortality is difficult to distinguish from immobilization. EPA integrated these hazard values into a geometric mean of 180 mg/L. LeBlanc (1980) also established a no observed effect concentration (NOEC) for mortality in *D. magna* exposed to methylene chloride concentrations of 54.4 mg/L for 48 hrs. This NOEC value is used to contrast with the EC₅₀s and LC₅₀s as the concentration at which methylene chloride is not expected to have an effect on aquatic invertebrates on an acute exposure basis.

EPA assigned one saltwater invertebrate (*Palaemonetes pugio*, daggerblade grass shrimp) study an overall quality level of high (Wilson, 1998), however, the authors did not provide a test substance source or substance purity information. The authors reported up to a three-day developmental delay for saltwater shrimp embryos exposed to 0.1 % v/v of methylene chloride for 96-hrs, and complete developmental arrest for embryo and larvae exposed to > 0.5 % v/v for 96-hrs. However, the test concentrations were reported in percent volume to volume (% v/v), and EPA could not accurately convert these values to weight per volume (mg/L) without making an assumption about the test substance purity. Because the study could not be compared to other data (i.e., freshwater invertebrates), it had lower relevance and, therefore, was not integrated into the risk evaluation.

There were no aquatic sediment studies available for methylene chloride; however, EPA was able to use a surrogate species to estimate toxicity. EPA considered using data on sediment species from analogous chemicals, but no appropriate analogue with appropriate data was identified for methylene chloride. Instead, because sediment organisms are expected to be exposed to freely dissolved methylene chloride in the surface water or pore water, daphnids were used as a surrogate species for estimating hazard in sediment invertebrates.

Aquatic Environmental Hazards from Subchronic and Chronic Exposures to Methylene Chloride

Amphibians: There were no chronic studies that encompassed amphibian metamorphoses and adult reproductive stages of the amphibian lifecycle. However, in the available, acceptable studies, amphibian embryo and larvae were the most sensitive life stages to subchronic exposures to methylene chloride in the aquatic environment. In the two studies by Birge (1980) and Black (1982) that EPA assigned an overall quality level of high, the authors continued exposures of embryos and larvae of seven amphibian species (*A. fowleri*, *R. catesbeiana*, *L. palustris*, *R. temporaria*, *X. laevis*, *L. pipiens*, and *A. gracile*) to methylene chloride for an additional 4 days post-hatch under flow-through conditions. The study authors included teratogenic embryos and larvae in mortality calculations to establish a 10% impairment value (LC₁₀) and LC₅₀ for *R. catesbeiana* (Birge et al., 1980) and *R. temporaria* (Black et al., 1982) exposed for 8 days and 9 days to methylene chloride, respectively. At control-adjusted concentrations, the LC₁₀ for *R. catesbeiana* was 1 mg/L, and the LC₁₀ for *R. temporaria* was 0.8 mg/L. The control-adjusted LC₅₀ for *R. catesbeiana* embryo and larvae exposed for 8 days was 17.8 mg/L, and for *R. temporaria* embryo and larvae exposed for 9 days was 16.9 mg/L. Impairment values and definitive LC₅₀s were not established for embryos of *A. fowleri*, *L. palustris*, *X. laevis*, and *L. pipiens* exposed for 6 to 9 days to the highest concentrations tested, because these species were

considerably more tolerant to exposures to methylene chloride. The authors determined a 9.5-day LC₅₀ of 17.8 mg/L for *A. gracile*, which is similar to the bullfrog and common frog hazard values, but because salamanders have a different biology from frogs, EPA did not integrate the data for *A. gracile*. A LC₁₀ was not established for this species. EPA integrated the bullfrog and common European frog LC₁₀s into a geometric mean of 0.9 mg/L, and their LC₅₀s into a geometric mean of 17.3 mg/L. EPA applied the acute-to-chronic ratio (ACR) of 10 to the integrated acute amphibian larval toxicity value of 26.3 mg/L for the more protective LC₅₀ value of 2.6 mg/L.

Fish: In fish, there were two studies with chronic exposure aquatic toxicity data, an *O. mykiss* (rainbow trout) study with embryos and larvae exposed to methylene chloride under flow-through conditions for up to 27 days ([Black et al., 1982](#)), and a study with *P. promelas* embryos and larvae exposed for 32 days ([Dill et al., 1987](#)). Both authors also had sub-chronic toxicity values for *P. promelas* (fathead minnow). After 9 days of exposure to methylene chloride, the minnow embryo and larvae (which hatched on day 4 of exposures) in the Black ([1982](#)) study had LC₅₀s > 34 mg/L, the highest concentration tested. In the chronic test with *O. mykiss* by Black ([1982](#)), the LC₅₀ for rainbow trout embryos exposed up to hatching at 23 days was 13.5 mg/L, and the LC₅₀ for larvae exposed up to four days post-hatch at 27 days was 13.2 mg/L. EPA integrated the trout data into a geometric mean of 13.3 mg/L. The Black ([1982](#)) study also indicated that there were no effects on survival of *O. mykiss* larvae exposed to methylene chloride at concentrations of 0.008 mg/L with survival decreasing to 85% at 0.4 mg/L, and 44% at 23.1 mg/L. The authors did not establish that the decreased survival at 0.4 mg/L was statistically significant, although survival data was adjusted for control mortalities. The authors noted teratic larvae were observed at exposure concentrations of 5.5 mg/L (the next highest test concentration) or greater. EPA considered the concentration of 0.4 mg/L as the NOEC for this study, and 5.5 mg/L as the lowest observed effect concentration (LOEC), and integrated these values into a geometric mean chronic toxicity value (ChV) for fish of 1.5 mg/L. *P. promelas* juveniles exposed for 8-days in the Dill ([1987](#)) sub-chronic study had an LC₅₀ of 471 mg/L. In the Dill ([1987](#)) 32-day study, there was statistically significant reduction in larval survival at the two highest concentrations tested, 209 and 321 mg/L, with 100% mortality within 96-hours post-hatch at 321 mg/L, which EPA interpreted as the 8-day LC₁₀₀ value for *P. promelas* embryos and larvae. The studies suggest that fathead minnow embryo and larvae are more sensitive to methylene chloride exposures than juveniles. The 32-day no observed effect concentration (NOEC) for mortality was 142 mg/L, and the lowest observed effect concentration (LOEC) for mortality was 209 mg/L. EPA integrated the 32-day NOEC and LOEC for mortality into a geometric mean, or maximum acceptable toxicant concentration (MATC) of 172.3 mg/L. Dill ([1987](#)) established a NOEC of 82.5 mg/L and a LOEC of 142 mg/L for loss of body weight in *P. promelas* exposed to methylene chloride, and a MATC of 108 mg/L from the geometric mean of the NOEC and LOEC.

Aquatic Invertebrates: There were no acceptable chronic exposure aquatic invertebrate studies, so EPA applied the acute to chronic ration (ACR) of 10 to the *D. magna* (water flea) acute EC₅₀/LC₅₀ integrated geometric mean of 180 mg/L to estimate the freshwater aquatic invertebrate chronic exposure toxicity value of 18 mg/L ([E I Dupont Denemours & Co Inc, 1987a](#); [Abernethy et al., 1986](#); [Leblanc, 1980](#)). In the absence of chronic exposure duration studies for aquatic invertebrates, EPA also used ECOSAR v.2.0, the Agency's application for estimating environmental hazards from industrial chemicals. ECOSAR classified methylene

chloride as a neutral organic, with a freshwater aquatic invertebrate ChV of 12 mg/L. ECOSAR also estimated a saltwater mysid ChV of 41.8 mg/L, which also falls within range of the aquatic invertebrate hazard value. The ECOSAR predicted ChVs support the freshwater invertebrate chronic hazard value of 18 mg/L.

Aquatic Plants (Algae): For aquatic plants hazard studies, algae are the common test species. Algae are cellular organisms which will cycle through several generations in hours to days, therefore the data for algae was assessed together regardless of duration (i.e., 48-hrs to 96-hrs).

For algae, there were two studies (under static conditions) that EPA assigned an overall quality level of high, a 72-hr exposure biomass inhibition in the green algae species *Chlamydomonas reinhardtii* (Brack and Rottler, 1994) and a 96-hr biomass inhibition (characterized by the authors as “the net production of algal cell density”) study with the green algae *Pseudokirchneriella subcapitata* (Tsai and Chen, 2007). The 96-hr EC₅₀ for *P. subcapitata* biomass inhibition was 33.1 mg/L, while the 72-hr EC₅₀ for *C. reinhardtii*, was 242 mg/L. The hazard value for *C. reinhardtii* is nearly an order of magnitude higher than the 96-hr EC₅₀ for *P. subcapitata*. While it is likely the hazard value for *C. reinhardtii* would have decreased had the study been extended to 96-hrs, the 72-hr EC₁₀ of 115 mg/L for 10% biomass inhibition in *C. reinhardtii* established by Brack (1994) is higher than the 96-hr EC₅₀ for *P. subcapitata*. The studies suggest that *P. subcapitata*, a static algal species that is an obligate phototroph, is more sensitive to methylene chloride exposures relative to *C. reinhardtii*, a motile algal species with two flagella that is a facultative heterotroph. In addition to the functional differences between the two algal species, the study durations vary by 24 hours, in which time multiple generations of algal cells would be produced. Therefore, the two hazard values were not integrated, and EPA used the 96-hour EC₅₀ of 33.1 mg/L for the more sensitive species, *P. subcapitata*, as the more protective value to represent hazards to green algae as a whole.

In one study that EPA assigned an overall quality level of medium, growth was measured via relative chlorophyll *a* absorbance in three green algae species, *C. vulgaris*, *P. subcapitata*, and *Volvulina steinii* exposed to methylene chloride under static conditions for 10 days (Ando et al., 2003). The study did not have critical details, such as analytical measurement of test concentrations, chemical substance source or purity, or an EC₅₀ calculated from the relative absorbance results. In addition, chlorophyll *a* is a pigment in the cells of algae that is an indirect indicator of growth that EPA does not consider relevant for hazard evaluation of green algae. Therefore, the study was not integrated into the environmental hazard calculation but is used here qualitatively. There was no significant change in the relative absorbance of chlorophyll *a* for *C. vulgaris* or *P. subcapitata* up to the highest nominal concentration tested, 2 mg/L. However, methylene chloride killed *V. steinii*, a flagellar alga, at the lowest nominal concentration tested, 0.002 mg/L. The authors attributed the variation in algal species sensitivity to methylene chloride to *V. steinii*'s high metabolism.

Table 3-1. Ecological Hazard Characterization of Methylene Chloride for Aquatic Organisms

Duration	Test organism	Endpoint (Freshwater)	Hazard values (mg/L)	Geometric Mean ¹ (mg/L)	Effect Endpoint	Citation (Data Evaluation Rating) ²
Acute	Amphibian	4 to 5-day LC ₅₀ (frog embryos & larvae)	23 - > 48	26.3	Teratogenesis Leading to Mortality	(Birge et al., 1980) (High); (Black et al., 1982) (High)
		5.5-day LC ₅₀ (salamander embryos & larvae)	23.9		Teratogenesis Leading to Mortality	(Black et al., 1982) (High)
	Fish	96-hour EC ₅₀ (adults)	99		Immobilization ³	(Alexander et al., 1978) (Medium)
		96-hour LC ₅₀ (juveniles and adults)	108 - 502	242.4	Mortality	(Alexander et al., 1978) (Medium); (Dill et al., 1987) (High); (Geiger et al., 1986) (High); (E I Dupont Denemours & Co Inc, 1987b) (High)
	Aquatic Invertebrate	48-hour EC ₅₀ /LC ₅₀	135.8 - 177	180	Immobilization and Mortality	(Abernethy et al., 1986) (Medium); (E I Dupont Denemours & Co Inc, 1987a) (High); (Leblanc, 1980) (High);
		48-hr NOEC	54.4			(Leblanc, 1980) (High)
Subchronic /Chronic	Amphibian	8 to 9-day (frog embryos & larvae) LC ₁₀	0.8 - 1	0.9	Teratogenesis Leading to Mortality	(Black et al., 1982) (High); (Birge et al., 1980) (High)
		LC ₅₀	16.9 - > 48	17.3		
	4 to 5-day LC ₅₀	2.6 (ACR10)	-			
		9.5-day LC ₅₀ (salamander embryos & larvae)	17.8		Teratogenesis Leading to Mortality	(Black et al., 1982) (High)
	Fish	8-day LC ₅₀ (juveniles)	471		Mortality	(Dill et al., 1987) (High)
		LC ₁₀₀ (embryos & larvae)	321			

Duration	Test organism	Endpoint (Freshwater)	Hazard values (mg/L)	Geometric Mean ¹ (mg/L)	Effect Endpoint	Citation (Data Evaluation Rating) ²
		9-day LC ₅₀ (embryo & larvae)	> 34		Teratogenesis Leading to Mortality	(Black et al., 1982) (High)
		23 to 27-day LC ₅₀ (embryo & larvae)	13.2 – 13.5	13.3	Teratogenesis Leading to Mortality	(Black et al., 1982) (High)
		23 to 27-day NOEC LOEC (embryo & larvae)	0.4 – 5.5	1.5	Teratogenesis	(Black et al., 1982) (High)
		32-day NOEC LOEC (embryo & larvae)	142 209	172.3 (MATC)	Mortality	(Dill et al., 1987) (High)
			82.5 142	108	Growth (Body Weight)	
	Aquatic invertebrate	48-hrs ⁴ EC ₅₀ /LC ₅₀	18 ⁴		Immobilization and Mortality	(Abernethy et al., 1986) (Medium); (E I Dupont Denemours & Co Inc. 1987a) (High); (Leblanc, 1980) (High)
	Algae	72-hour EC ₅₀	242		Biomass	(Tsai and Chen, 2007) (High); (Brack and Rottler, 1994) (High); (Ando et al., 2003)
		96-hour EC ₅₀	33.1			
		EC ₁₀	115		Biomass	(Brack and Rottler, 1994) (High)

¹ Geometric mean of definitive values only (i.e., > 48 mg/L was not used in the calculation).

² While the hazard values are presented in ranges, the citations represent all of the data included in the range presented.

³ Immobilization was reported by Alexander ([1978](#)) as loss of equilibrium, melanization, narcosis and swollen, hemorrhaging gills.

⁴ EPA applied the ACR of 10 to the geometric mean of the integrated acute duration aquatic invertebrate studies.

3.1.3 Weight of Scientific Evidence

During the data integration stage of systematic review EPA analyzed, synthesized, and integrated the data/information into Table 3-1. This involved weighing scientific evidence for quality and relevance, using a weight-of-scientific-evidence approach, as defined in 40 CFR 702.33, and noted in TSCA 26(i) ([U.S. EPA, 2018a](#)).

During data evaluation, EPA assigned studies an overall quality level of high, medium, or low based on the *TSCA criteria described in the Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). While integrating environmental hazard data for methylene chloride, EPA gave more weight to relevant data/information that were assigned an overall quality level of high or medium. Only data/information that EPA assigned an overall quality

level of high or medium was used for the environmental risk assessment. Data that EPA assigned an overall quality level of low was used to provide qualitative characterization of the effects of methylene chloride exposures in aquatic organisms. Any information that EPA assigned an overall quality of unacceptable was not used. EPA determined that data and information were relevant based on whether it had biological, physical/chemical, and environmental relevance ([EPA, 1998](#)):

- Biological relevance: correspondence among the taxa, life stages, and processes measured or observed and the assessment endpoint.
- Physical/chemical relevance: correspondence between the chemical or physical agent tested and the chemical or physical agent constituting the stressor of concern.
- Environmental relevance: correspondence between test conditions and conditions in the environment ([EPA, 1998](#)).

EPA used this weight-of-evidence approach to assess hazard data and develop COCs. Given the available data, EPA only used studies assigned an overall quality level of high or medium to derive COCs for each taxonomic group. To calculate COCs, EPA derived geometric means for each trophic level that had comparable toxicity values (e.g., multiple EC₅₀s measuring the same or comparable effects from various species within a trophic level). EPA did not use non-definitive toxicity values (e.g., EC₅₀ > 48 mg/L) to derive geometric means because these concentrations of methylene chloride were not high enough to establish an effect on the test organism.

To assess aquatic toxicity from acute exposures, data for three taxonomic groups were available: amphibians, fish, and aquatic invertebrates. For each taxonomic group, adequate data were available to calculate geometric means as shown in Table 3-1. The geometric mean of the LC₅₀s for amphibians, 26.3 mg/L, represented the most sensitive toxicity value derived from each of the three taxonomic groups, and this value was used to derive an acute COC as described in Section 3.1.4. This value is from two studies that EPA assigned an overall quality of high and represents two species of amphibians. The geometric mean of EC₅₀s/LC₅₀s for aquatic invertebrates, 180 mg/L, was used to derive an acute COC to use as a surrogate species hazard value for sediment aquatic organisms. This geometric mean is from three studies that EPA assigned an overall quality level of medium and high and represents one aquatic invertebrate species.

To assess aquatic toxicity from chronic exposures, data for two taxonomic groups were described in the acceptable literature: fish, and aquatic invertebrates. Because the most sensitive taxonomic group from the acute data, amphibians, was not represented in the available chronic data, EPA considered the acute hazard geometric mean of the LC₁₀s for amphibians for teratogenicity leading to mortality to estimate chronic hazard values for amphibians. When comparing these values to the other chronic data from fish and aquatic invertebrates, amphibians were again the most sensitive taxonomic group. Therefore, the amphibian ChV of 0.9 mg/L was used to derive a chronic COC in Section 3.1.4. This value was from two studies that EPA assigned an overall quality level of high and represents two species of amphibians. For comparison, EPA calculated a ChV for fish of 1.5 mg/L for teratogenesis from a study that EPA assigned an overall quality level of high, representing one species.

To assess the toxicity of methylene chloride to algae, data for two species were available from studies that EPA assigned an overall quality level of high. EC₅₀s measuring biomass inhibition ranged from 33.1 mg/L to 242 mg/L, and an EC₁₀ of 115 mg/L was also reported. The exposure durations for the two tests differed by 24 hours, and the two algal species were functionally different, so EPA used the EC₅₀ for biomass inhibition from the more sensitive species to represent algae as a whole. This value, 33.1 mg/L, from one high quality algae study representing one species, was used to derive an algae COC in Section 3.1.4.

Based on the estimated bioconcentration factor and bioaccumulation potential described in Section 2.1, methylene chloride does not bioaccumulate in biological organisms. Therefore, EPA did not assess hazards to aquatic species from trophic transfer and bioconcentration or accumulation of methylene chloride.

3.1.4 Concentrations of Concern (COC)

EPA calculated the COCs for aquatic species based on the environmental hazard data for methylene chloride, using EPA methods ([EPA, 2013b](#), [2012b](#)). While there were data representing amphibians, fish, aquatic invertebrates, and aquatic plants, the data were not robust enough to conduct a more detailed species sensitivity distribution analysis. Therefore, EPA chose to establish COC as protective cut-off standards above which acute or chronic exposures to methylene chloride are expected to cause effects for each taxonomic group in the aquatic environment. The COC is typically based on the most sensitive species or the species with the lowest toxicity value reported in that environment. For methylene chloride, EPA derived an acute and a chronic COC for amphibians, which represent the most sensitive taxonomic group to methylene chloride exposure. Because other chronic toxicity data were relatively close to the amphibian data, EPA also calculated a chronic COC for fish, and a chronic COC for aquatic invertebrates for comparison. An algal COC was also calculated. Algae was assessed separately and not incorporated into acute or chronic COCs, because durations normally considered acute for other species (e.g., 48, 72 hrs) can encompass several generations of algae.

After weighing the scientific evidence and selecting the appropriate toxicity values from the integrated data to calculate acute, subchronic/chronic, and algal COCs, EPA applied an assessment factor (AF) according to EPA methods ([EPA, 2013b](#), [2012b](#)), when possible. The application of AFs provides a lower bound effect level that would likely encompass more sensitive species not specifically represented by the available experimental data. AFs can also account for differences in inter- and intra-species variability, as well as laboratory-to-field variability. These AFs are dependent on the availability of datasets that can be used to characterize relative sensitivities across multiple species within a given taxa or species group. However, they are often standardized in risk assessments conducted under TSCA, since the data available for most industrial chemicals are limited. For fish and aquatic invertebrates (e.g., daphnia) the acute COC values are divided by an AF of 5. EPA does not have a standardized AF for amphibians. For amphibians, there may be more uncertainty in the subchronic studies, necessitating a more protective AF of 10. For chronic COCs, an AF of 10 is used. The COC for the aquatic plant endpoint is determined based on the lowest value in the dataset and application of an AF of 10 ([EPA, 2013b](#), [2012b](#)).

After applying AFs, EPA converts COC units from mg/L to µg/L (or ppb) in order to more easily compare COCs to surface water concentrations during risk characterization.

Acute COC

To derive an acute COC for methylene chloride, EPA used the geometric mean of the LC₅₀s for amphibians, which is the most sensitive acute value for aquatic species from the data integrated for methylene chloride, from two studies EPA assigned overall quality levels of high ([Black et al., 1982](#); [Birge et al., 1980](#)). The geometric mean of 26.35 mg/L was divided by the AF of 10 for amphibians and multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The acute COC = (26.3 mg/L) / AF of 10 = 2.63 mg/L x 1,000 = 2,630 µg/L or ppb.

- The acute COC for methylene chloride is 2,630 ppb.

EPA used aquatic invertebrate hazard values as surrogate species to address hazards to sediment invertebrates. EPA derived an acute COC from the geometric mean of the EC₅₀s and LC₅₀s from two *Daphnia magna* studies that EPA assigned an overall quality level of high ([E I Dupont Denemours & Co Inc, 1987a](#); [Leblanc, 1980](#)), and one study that EPA gave an overall quality levels of medium ([Abernethy et al., 1986](#)). The geometric mean of 180 mg/L was divided by the AF of 5 and multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The acute aquatic invertebrate COC = (180 mg/L) / AF of 5 = 36 mg/L x 1,000 = 36,000 µg/L or ppb.

- The acute aquatic invertebrate COC for methylene chloride is 36,000 ppb.

Chronic COC

EPA derived the amphibian chronic COC from the lowest chronic toxicity value from the integrated data, the amphibian geometric mean of LC₁₀ for developmental effects and mortality in common frogs and American bullfrogs in two studies EPA assigned overall quality levels of high ([Black et al., 1982](#); [Birge et al., 1980](#)). The LC₁₀ was then divided by an assessment factor of 10, and then multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The chronic COC = (0.9 mg/L) / AF of 10 = 0.09 mg/L x 1,000 = 90 µg/L or ppb.

- The amphibian chronic COC for methylene chloride is 90 ppb.

EPA also derived a chronic COC for fish and aquatic invertebrates for comparison to the amphibian chronic data. The fish chronic COC was derived from the most sensitive chronic toxicity value from the integrated data, the ChV measuring teratogenesis in rainbow trout from a study that EPA assigned a quality level of high ([Black et al., 1982](#)). The ChV was then divided by an assessment factor of 10, and then multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The chronic COC = (1.5 mg/L) / AF of 10 = 0.15 mg/L x 1,000 = 150 µg/L or ppb.

- The fish chronic COC for methylene chloride is 150 ppb.

To derive a chronic COC for aquatic invertebrates, EPA used the toxicity value derived from the integrated acute toxicity data, the geometric mean of 180 mg/L, calculated from data on the freshwater invertebrate species, *Daphnia magna*. EPA applied the acute-to-chronic ratio of 10, resulting in a chronic aquatic invertebrate ChV of 18 mg/L. This ChV was then divided by an AF of 10 and multiplied by 1,000 to convert mg/L to µg/L, or ppb.

The chronic COC for aquatic invertebrates = (18 mg/L) / AF of 10 = 1.8 mg/L x 1,000 = 1,800 µg/L or ppb.

- The aquatic invertebrate chronic COC for methylene chloride is 1,800 ppb.

Algal COC

The algal COC was derived from the hazard value for the static algae *Pseudokirchneriella subcapitata* from one study that EPA assigned an overall quality level of high ([Tsai and Chen, 2007](#)). This algal species was selected as the more sensitive species from the available data to represent algal species as a whole. The 96-hour EC₅₀ for biomass inhibition of 33.1 mg/L was divided by an assessment factor of 10, and then multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The algal COC = (33.1 mg/L) / AF of 10 = 3.31 mg/L x 1000 = 3,310 µg/L or ppb.

- The algal COC is 3,310 ppb.

3.1.5 Summary of Environmental Hazard

EPA concludes that acute exposures to methylene chloride present hazards for amphibians, with toxicity values ranging from 23 mg/L to > 48 mg/L, integrated into a geometric mean of 26.3 mg/L from the definitive hazard values for two frog species (based on teratogenesis leading to lethality in embryos and larvae). Acute exposures to methylene chloride also present hazards for fish, with an immobilization hazard value of 99 mg/L in adult fish. Juvenile and adult fish mortality hazard values from acute exposures ranged from 108 to 502 mg/L, and EPA integrated these values into a geometric mean of 242.4 mg/L. For freshwater aquatic invertebrates, acute exposure hazard values for immobilization and mortality ranged from 135.8 mg/L to 177 mg/L, integrated into a geometric mean of 180 mg/L.

For chronic exposures, methylene chloride presents a hazard to amphibians, with toxicity values ranging from 0.8 to > 48 mg/L. The lowest chronic hazard values for amphibians, 0.8 mg/L and 1 mg/L, for teratogenesis and lethality in embryos and larvae of two frog species, integrated into a geometric mean of 0.9 mg/L. For chronic exposures, methylene chloride also presents a risk to fish, with hazard values ranging from 0.4 to 209 mg/L for teratogenesis, teratogenesis leading to

mortality, mortality, and growth inhibition. EPA assessed a NOEC and LOEC of 0.4 mg/L and 5.5 mg/L, respectively, for fish larvae mortality in one study, and integrated these hazard values into a geometric mean of 1.5 mg/L. There were no chronic duration hazard data for aquatic invertebrates, so EPA applied the acute-to-chronic ratio of 10 to the acute exposure aquatic invertebrate hazard value of 180 mg/L, resulting in a chronic exposure hazard value for aquatic invertebrates of 18 mg/L. For algae, hazard values for exposures to methylene chloride from two algal species were 33.1 mg/L and 242 mg/L. The hazard value for the more sensitive green algae species, 33.1 mg/L, is used to represent algal species as a whole.

Concentrations of Concern (COC):

The acute and chronic COCs derived for aquatic organisms are summarized in Table 3-2. EPA calculated the acute COC for methylene chloride exposures in amphibians as 2,630 ppb, based on the geometric mean of LC₅₀s for amphibians from two studies that EPA assigned an overall quality level of high ([Black et al., 1982](#); [Birge et al., 1980](#)). EPA also calculated an acute aquatic invertebrate COC of 36,000 ppb, to address sediment invertebrate hazards. EPA calculated the chronic COC for methylene chloride in amphibians as 90 ppb, based on the chronic toxicity value derived from the geometric mean of the LC₁₀.

For comparison with other trophic levels, EPA calculated a fish chronic COC of 151 ppb, based on a geometric mean of a NOEC and LOEC from a study measuring teratogenesis in rainbow trout that EPA assigned a quality level of high ([Black et al., 1982](#)). EPA also calculated an aquatic invertebrate chronic COC for methylene chloride of 1,800 ppb, based on the geometric mean of EC₅₀s and LC₅₀s from aquatic invertebrate studies that EPA assigned overall quality levels of medium and high. As noted previously, algal hazard values from exposures to methylene chloride, for durations ranging from 48 hrs to 96 hrs are considered separately from other aquatic species, because algae can cycle through several generations in this time frame. The algal COC of 3,310 ppb is based on the lowest EC₅₀ value for one study that EPA assigned overall quality levels of high.

The embryos and larvae of amphibians were the most sensitive organisms to acute exposures to methylene chloride, whereas adult fish and aquatic invertebrates had hazard values roughly an order of magnitude higher. For chronic exposures, the embryos and larvae of amphibians again had the most sensitive hazard values, followed closely by the embryos and juveniles of fish. Chronic hazard values for aquatic invertebrates and hazard values for algae were at least an order of magnitude higher than for the amphibian and fish embryos and larvae.

Table 3-2. COCs for Environmental Toxicity

Environmental Aquatic Toxicity	Hazard Value (µg/L)	Assessment Factor	COC (µg/L or ppb)
Toxicity to Amphibians from Acute Exposures	26,300	10	2,630
Toxicity to Aquatic Invertebrates from Acute Exposures	179,980	5	36,000
Toxicity to Amphibians from Chronic Exposures	900	10	90
Toxicity to Fish from Chronic Exposures	1,510	10	151
Toxicity to Aquatic Invertebrates from Chronic Exposures	18,000	10	1,800
Algal Toxicity	33,100	10	3,310

3.2 Human Health Hazards

3.2.1 Approach and Methodology

EPA used the approach described in Figure 3-1 to evaluate, extract and integrate methylene chloride's human health hazard and dose-response information. This approach is based on the *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)) and the *Framework for Human Health Risk Assessment to Inform Decision Making* ([EPA, 2014a](#)).

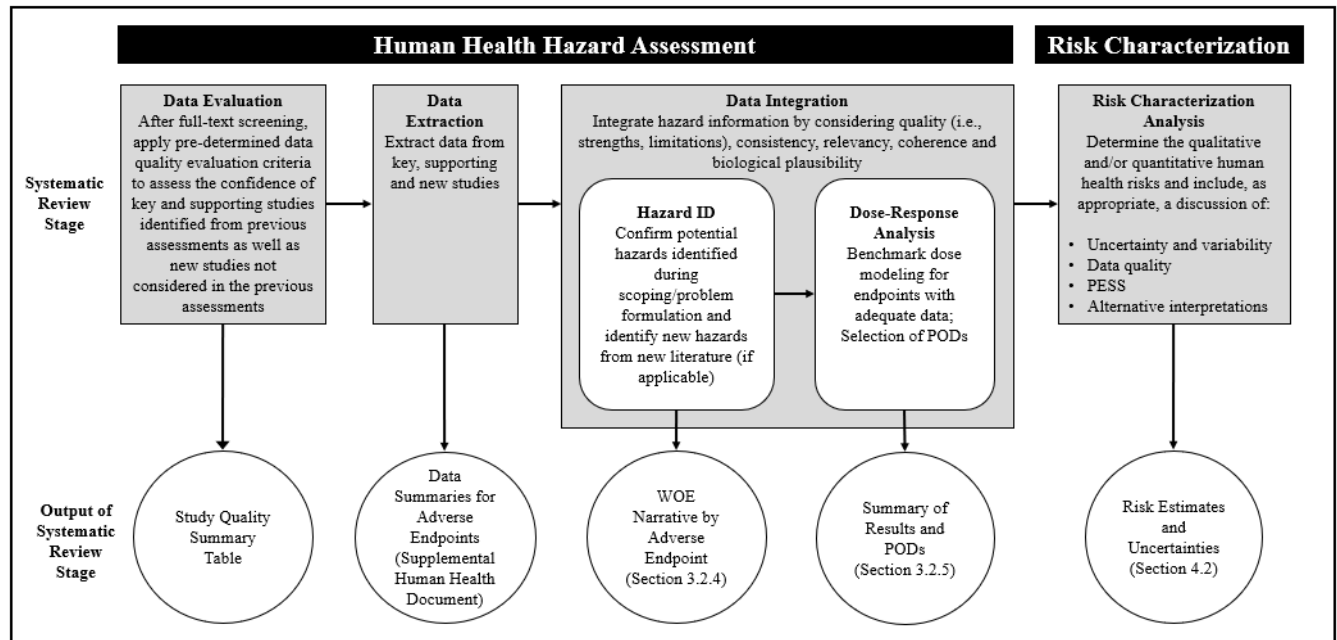


Figure 3-1. EPA Approach to Hazard Identification, Data Integration, and Dose-Response Analysis for Methylene Chloride

Specifically, EPA reviewed key and supporting information from previous hazard assessments as well as the existing body of knowledge on methylene chloride's human health hazards, which includes information published after these hazard assessments. The previous hazard assessments consulted by EPA include the following:

- *Spacecraft Maximum Allowable Concentrations (SMAC) for Selected Airborne Contaminants: Methylene chloride (Volume 2)* published by the U.S. National Academies ([Nrc, 1996](#));
- *OSHA Final Rules, Occupational Exposure to Methylene Chloride* by the Occupational Health and Safety Administration ([OSHA, 1997a](#));
- *Toxicological Profile for Methylene Chloride* by the Agency for Toxic Substances Disease Registry ([ATSDR, 2000](#));
- *Interim Acute Exposure Guideline Levels (AEGs) for Methylene Chloride* developed by the U.S. NAC on AEGs ([Nrc, 2008](#));
- *Acute Reference Exposure Level (REL) and Toxicity Summary for Methylene Chloride* published by the California Office of Environmental Health Hazard Assessment ([Oehha, 2008a](#));
- *Toxicological Review of Methylene Chloride* published in 2011 by EPA's IRIS ([U.S. EPA, 2011](#)); and
- *TSCA Work Plan Risk Assessment, Methylene Chloride: Paint Stripping Use* ([U.S. EPA, 2014](#)).

The health hazards of methylene chloride previously identified in these reviews were described and reviewed in this risk evaluation, including acute toxicity, neurotoxicity, liver toxicity, immunotoxicity, reproductive/ developmental toxicity, irritation/burns and genotoxicity/

carcinogenicity. EPA relied heavily on the aforementioned existing reviews along with scientific support from the Office of Research and Development (ORD) in preparing this risk evaluation. Development of the methylene chloride hazard and dose-response assessments considered EPA and NRC risk assessment guidance.

In addition to the primary literature cited in these previous assessments, EPA also conducted a search of newer literature to obtain information on all health domains. This process is outlined in Section 1.5. For human health hazard data, EPA obtained peer reviewed studies published from January 1, 2008 through March 2, 2017. EPA also obtained studies published after March 2017 that were identified by peer reviewers and public comments. Finally, EPA searched the gray literature, particularly studies submitted under certain sections of TSCA; some of these studies may have older dates (e.g., 1970s) but were still considered if they were not referenced in previous assessments.

The new literature was screened against inclusion criteria within the PECO statement. Relevant animal studies (i.e., potentially useful for dose-response) were further evaluated for data quality using criteria for animal studies described in *Application of Systematic Review in TSCA Risk Evaluations* ([U.S. EPA, 2018a](#)). Epidemiological studies were evaluated using *Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies* ([EPA, 2019a](#)). Because the key and supporting studies were considered in previous peer reviewed assessments to be studies useful and relevant for hazard identification, EPA skipped the screening step of the key and supporting studies and entered them directly into the data evaluation step based on their relevance to the risk evaluation.

For methylene chloride, the chosen key and supporting studies were initially identified as those used as the basis of acute values (California REL, SMAC, AEGLs and ATSDR minimum risk levels (MRLs)) and those from the IRIS assessment considered for the derivation of the inhalation reference concentration (RfC) and oral reference dose (RfD) as well as the suite of animal cancer bioassays that evaluated liver and lung tumors in addition to other tumor types that match those evaluated in recent epidemiology studies. In some cases, EPA expanded this list of studies reviewed to support the hazard assessment for a particular endpoint. For example, EPA evaluated the quality of all epidemiological studies that examined cancer endpoints to determine differences in quality and to understand patterns among the study results. Section 3.2.3 describes what was evaluated for data quality for each of the health domains.

EPA has not yet developed data quality criteria for all types of hazard information. For example, data quality criteria have not been developed for toxicokinetics and many types of mechanistic data that EPA typically uses for qualitative support when synthesizing evidence. Despite the lack of formal criteria, for methylene chloride, EPA qualitatively evaluated and summarized data (e.g., from human controlled experiments) if they were considered for the dose-response analysis or to determine their utility in supporting the risk evaluation.

Following the data quality evaluation, EPA extracted the toxicological information from each acceptable study into summary tables that include the endpoints considered for this assessment, the no-observed- or lowest-observed-adverse-effect levels (NOAEL and LOAEL) for non-cancer health endpoints by target organ/system, the incidence for cancer endpoints, and the overall data

quality evaluation ratings. The key/supporting studies and the newly identified studies found through searching recent literature are identified. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction of Human Health Hazard Studies* ([EPA, 2019o](#)) presents these tables.

Section 3.2.3 (Hazard Identification) discusses the body of studies for relevant health domains. EPA considered studies of low, medium or high confidence for hazard identification and focused on the following health domains considered relevant for methylene chloride: acute toxicity, neurotoxicity, liver toxicity, immunotoxicity, reproductive/ developmental toxicity, irritation and genotoxicity/carcinogenicity. Information from studies that were rated unacceptable were only discussed on a case-by-case basis for hazard identification and weight of scientific evidence assessment but were not considered for dose-response analysis. In some cases, additional studies not evaluated were also described within the hazard identification section as described in the health domain specific sections.

The weight of scientific evidence analysis (Section 3.1.3) included integrating information from toxicokinetic and toxicodynamic studies for the health domains described in Section 3.2.3. In particular, data integration considered consistency among the data, data quality, biological plausibility and relevance (although this was also considered during data screening). For each health domain, EPA determined whether the body of scientific evidence was adequate to consider the domain for dose-response modeling.

As presented in Section 3.2.5. (Dose-Response Assessment), data for the health domains with adequate evidence were modeled to determine the dose-response relationships (Appendix I and U.S. EPA ([2019h](#))¹¹). For the relevant health domains, EPA considered points of departure (POD) from studies that were PECO relevant, scored acceptable in the data quality evaluation and contained adequate dose-response information. For methylene chloride, studies used for dose-response modeling received high or medium quality ratings from the following health domains: acute toxicity (based on neurotoxicity), non-cancer liver toxicity and genotoxicity/carcinogenicity.

The POD is used as the starting point for subsequent dose-response (or concentration-response) extrapolations and analyses. PODs can be a NOAEL, a LOAEL for an observed incidence, or change in level of response, or the lower confidence limit on the benchmark dose (BMD)¹². The BMD analysis is discussed in Appendix I and the *Risk Evaluation for Methylene Chloride, Supplemental File – Methylene Chloride Benchmark Dose and PBPK Modeling Report* ([EPA, 2019h](#)). PODs were adjusted as appropriate to conform to the specific exposure scenarios evaluated (see Sections 3.2.5 and 4.3).

Inhalation acute human controlled experimental data and inhalation repeat-dose toxicity studies in animals were available for methylene chloride and were considered for dose-response assessment. No acceptable toxicological data are available by the dermal route. Furthermore, a

¹¹ *Risk Evaluation for Methylene Chloride – Methylene Chloride Benchmark Dose and PBPK Modeling Report* ([EPA, 2019h](#))

¹² The BMD is a dose or concentration that produces a predetermined change in response range or rate of an adverse effect (called the benchmark response or BMR) compared to baseline.

physiologically-based pharmacokinetic/pharmacodynamic (PBPK/PD) model that would facilitate route-to-route extrapolation to the dermal route has not been identified for methylene chloride. Therefore, inhalation PODs were extrapolated for use via the dermal route using models that incorporate volatilization, penetration, absorption and a permeability coefficient from an *in vitro* study in pig skin ([Schenk et al., 2018](#)) as described in both Section 2.4.2.3.1 and *Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment* ([EPA, 2019b](#)). EPA considered studies conducted via the inhalation route for this extrapolation for two primary reasons. First, these studies are already being used to calculate risks from inhalation in the current risk evaluation. Second, for cancer, the toxic moieties are metabolites of methylene chloride and both the inhalation and dermal routes are similar due to the fact that neither route includes a first pass through the liver (and subsequent metabolism) before entering the general circulation whereas first pass metabolism is important for the oral route. The PODs estimated based on effects in adult animals were converted to Human Equivalent Concentrations (HECs) for inhalation studies and Human Equivalent Doses (HEDs) when converting to the dermal route using species-specific PBPK models.

3.2.2 Toxicokinetics

Methylene chloride is quickly absorbed through inhalation exposure in humans and animals ([ATSDR, 2000](#)). Pulmonary uptake ranges between 40 and 60 percent ([Andersen et al., 1991](#); [Stewart et al., 1976](#); [Gamberale et al., 1975](#)), but may be up to 70 percent during the first minutes of exposure ([Riley et al., 1966](#)). In humans, uptake decreases as exposure duration and concentration increase ([Peterson, 1978](#); [Stewart et al., 1976](#)). A steady-state absorption rate is generally achieved within 2 hrs for exposures up to 200 ppm in humans ([Divincenzo and Kaplan, 1981](#); [Divincenzo et al., 1972](#)). One *in vitro* study ([Schenk et al., 2018](#)) using pig skin measured the dermal permeability of methylene chloride and estimated permeability coefficients of 8.66×10^{-3} cm/hr for the neat (100%) compound and 3.15×10^{-2} (1%) cm/hr for a 1% solution. Information from this study is used in the risk evaluation to estimate dermal absorption.

Methylene chloride is rapidly distributed throughout the body, including the liver, brain and subcutaneous adipose tissue, as identified in animal studies ([U.S. EPA, 2011](#); [ATSDR, 2000](#); [Carlsson and Hultengren, 1975](#)). Among fatality cases, the highest concentrations were usually found in the brain, then liver or kidneys and finally in the lungs and heart ([Nac/Aegl, 2008b](#)).

Metabolism occurs predominantly in the liver, with additional transformation in the lungs and kidneys ([ATSDR, 2000](#)). In the liver, two primary pathways are involved in the metabolism of methylene chloride. The cytochrome P450 (CYP450) mixed function oxidase (MFO) pathway (via CYP2E1) produces CO and CO₂, and saturation occurs at approximately 400-500 ppm after inhalation exposure in humans ([U.S. EPA, 2011](#)). The CO metabolite reacts with hemoglobin to form carboxyhemoglobin (COHb) ([ATSDR, 2000](#)).

The second pathway operates via glutathione S-transferase (GST); individuals with the theta 1 isozyme (GSTT1) metabolize methylene chloride to form formaldehyde and formic acid. In animals, saturation occurs at >10,000 ppm after inhalation exposure. Methylene chloride binds to the CYP reaction site with higher affinity than the GST site and COHb levels resulting from

methylene chloride's metabolism to CO can continue to increase and can reach peak levels 5 to 6 hours after exposure ([ATSDR, 2000](#)). Figure 3-2 outlines the biotransformation pathways for methylene chloride.

Major differences in affinity or other aspects of the CYP450 MFO pathway among species have not been identified ([Nac/Aegl, 2008b](#)). Studies generally indicate a 3- to 7-fold range in CYP2E1 activity among humans based on a variety of measures, with some research suggesting up to a 25-fold difference ([U.S. EPA, 2011](#)).

Comparing metabolism of methylene chloride by the GST pathway in liver and lung tissues among species, mice are more active than rats, humans and hamsters ([U.S. EPA, 2011](#)). Similarly, Thier et al. (1998) cited by U.S. EPA ([U.S. EPA, 2011](#)) found species' specific liver GSTT1 isozyme activity after methylene chloride exposure to be ordered as follows (from highest to lowest): mice, rats, human high conjugators, human low conjugators, hamsters and human non-conjugators. Thier et al. (1998) cited by U.S. EPA ([U.S. EPA, 2011](#)) also reported that high and low human conjugators exhibited GSTT1 activities in erythrocytes approximately 11 and 16 times higher than the human liver activities of high and low conjugators, respectively. Furthermore, the human high conjugator GSTT1 activity in erythrocytes was the same as male mouse liver activity and 61% of the female mouse liver activity. Among humans, the percent of GSTT1 +/+ individuals is 32%, whereas GSTT1 +/- individuals represent 48% and GSTT1 -/- individuals are 20% of the population ([Haber et al., 2002](#)).

The plasma half-life is estimated to be 40 minutes after inhalation exposure by human subjects ([ATSDR, 2000](#); [Divincenzo et al., 1972](#)). Unmetabolized methylene chloride is eliminated primarily through the lungs. Urine and feces also contain small quantities of unchanged methylene chloride ([ATSDR, 2000](#)). At low doses, a large percent of methylene chloride is transformed into COHb and eliminated as CO. At higher doses, more of the unchanged parent compound is exhaled ([ATSDR, 2000](#)).

Fetuses, infants and toddlers may be exposed to methylene chloride through breastfeeding and placental transfer. Methylene chloride has been detected in human breast milk (([Pellizzari et al., 1982](#); [Erickson et al., 1980](#)) and Vosovaja et al. (1974) as cited in [Jensen \(1983\)](#)). For example, mean concentrations of methylene chloride in breast milk for Soviet women workers who manufacture rubber articles were 74 ± 46 ppb in 17 of 28 samples (specimens with detectable levels) taken 5 ± 7 hours after the start of work, with levels declining after termination of work (Vosovaja et al. (1974) as cited in [Jensen \(1983\)](#)). Among babies born in 2015, the CDC 2018 breastfeeding report card found that the majority of newborns were breastfed. At 3 months, approximately half of old infants were exclusively ingesting breastmilk, and at 12 months, approximately a third were breastfed (<https://www.cdc.gov/media/releases/2018/p0820-breastfeeding-report-card.html>).

Methylene chloride can also cross the placental barrier and enter fetal circulation, with some research suggesting 2 to 2.5-fold lower concentrations in fetal blood, and other research identifying similar CO levels ([U.S. EPA, 2011](#)).

Blood concentrations of methylene chloride were lower than the detection level in 2,878 individuals who participated in the National Health and Nutrition Examination Survey (NHANES) based on subsamples of the U.S. population taken from the years 2009 and 2010 (CDC, 2019). Methylene chloride was found in the urine of workers employed at a pharmaceutical factory during a four-hour work-shift but was nearly eliminated during the overnight period following exposure (Hsdb, 2012).

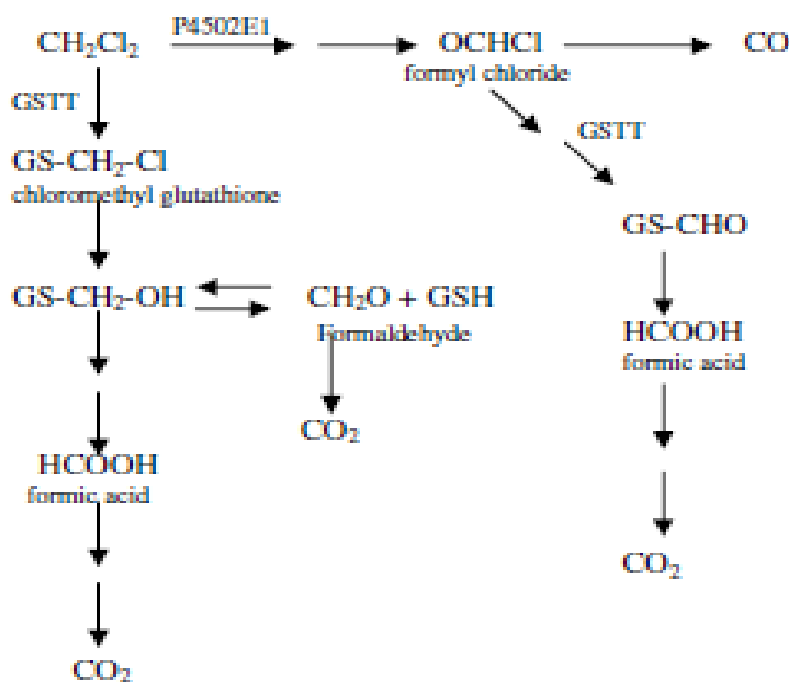


Figure 3-2. Biotransformation Scheme of Methylene Chloride (modified after Gargas et al., 1986).

Source: NAC/AEGL (2008b)

3.2.3 Hazard Identification

The methylene chloride database includes epidemiological studies, animal studies and *in vitro* studies. Epidemiological studies, animal studies and human experimental studies examined associations between methylene chloride exposure and multiple non-cancer effects and several types of cancer. Human controlled experiments also evaluated non-cancer effects from acute/short-term exposure. The following sections also describe several *in vitro* and some animal studies that evaluated biochemical and other endpoints used to consider the evidence related to modes of action.

EPA considered many of the studies as informative and useful for characterizing the health hazards associated with exposure to methylene chloride. EPA extracted the results of key and supporting studies from previous assessments and studies identified in the updated literature search into tables included in *Risk Evaluation for Methylene Chloride, Systematic Review*

Supplemental File: Data Extraction of Human Health Hazard Studies ([EPA, 2019o](#)). Several sections within Section 3.2.3 contain tables of data for given health domains.

Supplemental files contain data evaluations of these studies, including study strengths and limitations:

- *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Epidemiological Studies* ([EPA, 2019s](#));
- *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Human Controlled Experiments* ([EPA, 2019t](#)); and
- *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal and In Vitro Studies* ([EPA, 2019u](#))

The weight of scientific evidence section (3.1.3) identifies any study evaluation concerns that may have meaningfully influenced the reliability or interpretation of the results. Studies considered for dose-response assessment are discussed in Section 3.2.5.

3.2.3.1 Non-Cancer Hazards

EPA reviewed the scientific literature on non-cancer hazards of methylene chloride, based on systematic approaches described in Sections 1.5 and 3.2.4.1 and as presented in supplemental materials ([EPA, 2019s, t, u](#)). As a result of this review, EPA identified six adverse health effect domains: effects from acute/short-term exposure, liver effects, immune system effects, nervous system effects, reproductive/ developmental effects and irritation/burns. The following sections present data specific to each of these domains.

3.2.3.1.1 Toxicity from Acute/Short-Term Exposure

Neurotoxicity and neurological effects were the most frequently observed outcomes in the available acute and short-term studies. Furthermore, acute lethality in humans following inhalation relates to CNS depressant effects, which include loss of consciousness and respiratory depression resulting in irreversible coma, hypoxia and eventual death ([Nac/Aegl, 2008b](#)). Animal studies have also primarily identified CNS effects in acute exposure studies.

Although human and animal studies have identified other effects (including immunosuppression, liver effects, cardiac toxicity), the endpoints are observed either less often or at air concentrations higher than those associated with CNS effects.

For the current risk evaluation, EPA relied on the human controlled experiments and used a single study ([Putz et al., 1979](#)) that identified CNS effects. The following sections describe: 1) human acute controlled experimental studies and case reports of fatalities or high exposures; 2) acute exposure animal studies; and 3) the continuum of potential neurological effects, CNS depression, other severe effects including death.

Humans

Several of the acute human experimental studies resulting in CNS-related effects form the basis of acute exposure values such as the Spacecraft Maximum Allowable Concentration for Selected Airborne Contaminant (SMAC) ([Nrc, 1996](#)), Acute Exposure Guideline Levels (AEGs) 1 and 2¹³ ([Nac/Aegl, 2008b](#)) and the California Reference Exposure Level (REL) ([Oehha, 2008a](#)). EPA qualitatively reviewed these and other studies identified through backwards searching, drawing upon components developed for the formal human epidemiological and animal toxicity data quality criteria developed under TSCA. See *Risk Evaluation Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Human Controlled Experiments* ([EPA, 2019t](#)) for details regarding these reviews.

Table 3-3 outlines the studies that evaluated neurobehavioral effects.¹⁴ Putz et al. ([1979](#)) exposed 12 adults (males and females) to 195 ppm methylene chloride (measured) or 70 ppm CO for four hours; both exposures were designed to result in a COHb level of 5%. In a dual task, participants manipulated a lever to position a beam in the center of an oscilloscope (to measure eye-hand coordination) and also monitored peripheral stimuli visually for presence of an increase in light intensity of signal (to measure visual peripheral changes). Methylene chloride resulted in a decrease in visual peripheral performance of 7% at one and one-half hours and 17% at four hours and a 36% decrease in eye-hand coordination at four hours only. CO resulted in a 23% decrease in eye-hand coordination and an 11% decrease in visual performance at four hours. Both chemicals resulted in similar auditory decrements (~ 16-20%). The authors conclude that the tasks resulted in a decrease in speed and precision of psychomotor performance, which in turn, is hypothesized to indicate a temporary decrease in CNS activation. They also note that effects were observed usually only when the task was difficult or demanding ([Putz et al., 1979](#)). The study used a double-blind design but use of a single exposure concentration resulted in a medium data quality rating.

Stewart et al. ([1972](#)) evaluated three adult males and reported increased peak to peak amplitude visual evoked responses (VER) after a one-hour exposure to 514 ppm that returned to control levels soon after exposure ceased. COHb levels increased in these subjects as well. These types of VER changes have been observed to accompany initial phases of CNS depression ([Stewart et al., 1972](#)). [Stewart et al. \(1972\)](#) also reported symptoms of lightheadedness and difficulty enunciating words. Although the more objective measures from this study such as VER are of higher quality (with a medium data quality rating), EPA gave the symptom reports a low data quality rating because it is not known whether subjects and investigators were blinded to the subjects' exposure status.

¹³ The National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee) develops AEGs, which are applicable to emergency exposure periods ranging from 10 minutes to 8 hours. Three AEGs are established as air concentrations above which the general population (and susceptible subpopulations) could experience the following:

- **AEGL-1:** notable discomfort, irritation, or asymptomatic, non-sensory effects that are not disabling and are transient and reversible after exposure cessation;
- **AEGL-2:** irreversible or other serious, long-lasting adverse health effects or inability to escape; and
- **AEGL-3:** life-threatening health effects or death ([Nac/Aegl, 2008b](#)).

¹⁴ Several additional studies that linked methylene chloride exposure with COHb levels were also used in setting the SMAC.

Winneke (1974) reported effects similar to Putz et al. (1979). Eight to 18 adult females were exposed to 300, 500 or 800 ppm methylene chloride. Additional subjects were exposed to 50 or 100 ppm CO. At 800 ppm for four hours, methylene chloride resulted in decreases in all psychomotor performance measures except one, and a majority of the measures (10 of 14) were statistically significantly different from controls ($p < 0.05$ or < 0.01). Methylene chloride also resulted in decrements in a visual task (flicker fusion performance) at ≥ 300 ppm, with marked depression at 800 ppm ($p < 0.05$ or < 0.01). Auditory tasks also showed changes ($p < 0.05$) in several of the experiments, including at 300 ppm. However, visual and auditory effects were not consistent; for example, another experiment within this publication did not result in effects at 300 or 500 ppm. The authors concluded that this impaired performance was a sign of CNS-depression due to methylene chloride exposure. In contrast, no changes were observed after four hours of CO exposure (Winneke, 1974). Overall, EPA gave this study a medium data quality rating based on multiple exposure concentrations but use of a single blind method that was not well described.

Another study (Gamberale et al., 1975) used an inhalation method with 14 males that included a breathing valve that included menthol to disguise the odor of methylene chloride rather than a chamber to generate methylene chloride concentrations in air. Gamberale et al. (1975) did not identify significant decreases in tests of reaction time or a short-term memory test. These tests used a repeated-measure design (exposure to 250, 500, 750 or 1000 ppm methylene chloride consecutively for 30 minutes each, starting with the lowest exposure and successively moving to the highest with no breaks in exposure). Each test was administered within each of the 30-minute time periods. The subjects exhibited differences in perception of their own condition ($p < 0.005$); the authors noted this to be a subjectively favorable change. Heart rate was slightly lower with methylene chloride (not statistically significant). Other measures were not statistically significantly different from controls except for one of the simple reaction time tests during one exposure period. The authors provided very few details on the method of methylene chloride generation, and they did not measure methylene chloride levels in the breathing valve in inspiratory air. Thus, EPA gave the study a low data quality rating.

DiVincenzo et al. (1972) evaluated cerebral and motor functions of males exposed to 100 or 200 ppm methylene chloride for two or four hours. The authors evaluated the time it took to insert wooden pegs in a pegboard while simultaneously performing an arithmetic task. However, the authors provided only a brief statement that no changes were observed in the pegboard exercise or in subjective measures (also not defined). The authors did not report on results of the arithmetic task. Based on lack of information regarding results as well as whether negative controls were used, EPA gave this study a low data quality rating. Also, blinding was not mentioned, further resulting in low confidence regarding any subjective measures.

Kozena et al. (1990) examined sixteen healthy male volunteers exposed to methylene chloride for 1 hour using a double-blind experiment. Methylene chloride concentrations increased in geometrical steps (five minutes each except for the last exposure, which was 10 minutes) from zero to 720 ppm. The authors evaluated reactions to weak auditory stimuli and subjective feelings (including sleepiness, fatigue, mood changes) before, during and after exposure and found no differences from controls. Based on use of a half mask for exposure generation and

lack of understanding about comparability of the resulting exposure concentrations, EPA gave this study a low data quality rating.

Winneke and Fodor (1976) exposed females to methylene chloride in an exposure chamber conducted tasks that included adding numbers and letter cancelling (not further described), which were then interrupted to determine performance on critical flicker frequency (CFF). The authors report a methylene chloride-induced depression of CFF (p of 0.005). Winneke and Fodor (1976) also apparently describe experiments by Winneke (1974) that are already described above so those are not described here again. EPA gave this study a low data quality rating because details were limited regarding the outcome assessment methodology and the lack of reporting the results of the adding numbers component.

Other symptoms and effects have also been reported after acute methylene chloride exposures from case reports. For example, Preisser et al. (2011) reported nausea and irritation. Effects on lung, liver or kidney have also been reported in humans as primary signs of methylene chloride toxicity (Nac/Aegl, 2008b). In some cases, high COHb levels (i.e., up to 40 percent) are also observed (Nac/Aegl, 2008b).

Cardiotoxicity has been identified much less often or at higher concentrations. A few lethal cases exhibited cardiotoxic effects. One fatality was attributed to myocardial infarction without any signs of reported CNS depression, but other deaths due solely to cardiotoxic effects have not been reported (Nac/Aegl, 2008b). It is possible, however, that underlying heart disease may lead to dysrhythmia and contribute to the cause of death from methylene chloride (Macisaac et al., 2013). Some non-lethal case reports in humans have identified electrocardiogram [ECG] changes but at concentrations higher than those associated with CNS effects (U.S. EPA, 2011; ATSDR, 2000). Preisser et al. (2011) identified chest tightness (a possible cardiac sign). Increased COHb concentrations, however, have been associated with decreased time to angina in persons with cardiac disease while exercising (Nac/Aegl, 2008b). Based on this decreased time to angina, EPA considers individuals with cardiac disease to be an important susceptible subpopulation as further discussed in Sections and 4.4.5.

Animals

Neurological evaluations in animals during and after acute inhalation exposure to methylene chloride have resulted in CNS depressant effects that include decreased motor activity, impaired memory and changes in responses to sensory stimuli (U.S. EPA, 2011). Weinstein et al. (1972) and Heppel and Neal (1944) reported decreased spontaneous activity in rodents after exposure to 5000 ppm for up to seven or 10 days, respectively. Clinical signs along with decreased activity reported by Weinstein et al. (1972) suggested CNS depression. Kjellstrand et al. (1985) found that mice exhibited an initial increase in activity, and then decreased activity, after acute exposure \geq 600 to 2500 ppm. Rebert et al. (1989) identified visual and somatosensory responses in an acute study at concentrations up to 15,000 ppm that collectively suggested CNS depressive effects. Savolainen et al. (1981) identified increased preening by rats exposed to 500 ppm for six days, and Dow (1988) found changes observed on an electroencephalogram (EEG) and effects on somatosensory evoked responses after acute exposure by rats to \geq 2000 ppm methylene chloride.

[Shell Oil \(1986\)](#), submitted under TSCA, evaluated liver changes in mice and rats at 2000 and 4000 ppm after 1 and 10 days. Mice exhibited changes in liver weights (decreased at one day, increased at 10 days), but no changes in liver morphology. In contrast, all exposed rats had increased numbers of eosinophils in centrilobular cells and seven of 10 rats at the highest concentration exhibited increased incidence of mitotic figures in the midzone, adjacent to the area with eosinophilia. The overall data quality rating for this study is high.

After short-term exposure, Bornschein et al. ([1980](#)), reported increased general activity and delayed rates of habituation to a novel environment in rats exposed to 4500 ppm before (about 21 days) and/or during gestation (to day 17). Alexeeff and Kilgore ([1983](#)) identified a statistically significant difference in a passive avoidance learning task among three-day old mice exposed to ~47,000 ppm methylene chloride via inhalation compared with controls. In contrast, these authors did not observe any differences for 5- and 8-week old mice ([Alexeeff and Kilgore, 1983](#)).

Effects other than nervous system changes have also been reported in animals after acute exposure. CD-1 mice exhibited a localized immunosuppressive effect in the lung from inhalation of 100 ppm methylene chloride for three hours ([Aranyi et al., 1986](#)). After exposure to 2000 and 4000 ppm after one or 10 days of exposure, mice exhibited changes in liver weights, whereas rats exhibited increased numbers of eosinophils in centrilobular cells (both concentrations) and increased incidence of mitotic figures (highest concentration) ([Shell Oil, 1986](#)). Mice exhibited lung effects (on club cells) in this study at one day but not after 10 days ([Shell Oil, 1986](#)).

A few studies in animals have identified cardiac effects at higher concentrations. [Clark and Tinston \(1982\)](#) as cited in ([Nac/Aegl, 2008b](#)), first injected beagle dogs with adrenaline, exposed them to methylene chloride for 5 minutes and finally challenged them with another adrenaline injection. The EC₅₀ for cardiac sensitization to adrenaline was 25,000 ppm. Cardiac sensitization occurred upon ventricular tachycardia/ventricular fibrillation. Two other studies cited by NAC/AEGL ([2008b](#)) identified some additional cardiac effects but only after tracheal cannulation and at concentrations of 15,00 ppm and higher ([Aviado et al., 1977](#); [Oettingen et al., 1950](#)). As a result of these studies, NAC/AEGL ([2008b](#)) identified methylene chloride as arrhythmogenic.

Potential for Severe Effects

Given the potential for severe effects (including death) from the use of methylene chloride, EPA investigated the extent to which data are available to quantify the relationship between exposure and such effects. Overall, human studies, case reports and animal studies raise important questions regarding concentrations and exposure durations at which more severe effects occur.

In acute human experimental studies, nervous system effects ranged from nerve conduction changes to more severe motor impairment starting at a 1.5 hr inhalation exposure to 195 ppm to a 4-hr 800 ppm exposure (see Table 3 3). However, there is some uncertainty in the nature of the dose-response relationship. Both visual and auditory vigilance tests conducted by [Winneke \(1974\)](#) resulted in a similar or greater magnitude of effect at 300 ppm compared with 500 ppm.

Known or possible association between death from accidents with nervous system effects have been documented in an epidemiological study of methylene chloride and a supporting study on solvents. Lanes et al. (1990) found methylene chloride exposure to be associated with excess mortality from accidents at work (with 8-hr time-weighted averages (TWAs) ranging from below detection to 1700 ppm). Furthermore, Benignus et al. (2011) modeled increases in fatal car accidents from neurobehavioral changes resulting from small increases in solvent concentration.

Human fatalities have been documented in case studies where workers were using methylene chloride, with estimated air concentration ranges and exposure durations that appear to overlap with the human experimental studies that identified effects that were less severe. For example, one person was found dead 20 to 30 minutes after being seen alive; air samples taken after exposure were as low as 68-109 ppm at the level of the upper airways and 25,100 ppm at 25 cm above the solvent surface (Nac/Aegl, 2008b). Also, individuals have been found dead after an estimated 2 or 2.5 hrs of exposure with estimated air concentrations ranging from a 1-hr TWA in a bathroom of 637-1060 ppm (with a 1-hr TWA in the bathtub of ~11,600 to 19,400 ppm) up to 53,000 ppm in a squash court (NIOSH, 2011a; Nac/Aegl, 2008b). Information from these reports is limited and imprecise because air concentrations are measured after the individual died or are estimated based on amounts of methylene chloride used and room sizes and exposure durations are also estimated and may not be well known.

Lethality data in animals does suggest a steep dose-response curve, with an increase in mortality from 0 to 100% for an approximately twofold increase in exposure concentration (Nac/Aegl, 2008b). Appendix J presents additional details regarding fatalities associated with methylene chloride exposure.

Government and non-governmental organizations have established emergency guideline exposure levels for methylene chloride. The NIOSH guidance states that a value of 2300 ppm (7981 mg/m³) as immediately dangerous to life or health (IDLH) (NIOSH, 1994). Individuals should not be exposed to methylene chloride at this level for any length of time. The IDLH is based on acute inhalation toxicity data in humans. The AEGL-3 values for death range from 12,000 ppm (42,000 mg/m³) to 2100 ppm (7400 mg/m³) for 10-min to 8-hr time periods, respectively and are based on mortality from CNS effects in rats and COHb formation in humans (Nac/Aegl, 2008b).

Given the possibility that death or other severe effects may occur within the range of concentrations at which less severe effects occur, EPA considers Putz et al. (1979) to be the most relevant study to estimate risks of effects from acute exposure.

Sections on liver effects (Section 3.2.3.1.2), nervous system effects (Section 3.2.3.1.4) and immune system effects (Section 3.2.3.1.3) describe studies considered for modes of action for these endpoints.

Table 3-3. Human Controlled Inhalation Experiments Measuring Effects on the Nervous System*

Subjects	Concentrations	Duration	Endpoints (and timepoints) measured	COHb value	Effects observed	Reference	Qualitative data quality evaluation
6 males/6 females, 18-40 yrs, nonsmokers, good vision, no prior solvent exposure [subjects served as their own controls], Double blind design	(n = 12) 0, 195 ppm ^a (measured)	4 hrs = three 80-min blocks, 8-9 min rest btwn blocks	1) Dual task: Eye-hand coordination/ visual peripheral (4x, before/through exposure, ending at 4 hrs) 2) Auditory vigilance (3x, early during and through exposure period)	5.1% post-exposure	After 4 hrs: 1) 36%↓ hand/eye; 17%↓ visual peripheral (p < 0.01) 2) ~17% ^b ↓ auditory vigilance (p < 0.01) After 1.5 hrs: 1) 7% ↓ visual peripheral (p < 0.01)	Putz et al. (1979)	Medium; double-blinded, single concentration
11 males, 23-43 yrs, nonsmokers [pre-exposure values for each subject served as controls]	Experiment 2 ^e (n = 3): 986 ppm (measured)	2 hrs	1) Symptoms (1 hr pre-exposure; throughout exposure) 2) Visual evoked response (VER) (1x before, 2x during exposure and at 1 hr post-exposure) 3) Hematology/clinical chemistry/urinary urobilinogen (pre-exposure; up to 24 hrs post exposure)	10.1% @ 1 hr post-exposure; 3.9% @ 17hrs	1) Mild lightheadedness (2 subjects); difficult enunciation (1 subject) ^c 2) VER – Alterations in all 3 subjects ^d	Stewart et al. (1972)	Medium for VER; Low for symptoms due to lack of blinding
	Experiment 3 (n = 3): mean = 691 ppm; (514 ppm 1 st hr; 868 ppm 2 nd hr) vapor (measured)	2 hrs	1) Symptoms (1 hr pre-exposure; throughout exposure) 2) VER (1x before, 2x during exposure and ~ 1 hr post-exposure) 3) Hematology/clinical chemistry/urinary urobilinogen (pre-exposure; up to 24 hrs post exposure)	8.5% @ 2.5 hrs post-exposure ^b	1) Lightheadedness (1 subject; 2 nd hr) 2) VER – alterations (3 subjects) 3) No changes		
	Experiment 4: (n = 8): 515 ppm	1 hr	1) Symptoms (1 hr pre-exposure; throughout exposure) 2) Hematology/clinical chemistry (<i>presumably</i> pre-exposure; up to 24 hrs post exposure)	3.4% @ 1 hr post-exposure	1) None identified 2) No ↑ in RBC (red blood cell) destruction		
Females [unclear whether subjects served as their own controls],	Experiment 1 ^{g, h} (n = 8): 0, 500 ppm	3.8 hrs	1) Auditory vigilance (4x during exposure) 2) Visual critical flicker fusion (CFF)		1) Auditory: omission errors (p < 0.05) 2) Visual CFF: Not stat. sig (ANOVA ¹ for both)	Winneke, (1974)	Medium; single blinded

Subjects	Concentrations	Duration	Endpoints (and timepoints) measured	COHb value	Effects observed	Reference	Qualitative data quality evaluation
authors conclude that the study was single-blinded based on lack of odor (expect at 800 ppm)	Experiment 2 (n = 6): 0, 300, 800 ppm	3.8 hrs	1) Auditory vigilance (4x during exposure) 2) Visual CFF (1x before; 4x during exposure)		1) Auditory: omission errors (p < 0.05) 2) Visual CFF (p < 0.05) (ANOVA for both)		
	Experiment 3 (n = 6): 0, 300, 500 ppm	3.8 hrs	1) Auditory vigilance (4x during exposure) 2) Visual CFF (1x before; 4x during exposure)		1) Auditory: not stat. sig. 2) Visual CFF: not stat. sig. (ANOVA for both)		
	Experiment 2 + 3 (n = 12): 0, 300 ppm	3.8 hrs	1) Auditory vigilance (4x during exposure) 2) Visual CFF (1x before; 4x during exposure)		1) Auditory: omission errors (p < 0.05) 2) Visual CFF (p < 0.01) (ANOVA for both)		
	Experiment 4 ^a (n = 18): 0, 800 ppm	4 hrs	1) Auditory vigilance (2x during exposure) 2) Visual CFF (1x before; 3x during exposure) 2) Comprehensive battery of 14 psychomotor tests ^f (near end of exposure)		1) Auditory: reaction time (p < 0.05; ANOVA) 2) Visual CFF: not stat. sig. 3) 10 tests ↓ (5 @ p < 0.01; 5 @ p < 0.05); Steadiness (1 test), Hand precision (2 right hand tests), pursuit tracking (single test) not stat. sig. (paired t-values)		
Males, 20-30 yrs, identified as healthy	(n = 14) 0, 250, 500, 750, 1000 ppm	2 hrs (30 min each to increasing concentration without a break in exposure)	1) Subjective perceptions 2) Reaction time (RT) – addition 3) Simple reaction test 1 4) Short-term memory 5) Simple reaction test (Each test conducted during each exposure concentration and for controls)	~5%	1) Perceptions - individual measures not statistically significant; as a whole, changes were observed (p < 0.005), although authors described this as subjectively positive 3) Simple RT 1 – changes only at the highest concentration (p < 0.05) 2, 4 and 5) RT addition, Short-term memory, simple RT 2 – no stat. sig. changes	Gamberale et al. (1975)	Low – use of breathing valve with limited details and no analytical monitoring; Impact of using menthol not known
Males, 28 to 60 yrs, inclusion required medical approval	100, 200 ppm (n = 11)	2 and 4 hrs	1) Pegboard activity – time required to place pegs in proper holes (for 2 hr: at beginning, 1 hr and 1hr/40 min; for 4 hr: added time at 2 and 3 hrs; 5 trials at each timepoint), 2) Subjective measures (continuous surveillance)		1) No changes (details not provided) 2) No changes (details not provided)	DiVincenzo et al. (1972)	Low – lack of detail regarding results and use of controls

Subjects	Concentrations	Duration	Endpoints (and timepoints) measured	COHb value	Effects observed	Reference	Qualitative data quality evaluation
Males, 19-21 yrs, healthy, paid volunteers, double-blind design	0 (n = 42) Increasing conc to approximate 144 ppm (w/peak of 720 ppm at end of exposure) (n = 16)	1 hr	1) weak auditory stimuli (5 to 25 sec during 1 hr, repeated 3x – before, during and after exposure) 2) Subjective measures (sleepiness, fatigue, changes in mood)	NA	1) No changes 2) No changes	Kozena et al. (1990)	Low – lack of information on exposures
Females, 22-31 yrs, single-blind design not well described [subjects served as their own controls]	0, 500 ppm (n = 12, groups of 3)	2 hrs 20 min	1) alternating task of adding numbers and letter cancelling 2) Visual CFF (4 x during exposure)	NA	1) No changes 2) Visual CFF (p of 0.005)	Winneke and Fodor (1976)	Low – limited details on outcome method and results

*Hematology measured in one study

^a CO also evaluated but not included in table

^b Estimated from graph

^c Individuals were inadvertently exposed to methylene chloride before exposure, resulting in breath levels of 10 ppm and higher (graph is exponential and difficult to read above 10); this didn't appreciably alter COHb levels.

^d Information on statistical significance not presented.

^e Experiment 1 measured COHb in one individual after 213 ppm vapor exposure for 1 hour; a value of 2.4% @ 3 hrs post-exposure was observed

^f Tapping (hand movements without eye-hand coordination- 1 test); two plate tapping (arm movements: some eye-hand coordination – 1 test); steadiness (hand/arm - 2 tests); hand precision (6 total tests – 3 for each hand); pursuit tracking (visual-motor control of large muscle groups – 1 test); reaction speed (visual/gross motor reaction – 3 tests)

^g There was an experiment 0 (pilot study) – 0, 500 ppm (n = 12) – results of visual CFF show a decrement (p < 0.01); auditory vigilance and other un-named tasks were not s.s.

^h The authors state that the measured values are 317 ppm, 470 ppm and 751 ppm; those values are not included in the table because it is not clear whether they represent averages across experiments or are specific to one of the experiments.

ⁱ ANOVA = analysis of variance

3.2.3.1.2 Liver Effects

A limited number of human studies and multiple animal studies have identified liver effects associated with methylene chloride exposure. EPA focused on evaluating human epidemiological studies as well as chronic inhalation studies in animals. Other animal studies discussed in previous peer-reviewed assessments are considered acceptable for supporting the weight of scientific evidence.

Humans

Few epidemiological studies evaluated non-cancer liver effects, and limited evidence was identified in studies that measured relevant endpoints. Three acceptable epidemiological studies measured bilirubin and serum enzyme concentrations in workers exposed to methylene chloride ([Soden, 1993](#); [General Electric Co, 1990](#); [Ott et al., 1983b](#)).¹⁵ Two of these studies found some evidence of increasing levels of serum bilirubin with increasing exposure but no consistent trends for other serum hepatic enzyme levels (γ -glutamyl transferase, aspartate amino transferase (AST) and alanine transaminase (ALT)) ([General Electric Co, 1990](#); [Ott et al., 1983b](#)). EPA gave medium data quality ratings to all three studies. Although increased bilirubin is of concern, EPA did not consider this to be an endpoint appropriate for considering in the current risk evaluation because these data don't provide clear evidence of adverse liver effects.

In the updated literature search, EPA identified only one additional study that evaluated any liver effects. Silver et al. ([2014](#)) reported no increase in standardized mortality ratios (SMR) for cirrhosis and other chronic liver diseases in a cohort of microelectronics and business machine workers exposed to multiple solvents, metals, glycol ethers and other chemicals. Individuals were exposed for an average of 5.2 to 9.8 yrs. depending on sex and whether they were salaried or hourly from 1969 to 2001 when compared with death rates in the U.S. population. There was some exposure to methylene chloride, but the SMRs were not specific for methylene chloride exposure. Silver et al. ([2014](#)) received a medium data quality rating.

Overall, the human data are not conclusive with respect to methylene chloride's association with liver effects based on the limited database and endpoints evaluated.

Animals

Section 3.2.3.1.2 outlines liver effects in chronic and subchronic studies. Section 2.2.3.1.1 describes shorter-term and acute exposure studies. In chronic inhalation studies in animals, liver effects were often the most sensitive effects. Rats exhibited vacuolization and sometimes necrosis ([Nitschke et al., 1988a](#); [NTP, 1986](#); [Burek et al., 1984](#)), hemosiderosis ([NTP, 1986](#)) and acidophilic and basophilic foci ([Aiso et al., 2014a](#)). Mice showed degenerative changes in hepatocytes in one chronic inhalation study ([NTP, 1986](#)). No liver effects were observed in hamsters after chronic inhalation ([Burek et al., 1984](#)). U.S. EPA ([2011](#)) notes that vacuolization was consistently identified, and lipids were observed in the vacuoles. Data quality ratings for the chronic studies are high.

In the updated literature search, Aiso et al. ([2014a](#)), a chronic inhalation study, found that relative liver weights of rats were decreased > 10% only at the lowest concentration (1000 ppm) in males ($p < 0.01$). In females, absolute and relative liver weights were increased by 11%, 25% and 25% and by 11%, 22% and 29% at 1000, 2000 and 4000 ppm, respectively ($p < 0.01$). In males, acidophilic and basophilic cell foci were increased at 1000 or 2000 ppm without a dose response. In females, lesions were increased

¹⁵ GE ([1990](#)) is the same reference as [Kolodner et al. \(1990\)](#), which is cited in U.S. EPA ([2011](#)).

and showed more of a dose-response, although Aiso et al. (2014a) did not report results of trend tests. The authors classified the altered acidophilic and basophilic cell foci as preneoplastic proliferative lesions. However, EPA did not observe correlations between the pre-neoplastic foci and tumors in this study. For example, these foci were not significantly increased in mice, even though the incidences of hepatocellular adenomas and carcinomas were significantly increased in a dose-response trend. Also, these foci were also not well correlated in rats. Therefore, EPA considers the foci identified in this study to be non-neoplastic and rats appear to be more sensitive to the effect.

In subchronic inhalation studies, rats and dogs exhibited fatty livers, mice exhibited hepatic degeneration and vacuolization and monkeys exhibited borderline effects (NTP, 1986; Haun et al., 1972; Haun et al., 1971). However, a 90-day study by Leuschner et al. (1984) found no changes in liver weights, related biochemistry or histopathology in Sprague-Dawley rats or Beagle dogs at concentrations as high or higher than other studies that showed effects. The reason for this negative study is not clear but Leuschner et al. (1984) did not identify the organs evaluated histologically and identified results of biochemical and other analyses in the text only as “no intolerance phenomena” without any tabular information presented. EPA identified a 90-day oral dog study submitted under TSCA that was not reported in U.S. EPA (2011). Four dogs at the highest dose of 200 mg/kg-bw/day exhibited inflammatory cell foci in livers compared with one control animal with the effect (General Electric Co, 1976b). Foci were slight or very slight in severity and not accompanied by biochemical changes. This study received a high overall data quality rating.

Mechanistic Data

Although U.S. EPA (2011) discussed modes of action related to liver tumors, limited research has focused on the mechanisms related to non-cancer liver effects. When U.S. EPA (2011) investigated metrics for dose-response modeling, considering the metabolites of the CYP pathway showed more consistency between the inhalation and oral routes compared with results of the GST pathway or considering AUC of the parent compound. Although not definitive, this could suggest metabolites of the CYP pathway may be involved in non-cancer liver endpoints. U.S. EPA (2011) indicated exposure of Wistar rats to 500 ppm resulted in increased hemochrome content in liver microsomal cytochrome P450 (CYP) (Savolainen et al., 1977), which could represent an adaptive response. Also, mouse hepatocyte degeneration was related to dissociated polyribosomes and rough endoplasmic reticulum swelling (Weinstein et al., 1972).

In the updated literature search, EPA identified a few studies that examined changes in gene and protein expression and enzymatic activities in livers of rats or in one case, fish. Oral studies in rats and one study in fish identified liver-related biochemical changes but none provide definitive or specific information on modes of action for methylene chloride related to non-cancer liver toxicity. In rats, methylene chloride was associated with increased biliary output after induction of nitric oxide (NO) by carbon monoxide (CO), which increased biliary excretion of glutathione (GSH) (Chen et al., 2013). Kim et al. (2010) found expression of the protein α -2 μ globulin was decreased (0.92 vs. 1), whereas GST- α (1.13 vs. 1) and phenylalanine hydroxylase (1.17 vs. 1) were increased in livers of rats orally exposed to methylene chloride. Likewise, seven of 1,100 proteins (three paralogues of GST, β -1-globin - part of hemoglobin that binds CO₂, two hemoglobin β -2 subunits and α -2 globulin) in livers of rats dosed orally with methylene chloride were downregulated compared with controls (Park and Lee, 2014). In rat livers, methylene chloride also downregulated genes that are downregulated in T-cell prolymphocytic leukemia (Kim et al., 2013). Dzul-Caamal et al. (2013) didn't identify increased formaldehyde or reactive oxygen species (ROS) as H₂O₂ in livers of fish but identified increasing lipid peroxidation and oxidation of proteins with increasing doses of methylene chloride.

Table 3-4. Liver Effects Identified in Chronic and Subchronic Animal Toxicity Studies of Methylene Chloride

Target Organ/System	Study Type	Species/Strain/Sex (Number/group)	Exposure Route	Doses/Concentrations	Duration	NOAEL/LOAEL reported by study authors	NOAEL/LOAEL (mg/m ³ or mg/kg-day) (Sex)	Effect	Reference	Data Quality Evaluation
Hepatic	Chronic	Rat, F344, M/F (n=100/group)	Inhalation, vapor, whole body	0, 3510, 7019 or 14,038 mg/m ³ (0, 1000, 2000 or 4000 ppm)	6 hours/day, 5 days/week for 2 years	NA	LOAEL= 3510 (M/F)	Hepatocyte vacuolation and necrosis, hemosiderosis in liver (M/F); hepatocyte-megaly (F)	NTP (1986)	High
Hepatic	Chronic	Rat, Sprague-Dawley, M/F (n~190/group)	Inhalation, vapor, whole body	0, 1755, 5264 or 12,283 mg/m ³ (0, 500, 1500 or 3500 ppm)	6 hours/day, 5 days/week for 2 years	NA	LOAEL= 1755 (M/F)	Hepatocyte vacuolation (M/F); multinucleated hepatocytes (F)	Burek et al. (1984)	High
Hepatic	Chronic	Rat, Sprague Dawley, M/F (n=180/group)	Inhalation, vapor, whole body	0, 176, 702 or 1755 mg/m ³ (0, 50, 200 or 500 ppm)	6 hours/day, 5 days/week for 2 years	NA	NOAEL= 702 (F)	Hepatic lipid vacuolation and multinucleated hepatocytes	Nitschke et al. (1988a)	High
Hepatic	Chronic	Mouse, B6C3F1, M/F (n=100/group)	Inhalation, vapor, whole body	0, 7019 or 14,038 mg/m ³ (0, 2000 or 4000 ppm)	6 hours/day, 5 days/week for 2 years	NA	LOAEL = 7019 (F)	Hepatocyte degeneration; (↑ hepatocellular adenoma or carcinoma)	NTP (1986)	High
Hepatic	Chronic	Mouse, B6C3F1, M/F (n=20/group)	Inhalation, vapor, whole body	0, 1843, 3685, 7371, 14,742 or 29,483 mg/m ³ (0, 525, 1050, 2100, 4200 or 8400 ppm)	6 hours/day, 5 days/week for 13 weeks	NA	NOAEL= 7371 (F); NOAEL = 14,742 (M)	Hepatocyte centrilobular degeneration	NTP (1986)	High

Target Organ/System	Study Type	Species/Strain/Sex (Number/group)	Exposure Route	Doses/Concentrations	Duration	NOAEL/LOAEL reported by study authors	NOAEL/LOAEL (mg/m ³ or mg/kg-day) (Sex)	Effect	Reference	Data Quality Evaluation
Hepatic	Chronic	Rat, F344, M/F (n=170/group + 270 controls)	Oral, drinking water	0, 6, 52, 125 or 235 mg/kg-day (M); 0, 6, 58, 136 or 263 mg/kg-day (F)	104 weeks	NA	NOAEL= 6 (M/F)	↑ Non-neoplastic Foci/areas of alteration (M/F); ↑ incidence of neoplastic nodules; fatty liver changes (incidence N/A)	Serota et al. (1986a)	High
Hepatic	Subchronic	Rat, F344, M/F (n=30/group)	Oral, drinking water	0, 166, 420 or 1200 mg/kg-day (M); 0, 209, 607 or 1469 mg/kg-day (F)	90 days	NA	LOAEL= 166 (M); LOAEL = 209 (F)	Hepatic vacuolation (generalized, centrilobular, or periportal)	Kirschman et al. (1986)	Low
Hepatic	Chronic	Mouse, B6C3F1, M/F (n=125, 200, 100, 100 and 125 [M]; n=100, 100, 50, 50 and 50 [F])	Oral, drinking water	0, 61, 124, 177 or 234 mg/kg-day (M); 0, 59, 118, 172 or 238 mg/kg-day (F)	104 weeks	NA	NOAEL= 185 (M/F)	Some evidence of fatty liver; marginal increase in the Oil Red-O-positive material in the liver	Hazleton Labs (1983)	Medium
Hepatic	Subchronic	Mouse, B6C3F1, M/F (n=30/group)	Oral, drinking water	0, 226, 587 or 1911 mg/kg-day (M); 0, 231, 586 or 2030 mg/kg-day (F)	90 days	NA	NOAEL= 226 (M)	Hepatic vacuolation (increased severity of centrilobular fatty change)	Kirschman et al. (1986)	Low
Hepatic	Chronic	Rat, F344/DuCrj	Inhalation, vapor, whole body	0, 3510, 7019 or 14,038 mg/m ³ (0, 1000, 2000 or 4000 ppm)	6 hours/day, 5 days/week for 2 years	NA	LOAEL = 3510 mg/m ³ (F)	Increased basophilic foci and increased abs/rel liver wt (p < 0.01)	Aiso et al. (2014a)	High

Target Organ/System	Study Type	Species/Strain/Sex (Number/group)	Exposure Route	Doses/Concentrations	Duration	NOAEL/LOAEL reported by study authors	NOAEL/LOAEL (mg/m ³ or mg/kg-day) (Sex)	Effect	Reference	Data Quality Evaluation
Hepatic	Subchronic	Dog/Beagle (M/F) (4/sex/ group)	Oral	0, 12.5, 50, 200 mg/kg-bw/day	90 days	Not Reported	NOAEL = 200 mg/kg-bw/day	No changes in clinical chemistry, gross pathology, organ weight, or histopathological lesions	General Electric Co (1976b)	High

3.2.3.1.3 Immune System Effects

EPA identified a limited number of human, animal and mechanistic studies of immune system effects. Some studies identified effects associated with methylene chloride but results are limited and conflicting.

Humans

From the updated literature search, EPA identified one epidemiological study that addressed an immune-related endpoint. Chaigne et al. (2015) is a case control study evaluating Sjogren's syndrome, which is an autoimmune epithelitis characterized by dry eyes and mouth, physical weakness and joint pain. Systemic symptoms are possible and individuals with this syndrome have an increased risk of lymphoma. The study identified 175 cases at three university hospitals in France and used a comparison group of healthy individuals from the same hospitals. The authors assessed exposure using a published job exposure matrix that accounted for probability, intensity, frequency and duration of exposure. The study authors did not adjust for confounding but did match cases and controls for age and gender. Cases and controls had similar smoking rates and socio-economic and socio-professional levels.

Exposure to methylene chloride was associated with Sjogren's syndrome based on an odds ratio (OR) of 9.28 (95% confidence interval (CI): 2.60-33.0) ($p < 0.0001$) (13 cases vs. 3 controls). Among patients with anti-SSA or anti-SSB antibodies¹⁶, the OR was 11.1 (95% CI: 2.38-51.8) ($p < 0.001$). For these two measures, methylene chloride had the highest ORs compared with other compounds. High cumulative exposure (exposure score > 1) to methylene chloride was not statistically significantly associated with Sjogren's syndrome, although the association was still greater than 1.0 (OR: 3.04; 95% CI: 0.50 – 18.3) (Chaigne et al., 2015). EPA determined an overall data quality rating of medium for Chaigne et al. (2015) due to lack of information on recruitment, participation and exposures.

Among U.S. Air Force base workers, men exhibited an increased risk of bronchitis-related mortality when exposed to methylene chloride (hazard ratio (HR): 9.21; 95% CI: 1.03–82.69) (Radican et al., 2008). The HR is based on a total of four exposed cases and comparison of exposed and unexposed male workers. There could be multiple causes of the bronchitis (e.g., infection or other inflammatory processes). The authors used employment for at least one year as the exposure criteria, and exposure levels were not estimated but methylene chloride use was linked to specific departments at the air base (Radican et al., 2008). The model adjusted for age, race and gender, and evaluated 5-calendar year ranges but didn't adjust for socioeconomic status, which was quite different between exposed and control workers (i.e., salaried workers were $< 1\%$ and 61% among cases and controls, respectively). The study also did not adjust for co-exposures, even though 21 additional solvents and chemicals were evaluated separately. The study received a medium data quality rating. Lack of information on cause of bronchitis, exposure, the limited

¹⁶ SSA and SSB refer to Ro and La, respectively. These are ribonucleoprotein complexes (not compounds foreign to the body) and anti-SSA and anti-SSB are antibodies mounted in response to these complexes (Moutsopoulos and Zerva, 1990).

numbers of cases and the lack of adjustment for other chemical co-exposures makes it difficult to make strong conclusions regarding the association between methylene chloride and bronchitis.

[hoechst celanese corp \(1992\)](#) evaluated deaths from multiple causes in workers at a CTA fiber production work site in Maryland, as identified on death certificates, for workers employed from 1970 to 1989. Slight elevations in risk of mortality due to influenza and pneumonia were observed (SMR - males: 1.25; females: 4.36) when comparing workers ever exposed to the highest exposure group (> 350 ppm - ~ 700 ppm) to the Maryland county population in which the plant was located. The authors reported no statistically significant excesses of deaths but did not report the 95th % confidence intervals for the SMR. Workers in this highest group could have had portions of their work history exposed to lower (or no) concentrations. Employees may have also been exposed to ethers, halogenated hydrocarbons, hydrazines, inorganic dusts and many other compounds). EPA gave this study a data quality rating of medium. Because the comparison group included the working and non-working population, any effects of methylene chloride may have been attenuated based on greater illness in the controls unrelated to methylene chloride exposure, and some effects might have been associated with other chemical exposures that were not accounted for in the models. For these reasons, firm conclusions regarding the association with methylene chloride cannot be made from this study.

Hearne and Pifer ([1999](#)), in Part I of their study, found significantly lower than expected numbers of deaths due to infectious and parasitic diseases among triacetate film production workers compared with death rates/causes of individuals in the general population in New York (excluding New York City) in a 1946-70 cohort (employed in multiple divisions) followed through 1994 (SMR = 0; 95% CI: 0-66; $p \leq 0.05$). Although the study did not control for other chemical exposures, the analysis was limited to employees hired after methylene chloride became the principal solvent. (The authors do note that a 80% methylene chloride/20% methanol mixture was used in one of the divisions.) Employees worked for at least one year in one or more of the divisions. Exposure was calculated by multiplying methylene chloride air concentrations by the number of years exposure. For all diseases of the respiratory system, the SMR was 90 (95% CI: 58-134)¹⁷ (also compared with the New York state population). Similar to the previous study ([hoechst celanese corp, 1992](#)), the comparison populations of Hearne and Pifer ([1999](#)) included working and non-working individuals and thus could include individuals who may be not working due to illness.

Hearne and Pifer ([1999](#)) also conducted an analysis of employees in the roll coating department (Part II); about 30% were hired before methylene chloride was introduced. Similar to Part I, workers were employed for at least 1 year. The SMR for infectious and parasitic diseases was 67 (95% CI: 14-197)¹⁸ using unexposed Kodak Rochester employees as the comparison. The study's strength included its use of air monitoring values (> 1500 area samples and > 2500 personal monitoring samples for the Part I analysis). This study was rated high for data quality. The authors note that for Part I, regression modeling was adjusted for age, calendar year and time from first exposure, but it is not clear whether this was also done for the Part II analysis.

¹⁷ Using a similar metric as other studies, the SMR would be 0.90 (95% CI: 0.58-1.34).

¹⁸ Using a similar metric as other studies, the SMR would be 0.67 (95% CI: 0.14-1.97).

Lanes et al. (1993) assessed mortality among employees at a CTA fiber manufacturing plant in Rock Hill, South Carolina. Workers were employed for at least three months in jobs that entailed exposure to the highest concentrations of methylene chloride (median exposures of 140 to 745 ppm as 8-hr time-weighted averages). Methanol and acetone were also present but Lanes et al. (1993) didn't control specifically for these compounds. The analysis did control for age, race, gender and calendar period. The authors did not identify an increased risk of death from nonmalignant respiratory disease (SMR = 0.97; 95% CI: 0.42-1.90). The comparison death rates were taken from York County, South Carolina and could mask effects from methylene chloride if the illness rates unrelated to methylene chloride differed between workers and the county population. This study received a data quality rating of medium.

Animals

EPA identified no new animal studies that addressed immunomodulation in the updated literature search. U.S. EPA (2011) summarized two animal toxicity studies. Aranyi et al. (1986) evaluated several measures of immune response in acute inhalation studies using female CD-1 mice. Mice were challenged with live aerosolized *Streptococcus zooepidemicus* while simultaneously being exposed to methylene chloride vapor or filtered air. The authors recorded deaths over a 14-day period. Similarly, the authors measured clearance of aerosolized *Klebsiella pneumoniae* by pulmonary macrophages from CD-1 mouse lungs 3 hours after infection, comparing methylene chloride to air exposures. After a single 3-hour exposure to 95 ppm methylene chloride, deaths were increased by 12.2% ($p \leq 0.01$) from *S. zooepidemicus* infection compared with controls. Bactericidal activity of macrophages against *K. pneumoniae* was decreased by 12% ($p \leq 0.001$). In contrast, no changes in mortality rates or bactericidal activity were observed with either single or five daily 3-hr exposures to 51-52 ppm. EPA evaluated this study, which received a data quality rating of medium.

Warbrick et al. (2003) exposed Sprague-Dawley rats to 0 or 5187 ppm methylene chloride for 6 hrs/day, 5 days/week for 28 days. On day 23, all rats were injected with sheep red blood cells. Immunoglobulin M (IgM) antibody responses did not differ between methylene chloride-exposed rats and negative controls. Relative spleen weights were reduced in females. This study received a data quality rating of high.

NTP (1986) identified splenic fibrosis at ≥ 2000 ppm in rats and splenic follicular atrophy in mice at 4000 ppm in a two-year inhalation study. Other two-year inhalation studies (Nitschke et al., 1988a; Burek et al., 1984) did not identify histopathological changes in the spleen, lymph node or thymus of rats or hamsters. None of the two-year studies evaluated functional immunity or identified patterns of inflammatory cells in the respiratory tract. None of these studies found increased infections in dosed animals. All two-year studies received high data quality ratings.

Mechanistic Data

U.S. EPA (2011) did not discuss any mechanistic/*in vitro* studies related to immunotoxicity. EPA identified only two relevant studies from the updated literature search that address immune-related activity. In one study, Kubulus et al. (2008) treated male rats with hemin arginate, induced hemorrhage, then treated the rats with a heme oxygenase-1 blocker, and finally

administered methylene chloride. Methylene chloride treatment resulted in decreased pro-inflammatory cytokine TNF-alpha and increased the anti-inflammatory cytokine IL-10 levels, similar to treatment with hemin arginate alone. The authors hypothesized that the MOA for these changes in cytokine levels was related to carbon monoxide generation ([Kubulus et al., 2008](#)).

Mitochondrial activity was assessed by measuring cell viability of peripheral blood mononuclear cells (PBMC) of carp (*Cyprinus carpio carpio*), and ROS were also evaluated in PBMC by measuring oxidation of substrates that generate fluorescent compounds ([Uruga-Tovar et al., 2014](#)). Methylene chloride increased mitochondrial activity and H₂O₂ in a dose-dependent fashion. Overall, the authors demonstrated immunomodulatory effects of methylene chloride in PBMC of carp (*Cyprinus carpio carpio*) that included an acute pro-inflammatory state. Reports of measuring ROS have not been performed on PBMC of the carp prior to publication by Uruga-Tovar et al. ([2014](#)). Therefore, conclusions from the study should be considered with caution and cannot be compared with other compounds.

3.2.3.1.4 Nervous System Effects

Nervous system effects related to methylene chloride exposure include effects related to CNS depression in humans as well as spontaneous activity and other effects in animals. Developmental neurotoxicity has also been observed in human studies and a limited number of animal studies. A limited number of mechanistic studies are also available. EPA focused on evaluating the human experimental studies. Previous peer-reviewed assessments discussed the animal and *in vitro* studies, and these are considered acceptable for supporting the weight of scientific evidence. This section focuses on both longer-term and developmental neurotoxicity studies; section 3.2.3.1.1 describes other acute studies.

Nervous System Effects in Adults

Humans

Silver et al. ([2014](#)) reported no increased deaths from malignancies (SMR of 0.07 with 95% CI of 0.0 to 3.83) or nonmalignant diseases of the nervous system from methylene chloride exposure (SMR 1.04 with 95% CI of 0.83 to 1.31) in a cohort of microelectronics and business machine workers exposed at least 91 days from 1969 to 2001 when compared with death rates in the U.S. population (control group). The characteristics of the general population are likely to differ from the worker population; often, morbidity and mortality rates are lower for workers than for the full population, which includes individuals who are unable to work due to illness ([Li and Sung, 1999](#)). Using this dissimilar control group could mask possible effects observed in workers. Also, the model didn't adjust for other chemical exposures. This study received a data quality rating of medium.

In a case-control study of occupational exposure in a plastic polymer plant that received a data quality rating of medium, exposure to methylene chloride was associated with neurological symptoms (i.e., dizziness and vertigo) ([General Electric Co, 1990](#)). The high methylene chloride exposure group was exposed to a mean concentration of 49 ppm. It is likely that workers were exposed to other chemicals in addition to methylene chloride (e.g., phenol and small amounts of other chemicals).

In a study designed to evaluate persistence of nervous system effects, Lash et al. (1991) examined retired aircraft maintenance workers employed in jobs associated with paint stripping, which mainly use methylene chloride. Workers were exposed for ≥ 6 years with an average length of retirement of approximately five years. Controls were retired mechanics at the same maintenance base where aircraft are maintained/repainted and that had little solvent exposure. The study evaluated 33 symptoms primarily related to CNS effects and physiological measurements. The only large differences between the exposed and control groups was a lower score on attention tasks (effect size approximately -0.55 , $p = 0.08$) and complex reaction time (effect size approximately -0.40 , $p = 0.18$) and a higher score on verbal memory tasks (effect size approximately 0.45 , $p = 0.11$). Sample sizes are low, and the study does not discuss other possible pollutant exposures (Lash et al., 1991). EPA gave this study an overall data quality rating of medium.¹⁹

Data from several cohorts report SMRs related to suicide risk. Hearne and Pifer (1999) report SMRs of 1.8 in two separate cohorts of workers in triacetate film production in Rochester, New York (95% CI: 0.98-3.0 for one cohort and 0.81-3.4 for the other cohort). Similarly, [hoechst celanese corp \(1992\)](#) reports increased risk for the highest exposure group of 350-700 ppm in Maryland triacetate fiber production workers (SMR = 1.8; 95% CI: 0.78- 3.6). Tomenson et al. (2011) didn't identify increased risk. Data quality ratings are high for Hearne and Pifer (1999) and medium for [hoechst celanese corp \(1992\)](#) and Tomenson et al. (2011). Lanes et al. (1993) identified an SMR of 1.19 for suicide risk but U.S. EPA (2011) states that the SMR appears to be incorrect and should be 0.77 (based on numbers of reported expected and observed cases).

Animals

A subchronic study identified CNS depressive effects (incoordination, lethargy) in dogs, monkeys and mice, but not rats; brain edema was also observed in dogs (Haun et al., 1971). Thomas et al. (1972) identified increased activity in mice after 14 weeks exposure to 25 ppm but no effects at 100 ppm. In contrast, a 13-week study using concentrations up to 2000 ppm did not identify any changes in sensory stimuli responses (Mattsson et al., 1990) but the measurements were conducted at least 65 hrs after the last exposure and thus, the study could only assess persistence of effects, not reversible effects that occurred during exposure.

Developmental Neurotoxicity

Humans

Between 2006 and 2015, five studies (Talbot et al. (2015); Roberts et al. (2013); Kalkbrenner (2010); Windham et al. (2006); von Ehrenstein et al. (2014),); see Tables 4, 38, 41, and 57 in supplemental file *Data Extraction Tables for Human Health Hazard Studies*) investigated the

¹⁹In an evaluation of acetate film workers with similar results to other studies, Cherry et al. (1983) found exposure to methylene chloride was statistically significantly associated with sleepiness and tiredness during the morning shift, as well as changes in mood and a deterioration in digit symbol substitution tests. However, due to a loss of more than 50% of the participants with no comparison in attributes with individuals studied, Cherry et al. (1983) was given an unacceptable rating and cannot be relied upon to make conclusions.

association between modeled air emissions or outdoor air concentrations of numerous chemicals (including the 33-37 HAPs, or even more pollutants) and autism spectrum disorder (ASD) in regions across the United States. Methylene chloride was among the few chemicals in these studies that consistently identified odds ratios greater than one (ranging from 1.08 to 1.9), although most of the results lacked statistical significance with the lower end of the confidence interval ranges including values less than 1.0.

Animals

Bornschein et al. (1980) found delayed rates of behavioral habituation to novel environments in offspring from female rats exposed to 4500 ppm methylene chloride via inhalation before and/or during gestation. The effects were observed as early as 10 days of age in both sexes and still observed in 150-day male (but not female) rats. Alexeeff and Kilgore (1983) identified a statistically significant difference in a passive avoidance learning task among three-day old mice exposed to ~47,000 ppm methylene chloride via inhalation compared with controls. In contrast, these authors did not observe any differences for 5- and 8-week old mice (Alexeeff and Kilgore, 1983).

Nitschke et al. (1988b), a two-generation reproductive study in rats, Schwetz et al. (1975), a prenatal developmental toxicity study in rats and mice, and Hardin and Manson (1980), a reproductive/developmental study in rats using multiple exposure designs, did not identify nervous system effects. However, these studies did not measure neurobehavioral outcomes and also did not identify whether tissues of the nervous system were evaluated during histopathological examinations.

There is no single animal model for the complex syndrome that constitutes ASD, although animal study protocols that may approximate some aspects include evaluation of reciprocal social communicative behavior or repetitive and stereotyped behavior. Animal data using these protocols have not been identified for methylene chloride (Pelch et al., 2019).

Mechanistic Data

Solvents are known to produce generalized CNS depression (Moser et al., 2008). General depressants may initially suppress inhibitory systems at low doses to produce excitation and lead to a continuum of effects from excitation to sedation, motor impairment, coma, and ultimately death by depression of respiratory centers (Moser et al., 2008). Moser et al. (2008) discusses several hypotheses regarding mechanisms related to generalized CNS depression but notes that none are definitive. Across solvents, potency has been shown to be correlated with the olive oil:water or octanol:water partition coefficients, suggesting possible disruption of the lipid portions of cell membranes. CNS depression could result from membrane expansion or effects on mitochondrial calcium transport. The effect may also be related to interactions with ligand-gated ion channels and voltage-gated calcium channels, with specific gamma-aminobutyric acid (GABA) type A, N-methyl-D-aspartate (NMDA) and glycine receptors possibly involved (Moser et al., 2008).

Mechanistic information specific to methylene chloride is described for primary nervous system effects related to CNS depression including changes in locomotor activity as well as effects on motor coordination and learning and memory. Bale et al. (2011) reviewed data for methylene chloride and other solvents and note that they may act on several molecular targets in the CNS, likely through multiple mechanisms.

Some of the primary effects of methylene chloride are related to CNS depression and motor incoordination and abnormal gait. Studies have shown that GABA and glutamate receptors in the cerebellum may be involved in motor coordination and general CNS depression. Also, studies with toluene indicate that the dopaminergic system may be involved in changes in locomotion (Bale et al., 2011). Methylene chloride has been shown to increase dopamine along with serotonin in the medulla and increase GABA and glutamate in the cerebellum (Kanada et al., 1994). However, Kanada et al. (1994) did not measure functional changes resulting from these neurochemical changes. Therefore, EPA cannot make definitive conclusions about the associations between these changes and CNS depression and motor changes. Bale et al. (2011) also states that studies have not been conducted to evaluate the neurochemical basis for changes in spontaneous activity for methylene chloride. Data suggest that increased COHb levels result in CNS depression (Putz et al., 1979) but doesn't fully explain the independent and possible additive effect of methylene chloride because a weaker effect (or no effect) on the nervous system was observed with administration of exogenous CO compared with methylene chloride administration (Putz et al., 1979; Winneke, 1974).

Changes in deoxyribonucleic acid (DNA) concentration and enzyme activities in the cerebellum (Rosengren et al., 1986; Savolainen et al., 1981) may be associated with changes in motor activity and neuromuscular function. Among other endpoints, Savolainen (1981) measured changes in succinate dehydrogenase (SDH) from exposure to methylene chloride. SDH is a tricarboxylic acid cycle enzyme that is also part of the mitochondrial electron transport chain (Quinlan et al., 2013). Savolainen (1981) reported decreased SDH in the cerebellum, which coordinates motor activity. SDH levels recovered somewhat but still remained lower than controls during a second week of exposure and after a week-long recovery period. Effects were generally greater for a TWA concentration of 1000 ppm methylene chloride, which included 2 daily 1-hr exposures to 2800 ppm compared with a constant concentration of 1000 ppm (Savolainen et al., 1981). This greater effect may partly explain effects (e.g., respiratory depression, death) experienced by humans after high acute exposures.

Alexeeff and Kilgore (1983) showed that at 47,000 ppm, methylene chloride may affect learning and memory as evidenced by a change in passive avoidance conditioning, and Kanada (1994) showed that acetylcholine (ACh) levels were increased in response to methylene chloride and Bale (2011) notes that memory and cognition deficits are thought to be due to decreased cholinergic system functioning. The increase in ACh seen by Kanada (1994) could lead to altered cognition as a response to inhibiting nuclear ACh receptors to maintain normal function (Bale et al., 2011). Alternately, decreases in learning and memory function may be affected by decreased motor function and CNS depression (Bale et al., 2011); because learning and memory have not been routinely associated with methylene chloride and because the study (Alexeeff and Kilgore, 1983) that identified changes in learning and memory was conducted at a very high concentration, it seems plausible that the effects from methylene chloride may be at least partially related to CNS depression.

Decreased catecholamine in the caudate nucleus and decreased DNA content in the hippocampus as a result of methylene chloride may also suggest possible learning and memory impairment ([Rosengren et al., 1986](#); [Fuxe et al., 1984](#)) based on the location of these decreases. However, as noted above, changes in learning and memory have been identified in only limited studies in humans and animals.

Information is limited regarding the contribution of the parent compound, methylene chloride versus metabolite(s) to nervous system effects. Methylene chloride has been shown to distribute to the brain with higher concentrations than other tissues ([Nac/Aegl, 2008b](#)). Also, increased COHb levels can result in CNS depression e.g., ([Putz et al., 1979](#)) but a weaker effect or no effect was observed with exposure to exogenous CO compared with methylene chloride suggesting that at these concentrations COHb is not the only moiety leading to the effects and may play a minor role ([Putz et al., 1979](#); [Winneke, 1974](#)). CO and subsequently COHb may only result in significant neurobehavioral changes at higher concentrations ([NAC/AEGL, 2008a](#)).

3.2.3.1.5 Reproductive and Developmental Effects

In addition to the epidemiological studies related to nervous system effects noted previously, EPA identified several other relevant epidemiological studies of reproductive and developmental effects and identified effects, including developmental neurotoxicity (which are described in section 3.2.4.1.4), in some studies. EPA did not locate mechanistic data specific to reproductive and developmental toxicity.

Humans

[Brender et al. \(2014\)](#) was identified during the recent literature search. These authors evaluated the association between industrial air releases of chlorinated solvents (including methylene chloride) and birth defects in children. Cases and controls were mothers recruited from the same regions in Texas and birth defects identified from the Texas Birth Defects Registry. Exposure was estimated based on proximity of mothers' residences to emissions and the quantity of methylene chloride released. Differences in certain characteristics such as race, ethnicity and education were controlled for in the statistical analyses. Although methylene chloride was not associated with most birth defects, statistically significant relationships were observed among mothers 35 years or older for two defects: any oral cleft defect (OR = 1.38, with 95% CI: 1.14, 1.67) and cleft lip with or without cleft palate (OR = 1.53, with 95% CI: 1.21, 1.93). The authors also reported that significant linear trends were observed for the association between methylene chloride and isolated conotruncal heart defects for offspring of mothers of all ages (OR for the highest exposure risk value was 1.56, 95% CI: 1.05, 2.32). Selection bias appeared to be low, exclusions from the study were limited and the potential for exposure misclassification was considered to be low. In evaluating outcomes of interest, there is some uncertainty regarding whether exposure occurred during the first trimester; some exposure measurement error could if there is variability in methylene chloride during pregnancy. Because the models did not account for co-exposures to other chlorinated solvents or other chemicals, the association between individual chemicals and the birth outcomes is less certain. In other studies (e.g., the ASD epidemiological studies), methylene chloride was sometimes highly correlated with other compounds. Indeed, some of the other chemicals measured in separate models in this study were

associated with some of the same birth defects more often or showed associations larger in magnitude than methylene chloride. The data quality rating for this study is medium.

Other studies evaluated reproductive/developmental effects. [Bell et al. \(1991\)](#) examined the association between estimated methylene chloride air concentrations in the community surrounding the Eastman Kodak triacetate film facility in Rochester, New York and birth weight of children born to mothers in the surrounding population. Air dispersion modeling was used to estimate exposures; the highest predicted average methylene chloride air concentration in the studied community was $50 \mu\text{g}/\text{m}^3$. Birth certificates were obtained for the years 1976-1987. Because the number of births in non-whites was small, the analysis was restricted to the white population. At the levels of methylene chloride in this study, no significant adverse effect was found between any combination of methylene chloride exposure levels and birthweight. Comparing participants residing in the census tracts with the highest exposure group to the census tracts with no predicted exposure, the OR was 1.0 (95% CI: 0.81, 1.24). The authors note that the exposure estimates from the air dispersion modeling were higher than monitored values in the area. Also, the assignment of methylene chloride exposures to each birth was made using the predominant value of the isopleth for a census tract, and this could have led to some exposure misclassification. This study received a data quality rating of high.

[Taskinen et al. \(1986\)](#) examined spontaneous abortion rates in female workers employed in pharmaceutical factories in Finland. In addition to examining overall rates, [Taskinen et al. \(1986\)](#) conducted a case-control analysis to estimate association between spontaneous abortions and methylene chloride, a solvent commonly used in the pharmaceutical industry, as well as other chemicals. Forty-four cases and 130 controls were identified. For methylene chloride exposure, the prevalence of exposure was 29% and 14% in the cases and controls, respectively. The OR was 2.3 (95% CI: 1.0-5.7; $p = 0.06$); this OR didn't appear to account for co-exposure and possible confounders although controls were matched on maternal age. Less precise results (higher p values) that were similar in magnitude were noted for other solvents (OR range: 1.6 to 3.2). The OR for exposure to four or more solvents (OR: 3.5, $p = 0.05$) was greater than for one to three solvents (OR: 0.8, $p = 0.74$). EPA gave this a data quality score of low based on several measures including method of identifying exposures, temporality, covariate adjustment and characterization and confounding from co-exposures.

Male reproductive effects were investigated in a couple of case series reports. Kelly et al. ([1988](#)) cited in U.S. EPA ([2011](#)) studied 34 men working in the automotive industry who self-referred to a health clinic. Eight men who worked as bonders and routinely dipped hand-held pads (and didn't always use gloves) in buckets of methylene chloride had symptoms of testicular and epididymal tenderness, and sperm counts were $25 \times 10^6/\text{cm}^3$ (oligospermia can be defined as $20 \times 10^6/\text{cm}^3$). Despite not using contraception, the men had not conceived any children (and one reported a miscarriage) – conclusions about these results are not possible because there was no comparison group. Wells et al. ([1989](#)), however, reported a mean sperm count of $54 \times 10^6/\text{cm}^3$ in eleven furniture refinishers (none with oligospermia), slightly higher than the population value of $47 \times 10^6/\text{cm}^3$.

Animals

Animal studies show reproductive/developmental effects in some studies but not others. A two-generation inhalation toxicity study revealed no significant effects on fertility, litter size, neonatal survival, histopathological changes or growth rates in either generation (F1 or F2) of rats exposed up to 1,500 ppm methylene chloride ([Nitschke et al., 1988b](#)).

Raje et al. ([1988](#)) found some evidence of a decrease in fertility index after male mice were exposed to 144 and 212 ppm for 2 hrs/day for 6 weeks and then mated with unexposed females; fertility index values were 80% at each concentration compared with 95% at 0 and 100 ppm, but not statistically significant (overall X^2 p-value of 0.27). U.S. EPA ([2011](#)) conducted some statistical analyses – the trend test using a Cochran-Armitage exact trend test yielded a one-sided p-value of 0.059. Using the Fisher's exact test, one-sided p-value was 0.048 when comparing the combined 144 and 212 ppm groups with the 0 and 100 ppm groups; U.S. EPA ([2011](#)) suggested a NOAEC of 100 ppm (103 ppm) and lowest observable adverse effect concentration (LOAEC) of 150 ppm (144 ppm). This data quality rating is medium.

Pregnant mice and rats were exposed to 1,250 ppm methylene chloride for 7 hours/day during gestation days 6-15 ([Schwetz et al., 1975](#)) and exhibited certain skeletal variants after exposure. In rats, the incidence of ribs or spurs was decreased and incidence of delayed ossification of sternbrae was increased ($p < 0.05$ for both). Mice exhibited an increased number of litters with pups that had a single extra center of ossification in the sternum ($p < 0.05$) ([Schwetz et al., 1975](#)). Hardin and Manson ([1980](#)) did not identify statistically significant changes in the incidence of external, skeletal or soft-tissue anomalies in fetuses of female Long-Evans hooded rats exposed to 4500 ppm methylene chloride before and/or during gestation. However, decreased fetal body weights (by 9-11%) were observed when dams were exposed during gestation only (days 1-17) or both before (12-14 days) and during gestation (1-17 days) ($p < 0.05$ by two-way ANOVA).

Results of oral animal studies did not identify reproductive or developmental effects. Narotsky and Kavlock ([1995](#)) did not observe effects on pup survival, resorptions or weight after pregnant F344 rats were administered doses as high as 450 mg/kg-day on gestational days (GDs) 6–19, although maternal weight was decreased. No effects on reproductive performance endpoints (fertility index, number of pups per litter, pup survival) were found in studies in male and female Charles River CD rats administered methylene chloride via gavage for 18 weeks and administered doses up to 225 mg/kg-day with subsequent exposure to offspring for 13 weeks ([General Electric Company, 1976](#)).

Mechanistic Data

Other than studies measuring general modes of action of methylene chloride (e.g., oxidative stress, genotoxicity, increased COHb), EPA did not identify studies that link reproductive and developmental effects with specific cellular mechanisms.

3.2.3.1.6 Irritation/Burns

Human and animal data that evaluated or reported irritation and burns of skin, eyes, respiratory tract and gastrointestinal tract after use of methylene chloride are summarized below. EPA summarized several human case reports. EPA qualitatively evaluated a human controlled experiment (in consideration of using it for CNS effects from acute/short-term exposure – see Section 3.2.4.1.4); however, other studies were not evaluated for quality.

After two hours of exposure to 986 ppm methylene chloride in air, volunteers reported no symptoms of eye, nose or throat irritation ([Stewart et al., 1972](#)). This study was evaluated qualitatively ([EPA, 2019t](#)) and although the lack of blinding suggests low confidence in the subjective symptom results, the subjects would be likely to over-report (rather than under-report) symptoms if they knew they were exposed to methylene chloride.

Anundi et al. ([1993](#)) did report irritation to the eyes and upper respiratory tract among graffiti removers in an underground station in Sweden. The workers had been on the job between 3 months and 4.7 years. TWA exposures of 18-1,200 mg/m³ (5-340 ppm) were measured in this study and reported exposures to other chemicals were much lower and found in only a limited number of samples ([Anundi et al., 1993](#)).

A 21-year old male working in a furniture stripping shop had first and second-degree burns from direct contact with the liquid after being found slumped over a tank of methylene chloride ([Hall and Rumack, 1990](#)). Direct contact of eyes with methylene chloride in a workplace accident resulted in severe corneal burns; duration of contact is not known. Furthermore, air concentrations of 2300-7200 ppm resulted in irritation after 5-8 minutes ([Hall and Rumack, 1990](#)). Other case reports also indicate that methylene chloride can cause second and third degree burns upon direct contact with the liquid ([Wells and Waldron, 1984](#)).

In one suicide case, ingestion of paint remover containing 75–80% methylene chloride, resulted in death from corrosion of the gastrointestinal tract ([Hughes and Tracey, 1993](#)). The individual was exposed to methanol as well, which can cause respiratory (e.g., nasal) irritation ([EPA, 2013c](#)).

Small increases in corneal thickness and intraocular tension reported after exposure of rabbits to vapors of ≥ 490 ppm methylene chloride were reversible within 2 days after exposure ceased. Following direct eye contact with methylene chloride (0.1 mL), rabbits exhibited inflammation of the conjunctivae and eyelids and increases in corneal thickness and intraocular tension. The effects were reversible within 3 to 9 days ([Ballantyne et al., 1976](#)). NTP ([1986](#)) notes that inflammation and metaplasia in nasal cavities of rats exposed to methylene chloride may have been due to irritation.

Between 2007 and 2016, the Washington Poison Center in King County, WA received 150 calls related to methylene chloride. Thirty-six dermal and ocular cases required follow-up; seven were of moderate severity and the rest were minor. Among these cases, there were nine cases of burns (five were moderate) and three cases of corneal abrasion (two were moderate). Irritation and pain were identified in multiple reports with red eye and skin edema identified in some cases ([Fisk and Whittaker, 2018](#)).

3.2.3.2 Cancer Hazards

EPA identified several epidemiological studies published subsequent to the 2011 IRIS assessment ([U.S. EPA, 2011](#)) as well as one animal bioassay. EPA evaluated these studies as well as epidemiological and chronic animal bioassays from the IRIS assessment. The overall data evaluation ratings for all studies evaluated for data quality are included in the table throughout

this section. EPA also summarized genotoxicity data, which were evaluated for data quality. Other mechanistic studies are summarized but were not evaluated.

3.2.3.2.1 Carcinogenicity

The potential carcinogenicity of methylene chloride has been evaluated in a number of human epidemiological studies and animal cancer bioassays. These data are summarized by target tissue (liver, lung, breast, hematopoietic, brain/CNS and other neoplasms) below.

Liver Cancer

The human epidemiological data are inconclusive as to the association between liver and biliary tract cancer and methylene chloride exposure (Section 3.2.3.1.2). Epidemiological data are limited to four occupational cohort mortality studies of workers involved in CTA fiber ([Gibbs et al., 1996](#); [Lanes et al., 1993](#)) and film base production ([Tomenson, 2011](#); [Hearne and Pifer, 1999](#)) with contradictory findings, and a small cohort study of incident cholangiocarcinoma in Japanese offset-proof print workers that did not show an association with methylene chloride exposure ([Kumagai et al., 2016](#)).

Animal data ([Aiso et al., 2014a](#); [NTP, 1986](#)) provide clear and consistent evidence that methylene chloride induces liver tumors in male and female mice (Tables 3-6 and 3-7). Significant increases in the incidences of hepatocellular adenoma or carcinoma were observed in male and female B6C3F1 and Crj:BDF1 mice exposed via inhalation ([Aiso et al., 2014a](#); [NTP, 1986](#)). Male mice exposed by inhalation also exhibited a significant increase in the incidence of hepatic hemangiomas in the study by Aiso ([2014a](#)), and both male and female mice in this study showed significant exposure-related trends in the incidences of combined hemangiomas and hemangiosarcomas. Increased incidences of hepatocellular adenoma or carcinoma were also observed in male B6C3F1 mice exposed via drinking water ([Serota et al., 1986b](#); [Hazleton Laboratories, 1983](#)). In rats there have been suggestive findings related to liver tumors, with a significant increase in the incidence of hepatic neoplastic nodules or hepatocellular carcinomas in female F344 rats after drinking water exposure ([Serota et al., 1986a](#)) and a significant dose-related trend in the incidence of hepatocellular adenoma or carcinoma in male F344/DuCrj rats after inhalation exposure ([Aiso et al., 2014a](#)).

Table 3-5. Selected Effect Estimates for Epidemiological Studies of Liver Cancers

Reference	Type	SMR/ IRR	95% LCL	95% UCL	Study Quality Evaluation
<i>Liver and biliary tract</i>					
Lanes et al. (1993) (men and women)	SMR	2.98	0.81	7.63	Medium
Lanes et al. (1993) (men and women: ≥ 10 yrs employment, ≥ 20 yrs since first employment)	SMR	5.83	1.59	14.92	Medium
Hearne and Pifer (1999) (men)	SMR	0.42	0.01	2.36	High
Gibbs et al. (1996) (men)	SMR	0.81	0.02	4.49	High

Table 3-5. Selected Effect Estimates for Epidemiological Studies of Liver Cancers

Gibbs et al. (1996) (women)	SMR	(no exposed cases)			
Tomenson et al. (2011) (men)	SMR	(no exposed cases)			Medium
<i>Cholangiocarcinoma</i>					
Kumagai et al. (2016)	IRR	0.45	0.11	1.77	Medium

SMR = Standardized Mortality Ratio

IRR = incidence rate ratios

LCL = lower confidence limit

UCL = upper confidence limit

Table 3-6. Summary of Significantly Increased Liver Tumor Incidences in Inhalation Studies of Methylene Chloride

Male Mice	Concentration (mg/m ³)			
	0	3500	7000	14,000
<i>Aiso et al. (2014a) (BDF1)</i>				
Hepatocellular adenoma	10/50 [^]	13/50	14/50	15/50
Hepatocellular carcinoma	10/50 [^]	9/50	14/50	20/50*
Hepatocellular adenoma or carcinoma	15/50 [^]	20/50	25/50*	29/50*
Hepatic hemangioma	0/50 [^]	4/50	3/50	5/50*
Hepatic hemangioma or hemangiosarcoma	1/50 [^]	4/50	4/50	6/50
<i>NTP (1986) (B6C3F1)</i>				
Hepatocellular adenoma	10/50	NT	14/49	14/50
Hepatocellular carcinoma	13/50 [^]	NT	15/49	26/50*
Hepatocellular adenoma or carcinoma	22/50 [^]	NT	24/49	33/50*
Female Mice	Concentration (mg/m ³)			
	0	3500	7000	14,000
<i>Aiso et al. (2014a) (F344/DuCrj)</i>				
Hepatocellular adenoma	1/50 [^]	7/50*	4/49	16/50*
Hepatocellular carcinoma	1/50 [^]	1/50	5/49	19/50*
Hepatocellular adenoma or carcinoma	2/50 [^]	8/50*	9/49*	30/50*
Hepatic hemangioma or hemangiosarcoma	3/50 [^]	2/50	0/49	7/50
<i>NTP (1986) (F344)</i>				
Hepatocellular adenoma	2/50 [^]	NT	6/48	22/48*
Hepatocellular carcinoma	1/50 [^]	NT	11/48	32/48*

Table 3-6. Summary of Significantly Increased Liver Tumor Incidences in Inhalation Studies of Methylene Chloride

Hepatocellular adenoma or carcinoma	3/50 [^]	NT	16/48*	40/48*
	Concentration (mg/m³)			
Male Rats	0	3500	7000	14,000
<i>Aiso et al. (2014a) (F344/DuCrj)</i>				
Hepatocellular adenoma or carcinoma	1/50 [^]	0/50	2/50	3/50
<i>Study Quality Evaluation</i>				
Aiso et al. (2014a)	High			
NTP (1986)	High			

[^]Significant dose-related trend ($p \leq 0.05$)

*Significant pairwise comparison ($p \leq 0.05$)

NT = not tested

Table 3-7. Summary of Significantly Increased Liver Tumor Incidences in Oral Studies of Methylene Chloride

<i>Hazleton Labs (1983); Serota et al., (1986b) (B6C3F1)</i>					
Male Mice	Dose (mg/kg-day)				
	0	61	124	177	234
Hepatocellular adenoma	10/125	20/200	14/100	14/99	15/125
Hepatocellular carcinoma	14/125	33/200	18/100	17/99	23/125*
Hepatocellular adenoma or carcinoma	24/125	51/200	30/100*	31/99*	35/125*
<i>Serota et al. (1986a) (F344)</i>					
Female Rats	Dose (mg/kg-day)				
	0	6	58	136	263
Neoplastic nodules	0/135	1/85	2/85	1/85	3/85
Hepatocellular carcinoma	0/135	0/85	2/85	0/85	2/85
Neoplastic nodule or hepatocellular carcinoma	0/135^	1/85	4/85*	1/85	5/85*
<i>Study Quality Evaluation</i>					
Hazleton Labs (1983) Serota et al. (1986b)	Medium				
Serota et al. (1986a)	High				

^Significant dose-related trend ($p \leq 0.05$)*Significant pairwise comparison ($p \leq 0.05$)***Lung Cancer***

Most of the human data on lung cancer and methylene chloride exposure are not conclusive and most do not show an association with methylene chloride (Section 3.2.3.2). Standardized mortality rates for lung cancer were decreased (<1) in cohorts of CTA fiber or film workers (Tomenson, 2011; Hearne and Pifer, 1999; Tomenson et al., 1997; Gibbs et al., 1996; Lanes et al., 1993). In case-control studies, Vizcaya et al. (2013) and Mattei et al. (2014) found no excess risk of lung cancer among men with occupational exposure to methylene chloride. Although Mattei et al. (2014) observed an increased risk of lung cancer among women, further analysis indicated that the increase was largely attributable to perchloroethylene exposure.

Siemiatycki (1991), on the other hand, identified an increased risk (at significance level of $p = 0.10$) in a case-control study in males aged 35-70 in the Montreal area. Some studies that used population mortality rates and that were conducted using employees of companies with no-smoking policies may have been confounded by differences in smoking rates among the exposed and non-exposed populations.

In animal studies, methylene chloride produced large, statistically significant increases in lung tumor incidences in male and female mice exposed by inhalation ([Aiso et al., 2014a](#); [NTP, 1986](#)).

There was also some evidence for production of lung tumors in mice by oral exposure to methylene chloride. [Maltoni et al. \(1988\)](#) reported a nonsignificant dose-related trend for higher incidences of pulmonary adenomas in male, but not female, mice in an oral gavage study that was, however, terminated at 64 weeks due to high mortality. A 2-year drinking water study did not find any increase in lung tumor incidence in male or female mice ([Serota et al., 1986b](#)). Lung tumors were not increased by methylene chloride in rats or hamsters by inhalation or oral exposure ([Maltoni et al., 1988](#); [Nitschke et al., 1988a](#); [NTP, 1986](#); [Serota et al., 1986a](#); [Burek et al., 1984](#)).

Table 3-8. Selected Effect Estimates for Epidemiological Studies of Lung Cancers

Reference	Type	SMR/ OR	95% LCL	95% UCL	Study Quality Evaluation
Lanes et al. (1993) (men and women)	SMR	0.80	0.43	1.37	Medium
Hearne and Pifer (1999) (men)	SMR	0.75	0.49	1.09	High
Tomenson et al. (2011) (men)	SMR	0.48	0.31	0.69	Medium
Gibbs et al. (1996) (men)	SMR	0.55	0.31	0.91	High
Gibbs et al. (1996) (women)	SMR	2.29	0.28	8.29	High
Vizcaya et al. (2013)	OR	1.1	0.6	1.9	Medium
Mattei et al. (2014) (women)	OR	1.38	0.74	2.57	Medium
Siemiatycki et al. (1991) (all lung)^	OR	3.8	1.2	12.0	Medium
Siemiatycki et al. (1991) (squamous cell)^	OR	4.0	0.9	17.3	Medium

^ORs are for substantial exposure. Siemiatycki et al. ([1991](#)) also presents ORs for 'any' exposure, which are lower than for substantial exposures. Also, the LCL and UCL are the 90%ile values, not 95%ile values.

Table 3-9. Summary of Significantly Increased Lung Tumor Incidences in Inhalation Studies of Methylene Chloride

Male Mice	Concentration (mg/m ³)			
	0	3500	7000	14,000
<i>Aiso et al. (2014a) (BDF1)</i>				
Bronchoalveolar adenoma	7/50^	3/50	4/50	14/50
Bronchoalveolar carcinoma	1/50^	14/50*	22/50*	39/50*
Bronchoalveolar adenoma or carcinoma	8/50^	17/50*	26/50*	42/50*
<i>NTP (1986) (B6C3F1)</i>				

Table 3-9. Summary of Significantly Increased Lung Tumor Incidences in Inhalation Studies of Methylene Chloride

Bronchoalveolar adenomas	3/50 [^]	NT	19/50*	24/50**
Bronchoalveolar carcinomas	2/50 [^]	NT	10/50*	28/50*
Bronchoalveolar adenomas or carcinomas	5/50 [^]	NT	27/50*	40/50*
Female Mice	0	3500	7000	14,000
<i>Aiso et al. (2014a) (BDF1)</i>				
Bronchoalveolar adenomas	2/50 [^]	4/50	5/49	12/50*
Bronchoalveolar carcinomas	3/50 [^]	1/50	8/49	20/50*
Bronchoalveolar adenomas or carcinomas	5/50 [^]	5/50	12/49*	30/50*
Bronchoalveolar adenoma or carcinoma or adenosquamous carcinoma	5/50 [^]	5/50	12/49*	30/50*
<i>NTP (1986) (B6C3F1)</i>				
Bronchoalveolar adenomas	2/50 [^]	NT	23/48*	28/48*
Bronchoalveolar carcinomas	1/50 [^]	NT	13/48*	29/48*
Bronchoalveolar adenomas or carcinomas	3/50 [^]	NT	30/48*	41/48*
<i>Study Quality Evaluation</i>				
Aiso et al. (2014a)	High			
NTP (1986)	High			

[^]Significant dose-related trend ($p \leq 0.05$)

*Significant pairwise comparison ($p \leq 0.05$)

Breast Cancer

The available epidemiological data on breast cancer, including two occupational cohort mortality studies, a prospective population cohort study and a case-control study, provide inconclusive results. The mortality rate for breast cancer was less than unity in a cohort of CTA fiber production workers (Lanes et al., 1993), but an elevated HR was reported among Air Force base employees (Radican et al., 2008). Because exposure at the Air Force base was predominantly trichloroethylene, the CTA cohort provides greater specificity for methylene chloride. A case control study by Cantor (1995) showed increased ORs for breast cancer among women with the highest exposure probability; however, this study estimated exposure based on occupation reported on death certificates, instead of detailed job history obtained by in-person or proxy interview. Garcia (2015) found no increased risk when using modeled outdoor air concentrations from emissions (EPA NATA). A summary measure of multiple pollutants also did not yield an increased HR (HR = 1.05).

Animal data provide some evidence that methylene chloride induces mammary tumors in male and female rats following inhalation exposure. These incidences of mammary gland

fibroadenoma were significantly increased in male F344/DuCrj rats (Aiso et al., 2014a) and female F344 rats (NTP, 1986) exposed to methylene chloride via inhalation. Exposure-related trends were reported for both sexes. The incidence of this tumor was higher, and occurred at a lower concentration, in female rats compared to males. Significant increases were also reported in male rats for the combined incidences of mammary gland fibroadenoma or adenoma (Aiso et al., 2014a) and adenoma, fibroadenoma or fibroma (NTP, 1986). In female rats, the combined incidence of adenoma, fibroadenoma, or adenocarcinoma was increased (NTP, 1986). A significant dose-related trend was observed in the incidence of benign mammary tumors in male Sprague-Dawley rats (Burek et al., 1984). Chronic inhalation studies in mice and chronic oral studies in rats and mice did not demonstrate an increased incidence of mammary tumors.

Table 3-10. Selected Effect Estimates for Epidemiological Studies of Breast Cancers

Reference	Type	SMR/ OR/ HR	95% LCL	95% UCL	Study Quality Evaluation
Lanes et al. (1993)	SMR	0.54	0.11	1.57	Medium
Radican et al. (2008)	HR	2.36	0.98	5.65	Medium
Cantor et al. (1995) white women	OR	1.17	1.1	1.3	High
Cantor et al. (1995) black women	OR	1.46	1.2	1.7	High
Garcia et al. (2015)	HR	1.04	0.96	1.13	High

Table 3-11. Summary of Significantly Increased Mammary Tumor Incidences in Inhalation Studies of Methylene Chloride

Male Rats	Concentration (mg/m ³)			
	0	3500	7000	14,000
<i>Aiso et al. (2014a) (F344/DuCrj)</i>				
Mammary gland fibroadenoma	1/50 [^]	2/50	3/50	8/50*
Mammary gland fibroadenoma or adenoma	2/50 [^]	2/50	3/50	8/50*
Mammary gland fibroadenoma or adenoma or adenocarcinoma @	3/50 [^]	2/50	3/50	8/50
<i>NTP (1986) (F344)</i>				
Mammary gland subcutaneous tissue fibroma or sarcoma #	1/50 [^]	1/50	2/50	5/50
Mammary gland fibroadenoma	0/50 [^]	0/50	2/50	4/50
Mammary gland or subcutaneous tissue adenoma, fibroadenoma, or fibroma	1/50 [^]	1/50	4/50	9/50*

Table 3-11. Summary of Significantly Increased Mammary Tumor Incidences in Inhalation Studies of Methylene Chloride

<i>Burek et al. (1984) (Sprague-Dawley)</i>				
	Concentration (mg/m³)			
	0	1800	5300	12,000
Benign mammary tumors	7/92 [^]	3/95	7/95	14/97
Female Rats	Concentration (mg/m³)			
	0	3500	7000	14,000
<i>Aiso et al. (2014a) (F344/DuCrj)</i>				
Mammary gland fibroadenoma	7/50 [^]	7/50	9/50	14/50
Mammary gland fibroadenoma or adenoma	7/50 [^]	8/50	10/50	14/50
Mammary gland fibroadenoma or adenoma or adenocarcinoma @	7/50 [^]	9/50	10/50	14/50
<i>NTP (1986) (F344)</i>				
Mammary gland fibroadenoma	5/50 [^]	11/50*	13/50*	22/50*
Mammary gland adenoma, fibroadenoma, or adenocarcinoma #	6/50 [^]	13/50	14/50*	23/50*
<i>Nitschke et al. (1988a) (Sprague-Dawley)</i>				
	Concentration (mg/m³)			
	0	180	700	1800
Benign mammary tumors	52/70	58/70	61/70*	55/70
<i>Study Quality Evaluations</i>				
Aiso et al. (2014a)	High			
Burek et al. (1984)	High			
Nitschke et al. (1988a)	High			
NTP (1986)	High			

[^]Significant dose-related trend ($p \leq 0.05$)

*Significant pairwise comparison ($p \leq 0.05$)

@ Adenocarcinomas were observed in 0, 2, 1 and 0 female rats at 0, 3500, 7000 and 14,000 mg/m³; no malignant tumors were seen in male rats

Sarcoma incidence was observed in 1 male at the highest concentration (14,000 mg/m³); Adenocarcinomas/ carcinomas were observed in 1, 2, 2 and 0 female rats at 0, 3500, 7000 and 14,000 mg/m³

Hematopoietic Cancer

As presented in Table 3-12, the association between various hematopoietic cancers and exposure to methylene chloride has been examined in occupational cohort mortality studies ([Tomenson, 2011](#); [Radican et al., 2008](#); [Hearne and Pifer, 1999](#)) and population-based case control studies ([Christensen et al., 2013](#); [Morales-Suárez-Varela et al., 2013](#); [Barry et al., 2011](#); [Gold et al., 2010](#); [Wang et al., 2009](#); [Costantini et al., 2008](#); [Seidler et al., 2007](#); [Miligi et al., 2006](#)). Findings were inconsistent and inconclusive for most categories of hematopoietic cancers (leukemia, multiple myeloma, Hodgkin lymphoma, non-Hodgkin lymphoma (NHL)). However, ORs for B-cell subtypes of NHL were consistently increased in three case-control studies that evaluated this tumor type ([Barry et al., 2011](#); [Seidler et al., 2007](#); [Miligi et al., 2006](#)). For example, [Miligi et al. \(2006\)](#) identified an OR for B cell NHL of 3.2, which was higher than the ORs for all other chemicals studied. Despite these more consistent results for B-cell NHL, the studies did not control for other chemical exposures. In addition, there was evidence (e.g., for [Miligi et al. \(2006\)](#)) that some chemical exposures were highly correlated and other chemicals were also associated with the outcomes of interest, making it difficult to attribute effects to methylene chloride alone. NTP ([1986](#)), Mennear et al. ([1988](#)) (which is the published version of NTP ([1986](#))) and Aiso et al. ([2014a](#)) each reported an increased incidence of mononuclear cell leukemia in female (but not male) rats (Table 3-13). However, the incidences did not exhibit monotonic dose-response relationships.

Table 3-12. Selected Effect Estimates for Epidemiological Studies of Hematopoietic Cancers

Reference	Type	SMR/ OR/ HR	95% LCL	95% UCL	Study Quality Evaluation
<i>Non-Hodgkin Lymphoma (NHL)</i>					
Hearne and Pifer (1999)	SMR	0.49	0.06	1.78	High
Radican et al. (2008) (men) (women)	HR	2.02	0.76	5.42	High
		No observed NHL deaths			
Miligi et al. (2006)	OR	1.7	0.7	4.3	High
Wang et al. (2009)	OR	1.5	1.0	2.3	Medium
Christensen et al. (2013)	OR	0.6	0.2	2.2	Medium
<i>B-cell NHL</i>					
Seidler et al. (2007)	OR	2.7	0.5	14.5	High
Barry et al. (2011) (diffuse large B-cell lymphoma)	OR	2.10	1.15	3.85	High
Miligi et al. (2006) (small lymphocytic lymphoma*)	OR	3.2	1.0	10.1	High
<i>T-cell NHL (Mycosis Fungoides)</i>					
Morales-Suarez-Varela et al. (2013) (women)	OR	2.90	0.45	15.72	High

Table 3-12. Selected Effect Estimates for Epidemiological Studies of Hematopoietic Cancers

<i>Hodgkin Lymphoma</i>					
Hearne and Pifer (1999)	SMR	1.82	0.20	6.57	High
Seidler et al. (2007)	OR	0.7	0.2	3.6	High
<i>Multiple Myeloma</i>					
Hearne and Pifer (1999)	SMR	0.68	0.01	3.79	High
Radican et al. (2008) (men) (women)	HR	2.58	0.86	7.72	
		No observed multiple myeloma deaths			
Gold et al. (2010)	OR	2.0	1.2	3.2	Medium ^a
<i>Leukemia</i>					
Hearne and Pifer (1999)	SMR	2.04	0.88	4.03	High
hoechst celanese corp (1992) (Maryland cohort)	SMR	1.9	0.51	4.8	Medium
hoechst celanese corp (1992) (South Carolina cohort)	SMR	0.90	0.02	3.71	Medium
Tomenson et al. (2011)	SMR	1.11	0.36	2.58	Medium
Costantini et al. (2008)	OR	0.5	0.1	2.3	Medium
Costantini et al. (2008) (chronic lymphocytic leukemia*)	OR	1.6	0.3	8.6	Medium
Infante-Rivard et al. (2005)	OR	3.22	0.88	11.7	High
*These two diagnoses differ only in how they present (leukemia or lymphoma presentation).					
^a Downgraded from High (1.6) due to small numbers of exposed cases and controls					

Table 3-13. Summary of Mononuclear Cell Leukemia Incidences in Inhalation Studies of Methylene Chloride

	Concentration (mg/m ³)			
	0	3500	7000	14,000
Male Rats				
Aiso et al. (2014a) (F344/DuCrj)	3/50	3/50	8/50	4/50
NTP (1986) (F344/N)	34/50	26/50	32/50	35/50
Female Rats	Concentration (mg/m ³)			
	0	3500	7000	14,000
Aiso et al. (2014a) (F344/DuCrj)	2/50 [^]	4/50	8/50*	7/50

Table 3-13. Summary of Mononuclear Cell Leukemia Incidences in Inhalation Studies of Methylene Chloride

NTP (1986) (F344/N)	17/50	17/50	23/50 [#]	23/50 [#]
<i>Study Quality Evaluations</i>				
Aiso et al. (2014a)	High			
NTP (1986)	High			

[^]Indicates statistically significant exposure-related trend

^{*}Indicates statistically significant difference from concurrent control.

[#]Statistically significant difference from concurrent control by life table test.

Brain and CNS Cancer

Epidemiological data on brain and CNS tumors after methylene chloride exposure are inconclusive (see Table 3-14). Two occupational cohort studies (Tomenson, 2011; Hearne and Pifer, 1999) reported non-significantly elevated SMRs for brain and CNS cancers. Two case-control studies reported slightly increased ORs (Cocco et al., 1999; Heineman et al., 1994). The OR (1.2) reported by Cocco (1999) was statistically significantly increased. This study used an imprecise exposure assessment based on occupation reported on each subject's death certificate, and it is not known how the OR would change with more precise exposure information. Two case-control studies with more robust exposure assessments (Ruder et al., 2013; Neta et al., 2012) did not show increases in the ORs for two of the most common brain cancers (gliomas and meningiomas). The only animal evidence of brain or CNS tumors is the observation of low incidences of rare astrocytomas in methylene chloride-exposed Sprague-Dawley rats with incidences of 0, 1, 2, 1 (per 70 males/group) at 0, 50, 200, or 500 ppm (0, 175, 702, or 1755 mg/m³) (Nitschke et al., 1988a). No brain or CNS tumors were observed in F344 rats or in mice exposed by inhalation to higher concentrations (Aiso et al., 2014a; NTP, 1986).

Table 3-14. Selected Effect Estimates for Epidemiological Studies of Brain and CNS Cancers

Reference	Type	SMR/OR/ HR	95% LCL	95% UCL	Study Quality Evaluation
<i>Tumor type not specified</i>					
Hearne and Pifer (1999) (New York)	SMR	2.16	0.79	4.69	High
Tomenson et al. (2011) (U.K.)	SMR	1.83	0.79	3.60	Medium
Heineman et al. (1994) (U.S.)	OR	1.3	0.9	1.8	Medium
Cocco et al. (1999) (U.S.)	OR	1.2	1.2	1.3	Medium
<i>Meningioma</i>					
Cocco et al. (1999) (U.S.)	OR	1.2	0.7	2.2	Medium

Table 3-14. Selected Effect Estimates for Epidemiological Studies of Brain and CNS Cancers

Neta et al. (2012) (U.S.)	OR	1.6	0.7	3.5	High
<i>Glioma</i>					
Neta et al. (2012) (U.S.)	OR	0.8	0.6	1.1	High
Ruder et al. (2013) (U.S.)	OR	0.8	0.66	0.97	High

Other Cancers

Epidemiological studies provide limited data regarding other cancers. Carton et al. (2017), assigned a data quality score of medium, found no association between methylene chloride exposure and risk of squamous cell carcinoma of the head and neck in a case-control study of women in France. Dosemeci et al. (1999) found no increased risk of renal cell carcinoma in a population case-control study in Minnesota from exposure to methylene chloride estimated based on job matrices; this study was given a data quality rating of medium. Purdue et al. (2016) presents results of a sub-study within the population case-control U.S. Kidney Cancer Study and did not identify a statistically significant increase in kidney cancer. The ORs in this study for lower exposure probability groups were 1.2 (95% CI:0.6-1.4 in the lowest group) and the OR for the highest exposure probability group was 0.9 (95% CI: 0.6-1.6). Thus, no trend regarding increased risk was identified for the higher likely exposure group. Purdue et al (2016) received a high (1.4) data quality rating. Siemiatycki (1991), in a case-control study, identified an increased risk of rectal cancer (OR = 4.8; 90% CI: 1.7-13.8) among males aged 35-70 in the Montreal area identified as having significant exposure to methylene chloride (using a significance level of $p = 0.10$). This study received a data quality rating of medium.

Studies of other cancers in mice or rats exposed by inhalation reported increased incidences or dose-related trends in the incidences of adrenal gland pheochromocytomas, subcutaneous fibromas or fibrosarcomas, and endometrial tumors (Aiso et al., 2014a); mesotheliomas (Aiso et al., 2014a; NTP, 1986); hemangiomas or hemangiosarcomas (NTP, 1986); or salivary gland sarcomas (Burek et al., 1984). In general, these tumors occurred at low frequency and were not consistent across studies, species, or sexes, and the findings, therefore, are considered equivocal.

3.2.3.2.2 Genotoxicity and Other Mechanistic Information**Genotoxicity**

Methylene chloride has been tested for genotoxicity in both *in vivo* and *in vitro* systems and in mammalian and non-mammalian organisms. The vast majority of these studies received high data quality ratings, a few received medium scores and a few had unacceptable ratings. The following paragraphs summarize these results and Appendix K presents detailed tables of results for the high and medium quality studies. The supplemental file *Data Quality Evaluation of Human Health Hazard Studies – Animal and In Vitro Studies* (EPA, 2019u) presents the data quality ratings for all studies, both acceptable and unacceptable.

Positive results have generally been identified in systems that exhibit GST activity, specifically GSTT1, indicating that metabolites of the GST are likely responsible for the tumorigenic activity. Information indicates S-(chloromethyl)glutathione as most likely to result in genotoxic damage, but DNA damage resulting from formaldehyde, another metabolite of methylene chloride via the GST pathway, is also possible ([U.S. EPA, 2011](#)).

Thier et al. ([1998](#)) cited by U.S. EPA ([2011](#)) found species' specific liver GSTT1 isozyme activity after methylene chloride exposure to be ordered as follows (from highest to lowest): mice, rats, human high and low conjugators, hamsters and human non-conjugators. When comparing metabolism more generally by the GST pathway (irrespective of isozymes) in liver and lung tissues, mice also are more active than rats, humans and hamsters ([U.S. EPA, 2011](#)). However, human high conjugator GSTT1 activity in erythrocytes was the same as male mouse liver activity and 61% of the female mouse liver activity. These relative activities may be the reason for differences in genotoxicity among species as indicated below.

Increased frequencies of micronuclei and DNA damage were found in peripheral blood lymphocyte or leukocyte samples from workers exposed to methylene chloride ([Zeljetic et al., 2016](#)).

Studies in mice exposed to methylene chloride showed significant increases in chromosomal aberrations in the lung ([Allen et al., 1990](#)); micronuclei in peripheral erythrocytes ([Allen et al., 1990](#)); and DNA damage in the liver, lung, and peripheral lymphocytes ([Sasaki et al., 1998b](#); [Casanova et al., 1996](#); [Graves et al., 1995](#); [Graves et al., 1994b](#); [Casanova et al., 1992](#); [Allen et al., 1990](#)). No DNA damage or increased gene mutations were observed in the livers of *gpt* delta mice after 4 weeks of inhalation exposure to 800 ppm ([Suzuki et al., 2014](#)). This was a lower exposure concentration compared with the levels inducing DNA strand breaks (≥ 2000 ppm) or increased tumor incidences. It is possible that CYP2E1 metabolism was not saturated at the lower concentrations, limiting the formation of DNA-reactive GST metabolites.

Fewer *in vivo* data are available for rats, but available information shows positive evidence for DNA SSBs in rat liver after exposure to methylene chloride ([Kitchin and Brown, 1989](#)). Unlike mice, rats exposed via inhalation did not exhibit DNA SSBs in liver and lung cell homogenates or hepatocytes at 2,000 ppm or higher ([Graves et al., 1995](#); [Graves et al., 1994b](#)). Similar to results for mice, methylene chloride did not induce unscheduled DNA synthesis (UDS) in rat hepatocytes after inhalation ([Trueman and Ashby, 1987](#)). An intraperitoneal UDS study in rats was also negative ([Mirsalis et al., 1989](#)). Also similar to the results in mice, rats exposed to methylene chloride at a single 5 mg/kg intraperitoneal dose exhibited no DNA adducts in liver or kidney cells ([Watanabe et al., 2007](#)). Hamsters exposed to 4,000 ppm methylene chloride via inhalation for 3 days did not exhibit DNA-protein cross links in liver or lung cells ([Casanova et al., 1996](#)).

In vitro testing in human cells and cell lines showed that methylene chloride induced micronuclei ([Doherty et al., 1996](#)) and sister-chromatid exchange ([Olvera-Bello et al., 2010](#)) and exhibited a weak trend in DNA damage based on the comet assay ([Landi et al., 2003](#)). Methylene chloride did not induce DNA SSBs ([Graves et al., 1995](#)) or DNA-protein cross-links ([Casanova et al., 1997](#)) in human cells.

In vitro studies are also available for other mammalian tissues. Both mouse and rat hepatocytes showed DNA damage when incubated with methylene chloride *in vitro* ([Graves et al., 1994b](#)), and DNA-protein cross-links were observed in mouse (but not rat) hepatocytes ([Casanova et al., 1997](#)). In mouse club lung cells tested *in vitro*, DNA damage was induced by methylene chloride ([Graves et al., 1995](#)). *In vitro* testing of hamster cells for forward mutations, sister chromatid exchanges and DNA damage after methylene chloride exposure generally showed negative results when testing was conducted without the addition of GST activity from mice ([Graves et al., 1995](#); [Thilagar and Kumaroo, 1983](#); [Jongen et al., 1981](#)). When GST activity was added in testing of hamster cells, positive results were seen for *hprt* mutation ([Graves et al., 1996](#); [Graves and Green, 1996](#)), DNA damage ([Hu et al., 2006](#); [Graves and Green, 1996](#)), and DNA-protein cross-links ([Graves and Green, 1996](#); [Graves et al., 1994b](#)).

Both forward and reverse mutagenicity testing of methylene chloride in bacteria (*S. typhimurium* and *E. coli*) has yielded positive results both with and without exogenous metabolic activation, generally in strains such as TA100 and TA98 that have higher GST activity ([Demarini et al., 1997](#); [Pegram et al., 1997](#); [Graves et al., 1994a](#); [Roldán-Arjona and Pueyo, 1993](#); [Thier et al., 1993](#); [Dillon et al., 1992](#); [Zeiger, 1990](#); [Green, 1983](#); [Jongen et al., 1982](#); [Jongen et al., 1978](#)).

As an example of mutations associated with GSTT1 activity, Demarini et al. ([1997](#)) found that in *Salmonella*, methylene chloride was approximately 10 times more mutagenic in the presence of GSTT1 than in the absence of GSTT1. Furthermore, all methylene chloride-induced mutations induced G to A base substitutions in the presence of GSTT1, compared with only 15% G to A substitutions in the absence of GSTT1, showing the difference in mutation signature with GSTT1.

Other Mechanistic Data

Available data are not adequate to consider other modes of action for risk evaluation. Kari et al. ([1993](#)) (cited in U.S. EPA ([2011](#))) found no evidence of cytotoxicity or proliferative non-neoplastic lesions preceding tumors in a series of stop-exposure studies focused on the liver and lung. Also, sustained cell proliferation was not observed in livers of female mice exposed to methylene chloride ([Foley et al., 1993](#)) (cited in U.S. EPA ([2011](#))). There is no evidence of histologic changes or increased cell proliferation in lung tissue of female B6C3F1 mice exposed to methylene chloride for up to 26 weeks ([Kanno et al., 1993](#)). Although acute exposure produced cell proliferation in bronchiolar epithelium, it was not sustained with longer exposure; proliferation may have been a response to vacuolization of club cells and may have involved a CYP metabolite ([Foster et al., 1994](#)). Some cell proliferation has been observed at higher concentrations (5250-14000 mg/m³) in lungs of mice but not at lower concentrations (1750 mg/m³ and below) after acute exposure; data, however, are not available after longer-term exposure ([Casanova et al., 1996](#)). Finally, Aiso et al. ([2014a](#)) identified significant increases in hyperplasia in terminal bronchioles in mice only at 14,000 mg/m³ whereas lung tumors were significantly increased at ≥ 3510 mg/m³.

Andersen et al. ([2017](#)) identified changes in gene expression in mice exposed to methylene chloride, with marked changes occurring in several genes associated with circadian clocks.

Results indicate that liver and lung tumors from methylene chloride exposure appear to be related to core changes in circadian processes in liver and lung tissue. Andersen et al. (2017) also link circadian rhythms to metabolism showing different patterns in lung versus liver tissue. The common circadian clock effects are for genes that code for regulatory proteins. The authors also identified decreased tissue oxygenation from elevated COHb and the altered association of reduced oxygenation to both circadian cycle proteins and tissue metabolism as the likely mode of action for tissue responses to methylene chloride, but they note that this conclusion is tentative.

Data were not identified suggesting a receptor-mediated mode (e.g., peroxisome proliferation resulting from PPAR- α activation; enzyme induction by constitutive androstane receptor (CAR), pregnane X receptor (PXR), or aryl hydrocarbon receptor (AhR) activation).

3.2.4 Weight of Scientific Evidence

The following sections describe the weight of the scientific evidence for both non-cancer and cancer hazard endpoints. Factors considered in weighing the scientific evidence included consistency and coherence among human and animal studies, quality of the studies (such as whether studies exhibited design flaws that made them unacceptable) and biological plausibility. Relevance of data was considered primarily during the screening process but may also have been considered when weighing the evidence.

3.2.4.1 Non-Cancer Hazards

The following sections consider and describe the weight of the scientific evidence of health hazard domains discussed in Section 3.2.3.1. These domains include toxicity from acute/short-term exposure; liver effects; nervous system effects; immune system effects; reproductive and developmental effects; and irritation/burns.

3.2.4.1.1 Toxicity from Acute/Short-Term Exposure

Medium confidence human experimental studies of objective measures indicate that CNS depression is a sensitive and common effect after acute exposure (e.g., (Putz et al., 1979; Winneke, 1974; Stewart et al., 1972)). Although Stewart et al. (1972) also evaluated subjective symptoms, these results were given a low confidence rating due to lack of blinding. Information from case reports of accidental or large exposures supports this conclusion (Nrc, 2008). Data suggest that increased COHb levels result in CNS depression (Putz et al., 1979) but also support an independent and possible additive effect of methylene chloride with COHb levels based on a weaker (or no) effect on the nervous system from exogenous CO compared with methylene chloride administration (Putz et al., 1979; Winneke, 1974). Although COHb can continue to rise after exposure has ceased and thus COHb may still be relevant at longer time points, both Putz et al. (1979) and Winneke (1974) were conducted for 3.8 or 4 hours, and EPA considers Putz et al. (1979) to still be relevant for an 8-hour duration.

The nervous system effects are supported by inhalation toxicity data in animals showing CNS depression with decreased motor activity, changes in responses to sensory stimuli and some impairment of memory (U.S. EPA, 2011). Data from oral animal studies also identified nervous system effects that include sensorimotor and neuromuscular changes after acute and short-term exposure as well as excitability, autonomic effects, decreased activity and convulsions (one rat) after short-term exposure (Moser et al., 1995; General Electric Co, 1976a).

Cardiotoxicity has been rarely reported as the sole cause of deaths or poisonings from methylene chloride and is not identified as the most sensitive effect in available evidence ([Nac/Aegl, 2008b](#); [ATSDR, 2000](#)).²⁰ However, during exercise, individuals with cardiac disease have been identified as experiencing angina more quickly after CO exposure and resulting increases in COHb ([Nac/Aegl, 2008b](#)). Based on this evidence and the limited data that does suggest some association between methylene chloride and cardiac endpoints, EPA considers that increased COHb levels resulting from inhalation exposure to methylene chloride may also result in adverse effects in individuals with cardiac disease, a sensitive subpopulation. Data are available from human toxicokinetic studies that link increased methylene chloride exposure to increased COHb levels in blood; many of these studies ([Andersen et al., 1991](#); [Divincenzo and Kaplan, 1981](#); [Peterson, 1978](#); [Astrand et al., 1975](#); [Ratney et al., 1974](#)) were used as the basis of the SMAC.

Although acute effects other than CNS effects have been reported in human and animal studies (such as liver or lung effects), they are less often reported, based on inconclusive evidence or are not as sensitive (e.g., reported in lethal or non-lethal case reports after exposure to high or expected high methylene chloride concentrations) ([Nac/Aegl, 2008b](#)). Furthermore, although NAC/AEGL ([2008b](#)) report effects in lungs, liver and kidneys after acute high exposures, methylene chloride concentrations are most often highest in the brain after acute lethal concentrations.

Liver and lung effects were seen in an acute inhalation study in rodents but at higher concentrations and lung effects appeared to be transient ([Shell Oil, 1986](#)). Immunosuppressive effects were observed in rats after acute exposure to 100 ppm, a lower air concentration than the levels associated with CNS effects observed in human studies ([Aranyi et al., 1986](#)). However, immune effects were not considered for dose-response analysis because data are sparse and inconclusive when considered along with the human data on immune system effects (see Section 3.2.3.1.3).

Overall, there is evidence to support adverse effects following acute methylene chloride exposure that include nervous system effects and the potential for adverse cardiac-related effects from increased COHb in people with underlying cardiac conditions or heart disease. Therefore, effects resulting from acute exposure were carried forward for dose-response analysis.

3.2.4.1.2 Liver Effects

Most human epidemiological studies did not investigate non-cancer liver effects. Of the identified studies that measured changes in liver enzymes, two found evidence of increased serum bilirubin ([General Electric Co, 1990](#); [Ott et al., 1983a](#)). GE ([1990](#)) received a data quality rating of medium.

Both inhalation and oral studies identified liver effects as sensitive non-cancer effect linked with exposure to methylene chloride in animals. Vacuolization, necrosis, hemosiderosis and hepatocellular degeneration have been identified in subchronic and chronic inhalation studies in rats, mice, dogs and monkeys ([Mennear et al., 1988](#); [Nitschke et al., 1988a](#); [NTP, 1986](#); [Burek et](#)

²⁰ [Tomenson \(2011\)](#), [Lanes et al. \(1993\)](#) and [Hearne and Pifer \(1999\)](#) did not identify an increased risk of mortality from cerebrovascular disease or ischemic disease in three cohorts of workers producing cellulose triacetate film/fiber. These studies received data quality scores of medium (1.7), medium (1.8) and high (1.6), respectively.

[al., 1984](#); [Haun et al., 1972](#); [Haun et al., 1971](#)). A newer study ([Aiso et al., 2014a](#)) identified acidophilic and basophilic foci in rats but not mice after chronic inhalation exposure. An oral study also identified altered liver foci ([Serota et al., 1986a](#)). In both studies, liver foci were not correlated with tumors, and thus, EPA considers them to be non-neoplastic. Studies received high and medium data quality ratings.

Fatty liver, a more severe effect compared with vacuolization, was seen in rats and dogs ([Haun et al., 1972](#); [Haun et al., 1971](#)); oral studies also identified fatty liver in mice and rats ([Serota et al., 1986a, b](#)). Based on these fatty liver changes that can be considered a more severe effect and progression from vacuolization, U.S. EPA ([2011](#)) suggested that vacuolization should be considered toxicologically adverse and not simply an adaptive change.

U.S. EPA ([2011](#)) noted that limited MOA studies are available for methylene chloride regarding non-cancer liver effects. Information identified in the post-IRIS literature search is also limited and does not offer significant insight into the MOA as it relates to non-cancer liver toxicity. A specific MOA cannot be discerned from the changes in gene and protein expression measured in several studies ([Park and Lee, 2014](#); [Kim et al., 2013](#); [Kim et al., 2010](#)). Although Chen ([2013](#)) identified increased biliary excretion of GSH and increased bile secretion, again, it is not clear how these changes inform the vacuolization, necrosis and other apical effects observed in animal studies. [Dzul-Caamal et al. \(2013\)](#) identified lipid peroxidation and oxidation of proteins in livers of fish exposed to methylene chloride. Lipid peroxidation affects lipids directly but can also produce electrophiles and free radicals that can react with DNA and proteins ([Gregus, 2008](#)).

Overall, based on limited human evidence and evidence in multiple animal species from highly rated studies, there is evidence to support non-cancer liver effects following methylene chloride exposure. Therefore, this hazard was carried forward for dose-response analysis.

3.2.4.1.3 Immune System Effects

Overall, human, animal and mechanistic studies provide suggestive evidence of methylene chloride's association with immune-related outcomes. Appendix M presents a detailed evidence integration analysis of immune system effects.

Among the epidemiological studies, which received medium to high confidence ratings, three studies suggested an association between methylene chloride and immune-related, or possible immune-related, outcomes. Chaigne, et al. ([2015](#)) identified high-magnitude ORs spanning 9-11 (95% CI: 2.38-51.8) for methylene chloride's association with Sjogren's syndrome, an autoimmune disorder. Radican et al. ([2008](#)) also identified a high magnitude HR of 9.21 (95% CI: 1.03-82.7) for increased mortality from bronchitis, a less specific and not clearly immune-related endpoint. Finally, [hoechst celanese corp \(1992\)](#) found some elevation of mortality from flu and pneumonia associated with methylene chloride exposure (SMR 1.25 for males and 4.36 for females) that was not statistically significant. Despite these suggested associations, all studies had limited information on methylene chloride exposure, none controlled for other chemicals and Radican et al. ([2008](#)) investigated a non-specific outcome and used exposed and comparison populations with very different socioeconomic status. Given these limitations, the epidemiological studies were not used to estimate a quantitative dose-response relationship.

Two additional epidemiological studies found no or decreased associations with methylene chloride. Hearne and Pifer (1999) observed decreased mortality rates from infection or and Lanes et al. (1993) found no increase in mortality from non-malignant respiratory disease. These two studies used general population death rates and thus, the healthy worker effect²¹ may have resulted in attenuation of any possible association with methylene chloride.

Although one animal study is suggestive for immune-related effects, the body of scientific evidence from animals is limited. Aranyi et al. (1986), a medium quality study, investigated and identified increased mortality due to infection and impaired bacterial clearance and bactericidal activity. Warbrick et al. (2003), a high-quality study, found no differences in IgM antibody responses to sheep red blood cells among methylene chloride-exposed rats compared with controls. Warbrick et al. (2003) reported decreased spleen weights in female rats. NTP (1986) identified changes in the spleen (fibrosis and follicular atrophy of the spleen in rats and mice, respectively) but other chronic and subchronic inhalation studies didn't identify histopathological changes in spleens, lymph nodes, or thymi of rats. In addition, evidence is not available from other animal studies regarding changes in immune cell populations. Although there is some evidence for immunosuppression from Aranyi et al. (1986), EPA considers the database to be limited, with a lack of support from most other animal studies.

Data on modes of action are very limited. Methylene chloride may result in anti-inflammatory effects (as evidenced by changes in specific cytokines demonstrated by Kubulus et al. (2008)), but it has also been associated with generation of ROS in mononuclear cells (Uruga-Tovar et al., 2014). It is possible that multiple mechanisms may be at work, but with such limited data, EPA cannot conclude that methylene chloride has a specific MOA.

Overall there is some evidence to support immune system effects following methylene chloride exposure, but data are sparse with an apparent lack of consistency. Therefore, this hazard was not carried forward for dose-response analysis.

3.2.4.1.4 Nervous System Effects

CNS Depression and Spontaneous Activity

Based on the availability of multiple studies in humans and animals, CNS depression is a primary neurotoxic effect associated with methylene chloride. Mechanism studies are not definitive for this endpoint. Increased dopamine in the medulla and increased GABA and glutamate in the cerebellum by methylene chloride may be part of the MOA for these effects (Kanada et al., 1994); however, this study did not measure functional changes so firm conclusions regarding the MOA for CNS depression and motor changes are not possible. Studies have not been conducted to evaluate the neurochemical basis for changes in spontaneous activity for methylene chloride (Bale et al., 2011).

²¹ One aspect of the healthy worker effect is related to the fact that morbidity and mortality rates are generally lower in workers than the general population (Li and Sung, 1999), since the latter includes individuals who are unable to work due to illness.

Lash et al. (1991) identified decreased attention and complex reaction tasks among retired aircraft maintenance workers (data quality rating of medium). Although this study suggests a possible chronic nervous system effect, the effect was observed in only one study and was not statistically significant and so it is difficult to make conclusions from this study.

Although the MOA is not clearly delineated, multiple human and animal studies indicate that methylene chloride is associated with nervous system effects. Based on this evidence, EPA determined that methylene chloride should be brought forward for dose-response modeling. Specifically, CNS effects are brought forward for dose-response modeling of effects from acute/short-term exposure.

Developmental Neurotoxicity

Five epidemiological studies have evaluated the association between measured and modeled outdoor ambient air concentration estimates of many air pollutants (often starting with the 33-37 HAPs, although Roberts et al. (2013) investigated many more pollutants) and ASD for regions across the U.S. (Talbot et al., 2015; von Ehrenstein et al., 2014; Roberts et al., 2013; Kalkbrenner et al., 2010; Windham et al., 2006).

EPA has not advanced the ASD hazard to dose-response for several reasons. First, there are uncertainties in the modeled estimates of air concentrations from NATA. Specifically, the NATA data are annual average concentrations from the year of the pregnancy or within a few years of the pregnancy. However, an etiologically relevant time period of exposure for ASD is thought to be the perinatal period (Pelch et al., 2019; Kalkbrenner et al., 2010; Rice and Barone, 2000) and the lack of temporal specificity of the NATA data, especially when considering averages over multiple years, is a potential limitation. In addition, the estimates from these studies do not consider possible contribution of any unmeasured exposure by workers or indoor home exposures. Several of the current studies address multi-pollutant exposures within the same regression models but other studies only identify correlations among chemicals that are also independently associated with ASD. Therefore, certain methylene chloride odds ratios may be overstated in the studies that did not include these correlated chemicals in the same regression equation.

Animal studies identified effects on habituation, an early form of learning and memory, (Bornschein et al., 1980) and effects in other learning tests (Alexeeff and Kilgore, 1983) at high single concentrations following developmental exposure. However, these studies used only single high concentrations and were not considered appropriate to use in calculating risks.

Despite methodological limitations in the human studies and concentration limitations in the animal studies, the available information provides evidence of an association between methylene chloride exposure and developmental neurological effects.

3.2.4.1.5 Reproductive and Developmental Effects

Epidemiological studies sometimes identify reproductive/developmental effects, including oral cleft defects in mothers older than 35 years and heart defects in mothers of all ages (Brender et al., 2014) and spontaneous abortions (Taskinen et al., 1986). However, these studies didn't directly consider co-exposures within the same model as methylene chloride. Brender et al.

(2014) ran independent analyses with other chemicals, which showed associations in mothers of all ages or showed more positive associations. Taskinen et al. (1986) found that other chemicals resulted in similar magnitude of spontaneous abortions and furthermore, received a low data quality rating.

Some animal studies (Alexeeff and Kilgore, 1983; Bornschein et al., 1980; Hardin and Manson, 1980; Schwetz et al., 1975) identified effects that included developmental neurotoxicity but these were observed at higher concentrations (1,250, 4,500 or 47,000 ppm). Although Raje et al (1988) identified reduced fertility at 144 ppm, results failed to reach statistical significance in two of three statistical tests. Three oral reproductive/ developmental studies (Narotsky and Kavlock, 1995; Nitschke et al., 1988b; General Electric Company, 1976) didn't identify reproductive and developmental toxicity. Also, multiple animal studies used only a single concentration.

Some studies identify reproductive and developmental effects, including developmental neurotoxicity. Also, as noted in section 3.2.4.1.4, adults are sensitive to neurotoxicity and transfer of methylene chloride to the placenta is possible. Epidemiological studies lacked controls for co-exposures, animal studies observed effects mostly at higher methylene chloride concentrations in animals and EPA identified no relevant mechanistic information. Thus, EPA did not carry reproductive/developmental effects forward for dose-response.

3.2.4.1.6 Irritation/Burns

Data from case reports, an occupational study and animal data indicate that irritation is possible. Based on direct contact from accidents or suicide attempts, methylene chloride has been shown to result in burns to the eyes and skin (Fisk and Whittaker, 2018; ATSDR, 2000; Hall and Rumack, 1990). Gastrointestinal tract irritation is also expected, and was suggested in a suicide case, assuming methylene chloride was the causative agent (Hughes and Tracey, 1993). Irritation has been identified after inhalation of methylene chloride vapor in some cases (Anundi et al., 1993) but not others (Stewart et al., 1972).

Documentation that supports the OSHA (1997a) standard notes that methylene chloride may lead to a burning sensation if it remains on skin but notes that after short-term exposure, it is not corrosive. OSHA (1997a) states that individuals should avoid skin contact based on its irritating properties.

Based on data from humans and animals, there is evidence that methylene chloride is associated with irritation and possible burning of skin, eyes and mucous membranes. A full elucidation of the circumstances leading to irritation is not available because studies in humans are limited and it is not easy to quantify these effects. For these reasons, irritation and burns will not be carried forward for dose-response modeling but are qualitatively discussed in the risk characterization.

3.2.4.2 Genotoxicity and Carcinogenicity

There is sufficient evidence of methylene chloride carcinogenicity from animal studies. Methylene chloride produced tumors at multiple sites, in males and females, in rats and mice, by oral and inhalation exposure, and in multiple studies. The most prominent findings were significant increases in liver (hepatocellular adenoma/carcinoma) and lung (bronchoalveolar adenoma/carcinoma) tumor incidences in male and female B6C3F1 and Crj:BDF1 mice by

inhalation exposure in two separate bioassays ([Aiso et al., 2014a](#); [NTP, 1986](#)), liver tumors in male B6C3F1 mice exposed via drinking water ([Serota et al., 1986b](#); [Hazleton Laboratories, 1983](#)), and mammary gland tumors (adenoma/fibroadenoma) in male and female F344/N and F344/DuCrj rats exposed by inhalation in two separate bioassays ([Aiso et al., 2014a](#); [NTP, 1986](#)). Other findings potentially related to treatment included increases in liver tumors in male rats with inhalation exposure ([Aiso et al., 2014a](#)) and female rats with drinking water exposure ([Serota et al., 1986a](#); [Hazleton Laboratories, 1983](#)); hemangiomas/hemangiosarcomas in male and female mice by inhalation exposure ([Aiso et al., 2014a](#)); mononuclear cell leukemia in female rats by inhalation exposure ([Aiso et al., 2014a](#); [NTP, 1986](#)); mesotheliomas, subcutaneous fibromas/fibrosarcomas, and salivary gland sarcomas in male rats by inhalation exposure ([Aiso et al., 2014a](#); [NTP, 1986](#); [Burek et al., 1984](#)); and brain (glial cell) tumors in male and female rats by inhalation exposure ([Nitschke et al., 1988a](#)).

Although a number of relevant studies are available, findings were inconclusive for cancers of the liver, lung, breast, brain and CNS, and most hematopoietic cancer types, due to weaknesses of the individual studies and inconsistent results across studies. For these endpoints, the epidemiological studies provide only limited support for a relationship between methylene chloride exposure and tumor development.

While findings were also inconclusive for hematopoietic cancers (leukemia, multiple myeloma, Hodgkin lymphoma), including NHL, ORs for B-cell subtypes of NHL were consistently increased across all three case-control studies that evaluated this tumor type ([Barry et al., 2011](#); [Seidler et al., 2007](#); [Miligi et al., 2006](#)), and ranged from 1.6 to 3.2 with marginal statistical significance identified for two of the studies. Despite this greater consistency, the studies evaluating the B-cell subtypes did not adjust for other chemical co-exposures, and there was correlation among exposures for several chemicals. Furthermore, several chemicals showed some association with B-cell NHL. Thus, firm conclusions regarding the specific association between methylene chloride and the outcomes cannot be made.

Epidemiological studies inherently have limitations that decrease their ability to identify associations between outcomes and exposures. Although not a complete or exhaustive list, limitations regarding the epidemiological studies considered here and their ability to detect risks associated with methylene chloride are described here:

- 1) It is preferred that cohort studies use comparison (i.e. non-exposed) groups drawn from the same source population that are similar to the exposed groups to reduce the potential for selection bias. Most of the occupational cohort studies that evaluated risks by exposed workers to methylene chloride ([Tomenson, 2011](#); [Hearne and Pifer, 1999](#); [Gibbs et al., 1996](#); [Lanes et al., 1993](#)) used SMRs or standard incidence rates (SIRs), which use rates from the general population – whether working or not - as comparison groups. This may lead to the healthy worker effect, which results in selection bias and other types of biases, since the characteristics of the general population are likely to differ from the population of workers being evaluated (REFS). Morbidity and mortality rates are generally lower in workers than the general population ([Li and Sung, 1999](#)), since the latter includes individuals who are unable to work due to illness. According to Li and Sung ([1999](#)), some authors suggest that

the effect of these dissimilar groups (workers vs. general population) may be somewhat mitigated when considering mortality from cancer as an endpoint and for studies that included both active workers and retired individuals ([Hearne and Pifer, 1999](#)). The healthy worker survivor effect is another type of healthy worker effect that occurs when those who remain employed in the workforce are healthier than those who leave employment. This type of bias predominately serves to attenuate (bias towards the null value of no association) effect estimates related to the exposure(s) of interest. These types of comparisons can lead to other sources of bias beyond selection bias and may result in bias that is harder to gauge regarding direction and impact. It is likely that the effects of methylene chloride in several of these studies could be attenuated, such as in cohorts that use general population comparison groups or were subject to the healthy worker survivor effect.

- a. Ability to classify individuals by degree of exposure information was limited. For example, work histories were available for only 37% of the Lanes et al. ([1993](#)) cohort, and were not specific for 30% of the Tomenson et al. ([2011](#)) cohort. One study characterized methylene chloride exposure simply as yes/no ([Radican et al., 2008](#)). If exposure is misclassified, the results may be under or overpredicted. If misclassification is random, it is likely to underestimate effects, but if it is not random, effects may be under- or over-predicted ([Hennekens and Buring, 1987](#)).
- b. For lung cancer studies, smoking restrictions at work ([Tomenson, 2011](#); [hoechst celanese corp, 1992](#)) limits the ability to interpret the inverse association because of the potential for higher smoking rates in the general population. Lack of information/adjustment regarding smoking ([Lanes et al., 1993](#)) also limits the ability to interpret results. Some of these results may also be compounded by the aforementioned healthy worker effect.
- c. Low numbers of deaths or cases in several studies decrease study sensitivity making it difficult to detect an effect or interpret results. Examples include Hearne and Pifer ([1999](#)), Tomenson ([2011](#)), Radican ([2008](#)) and Christensen et al. ([2013](#)).

Some effects attributed to methylene chloride in epidemiological studies might instead be due to confounding. For example, if epidemiological studies did not control for exposures or report exposure information for other chemicals that are both positively associated with methylene chloride and cancer, adverse associations with methylene chloride may be overstated. For example, Miligi et al. ([2006](#)), Barry et al. ([2011](#)) and Seidler et al. ([2007](#)) identified some association between methylene chloride and B cell NHL but did not control for other chemical exposures. However, the only occupational epidemiological study to examine the impact of solvent co-exposure showed that multi-chemical adjustment only slightly changed the ORs ([Miligi et al., 2006](#)).

One set of data suggesting a cancer MOA are the multiple studies indicating mutagenicity associated with methylene chloride metabolites of the GST metabolic pathway catalyzed by the GSTT1 isoenzyme ([U.S. EPA, 2011](#)). There are numerous genotoxicity tests showing positive results for methylene chloride, including assays for mutagenicity in bacteria and mutagenicity,

DNA damage, and clastogenicity in mammalian tissues in vitro and in vivo ([IARC, 2016](#); [U.S. EPA, 2011](#)).

The most strongly positive results in mammalian tissues in vivo and in vitro were found in mouse lung and liver, tissues with the greatest rates of GST metabolism and the highest susceptibility to methylene chloride-induced tumors. To further strengthen the case for the role of GST-mediated metabolism, studies have demonstrated increases in damage with the addition of GSTT1 to the test system and decreases in damage by addition of a GSH depletory. The GSTT1 metabolic pathway has been measured in human tissues with activities that are generally lower than rodents. In addition, human cells have exhibited genotoxicity without exogenous addition of GSTT1 ([U.S. EPA, 2011](#)).

When comparing metabolism of methylene chloride by the GST pathway in liver and lung tissues among species, mice are more active than rats, humans and hamsters ([U.S. EPA, 2011](#)). Similarly, Thier et al. ([1998](#)) cited by U.S. EPA ([2011](#)) found species' specific liver GSTT1 isozyme activity after methylene chloride exposure to be ordered as follows (from highest to lowest): mice, rats, human high and low conjugators, hamsters and human non-conjugators.

Thier et al. ([1998](#)) cited by U.S. EPA ([2011](#)) also reported that high and low human conjugators exhibited GSTT1 activities in erythrocytes approximately 11 and 16 times higher, respectively, than the human liver activities of high and low conjugators. Furthermore, the human high conjugator GSTT1 activity in erythrocytes was the same as male mouse liver activity and 61% of the female mouse liver activity. Increased GSTT1 activity in some human tissues may be partly responsible for the observed associations between increased methylene chloride exposure and cancer incidence in certain epidemiological studies.

Based on the evidence, EPA believes that the cancer results in animal studies are relevant to humans. Reasons include the demonstration of mutagenicity in human cells without exogenous GSTT1 and detected GSTT1 activity in human cells, some of which is comparable to GSTT1 activity in mice.

Other possible MOAs are either not well established or have limited or no support. Andersen et al. ([2017](#)) identified the altered association of reduced oxygenation to both circadian cycle proteins and tissue metabolism as the likely MOA for tissue responses to methylene chloride. Changes in circadian rhythm have been associated with cancer, and some research also links hypoxia to changes in the circadian clock. IARC ([2019](#)) assigned night shift work as Group 2A, probably carcinogenic to humans. IARC ([2019](#)) also suggested that the mechanistic evidence included enhanced inflammation in rats; increased cell proliferation in transplanted tumors associated with light-dark schedule changes; and immune suppression in nocturnal rats, mice and Siberian hamsters. Altered tumor glucose metabolism was observed in female nude rats, consistent with the Warburg effect (glucose fermentation in cancer cells) ([Iarc, 2019](#)). In addition to the link between changes in the circadian clock and cancer, hypoxia has been shown to result in some changes in the circadian clock ([Andersen et al., 2017](#)).

However, certain mechanistic steps identified by IARC ([2019](#)) have not been established for methylene chloride. In particular, enhanced cell proliferation was either not observed in livers of

mice after 78 weeks (Foley et al., 1993) as cited in U.S. EPA (2011), or proliferation from acute and short-term exposure was not sustained after longer (83-93 days) exposure (Casanova et al., 1996; Foster et al., 1992) as cited in U.S. EPA (2011). In addition, although methylene chloride has been associated with immunosuppression (Aranyi et al., 1986), EPA has concluded that the evidence is limited. Furthermore, EPA did not identify an established adverse outcome pathway (AOP) describing the molecular initiating and key events for hypoxia leading to changes in the circadian clock and then subsequently to cancer.

U.S. EPA (2011) also evaluated sustained cell proliferation as an alternative MOA for methylene chloride-induced lung and liver cancer. Enhanced cell proliferation was not observed in the liver of female B6C3F1 mice exposed to 2000 ppm methylene chloride for up to 78 weeks (Foley et al., 1993) as cited in U.S. EPA (2011). Furthermore, acute and short-term inhalation studies showed enhanced cell proliferation in the lung; however, this effect was not sustained for longer exposure durations (83-93 days of exposure) (Casanova et al., 1996; Foster et al., 1992) as cited in U.S. EPA (2011). Also, data were not identified suggesting additional MOAs (e.g., peroxisome proliferation resulting from PPAR- α activation).

Although Andersen et al. (2017) provides an interesting hypothesis, EPA believes that the evidence for the MOA and specific information for methylene chloride are lacking. Furthermore, based on the identified additional biochemical and mechanistic data, EPA doesn't expect sustained cell proliferation to be important in the development of liver and lung tumors and no other receptor-mediated mechanistic information was identified. Therefore, U.S. EPA (2005a) indicates the need for a well-established MOA to consider deviating from the default methods of linear low-dose extrapolation.

In accordance with U.S. EPA (2005a) *Guidelines for Carcinogen Risk Assessment*, methylene chloride is considered "likely to be carcinogenic to humans" based on sufficient evidence in animals, limited supporting evidence in humans, and mechanistic data showing a mutagenic MOA relevant to humans. Therefore, this hazard was carried forward for dose-response analysis.

3.2.5 Dose-Response Assessment

3.2.5.1 Selection of Studies for Dose-Response Assessment

EPA evaluated data from studies described in Sections 3.2.3 and 3.2.4 to characterize the dose-response relationships of methylene chloride and selected studies and endpoints to quantify risks for specific exposure scenarios. The selected studies had adequate information to select PODs.

3.2.5.1.1 Toxicity from Acute/Short-Term Exposure

Based on the weight of scientific evidence evaluation, one health effect domain (CNS depression) was selected for dose-response analysis for effects from acute/short-term exposure. Information from human studies (controlled experiments) are available for this endpoint.

CNS Depression

As discussed in Section 3.2.3.1.1, several controlled experiments in humans are available that support the relationship between methylene chloride exposure and CNS effects. Although data

quality evaluation criteria are not available for the types of human studies considered, EPA qualitatively evaluated studies used as the basis for the American Conference of Government Industrial Hygienists (ACGIH) Threshold Limit Value (TLV)-TWA, California REL, SMAC, and other studies identified in backwards searching of these documents. Data are also available from animal studies to support this health effect domain during acute exposure, but the human studies are considered adequate and are preferable to animal studies.

A primary consideration for choosing studies for dose-response assessment includes use of objective tests (such as visual evoked responses) that measure CNS effects, and not simply subjective reports of symptoms, especially when it is not known whether the investigator and participants are blinded to the use of methylene chloride vs. control. Another consideration is appropriate generation of methylene chloride air concentrations. Finally, EPA determined that the changes in CNS effects are likely to be related not only to hypoxia from increased COHb levels but also from increased levels of methylene chloride concentrations in the brain; therefore, EPA placed greater importance on studies that identified effects from direct methylene chloride exposure, not effects modeled from COHb levels. Although COHb can continue to rise after exposure has ceased and thus COHb may still be relevant at longer time points, both Putz et al. (1979) and Winneke (1974) were conducted for 3.8 or 4 hrs and identified greater effects from methylene chloride compared to CO (and Winneke (1974) did not identify effects from CO). Thus, EPA considers direct CNS effects from methylene chloride to still be relevant for an 8-hr duration.

Based on these considerations, EPA chose Putz et al. (1979) to estimate risks from acute/short-term exposure. This study identified changes in visual peripheral response after 1.5 hrs (within a 4-hr exposure) in a dual complex task, adequately generated methylene chloride exposures and used a double-blind procedure. The study received a medium confidence rating. Although Winneke (1974) also identified similar effects from methylene chloride intake, the study did not test concentrations lower than 300 ppm. Because Putz et al. (1979) identified effects at a concentration not evaluated in other similar studies (195 ppm) and because CNS effects are critical effects that lead to more severe effects at higher concentrations and longer exposure durations, EPA chose Putz et al. (1979) for dose-response modeling for this endpoint.

3.2.5.1.2 Toxicity from Chronic Exposure

Non-Cancer

Hepatic effects are the primary dose-dependent non-cancer effects observed in animals after chronic and subchronic exposure to methylene chloride. Although a few other sensitive effects are observed for other health domains (e.g., some persistent nervous system effects in humans observed by Lash et al. (1991), decreased fertility identified by Rajee et al. (1988)), liver effects are more consistently observed. The hazard identification and weight of evidence sections (Section 1.5 and 3.2.1) both describe the evidence in more detail for each of these health domains.

EPA is relying on the dose-response modeling results presented in U.S. EPA (2011) from Nitschke (1988a) for rats. This study is the most suited to dose-response modeling because it is the chronic study with the lowest exposure concentrations and was rated high for data quality.

As a comparison, EPA also considered results from the recent study by Aiso et al. ([2014a](#)) in rats. However, the concentrations used in Aiso et al. ([2014a](#)) are higher (0, 3500, 7000 and 14,000 mg/m³) than the concentrations in the Nitschke et al. ([1988a](#)) study (0, 180, 700 and 1800 mg/m³).

The effects used in the dose-response modeling from both the Nitschke ([1988a](#)) and Aiso et al. ([2014a](#)) studies are included in Table 3-15.

Table 3-15. Candidate Non-Cancer Liver Effects for Dose-Response Modeling

Target Organ/System	Study Type	Species/Strain/Sex (Number/group)	Exposure Route	Doses/Concentration	Duration	NOAEL/LOAEL reported by authors	NOAEL/LOAEL (mg/m ³ or mg/kg-day) (Sex)	Effect	References	Data Quality Evaluation
Hepatic	Chronic	Rat, Sprague Dawley, M/F (n=180/group)	Inhalation, vapor, whole body	0, 176, 702 or 1755 mg/m ³ (0, 50, 200 or 500 ppm)	6 hours/day, 5 days/week for 2 years	NA	NOAEL= 702 (F)	Hepatic lipid vacuolation and multinucleated hepatocytes	Nitschke et al. (1988a)	High
Hepatic	Chronic	Rat, F344/DuCrj	Inhalation, vapor, whole body	0, 3510, 7019 or 14,038 mg/m ³ (0, 1000, 2000 or 4000 ppm)	6 hours/day, 5 days/week for 2 years	NA	LOAEL = 3510 mg/m ³ (F)	Increased basophilic foci and increased abs/rel liver wt (p < 0.01)	Aiso et al. (2014a)	High

Cancer

The epidemiological studies generally provide only limited support for the relationship between methylene chloride exposure and tumor development. Therefore, EPA relied on inhalation rodent cancer bioassays to model the dose-response relationship. EPA modeled both the tumor response data from NTP (1986) and data from a recent publication (Aiso et al., 2014a).

EPA modeled the same tumor response data from NTP (1986) chosen for the inhalation unit risk (IUR) as was modeled by U.S. EPA (2011), (i.e., liver, lung and mammary gland tumors). EPA also included modeling with the full set of dichotomous models available in benchmark dose software (BMDS) to evaluate the sensitivity of the model output to the model choice.

EPA also modeled dose-response data for several tumor types from a study published subsequent to the IRIS assessment (Aiso et al., 2014a). The tumors modeled included those with positive trend tests, significant pairwise differences from controls, the most sensitive tumors as well as the clearest dose-response data. EPA modeled lung and liver tumors in male and female mice. In rats, EPA modeled mammary and subcutis tumors. Although EPA could have included tumor types that had positive trend without statistically significant pairwise comparisons (similar to the evaluation by U.S. EPA (2011)), the excluded tumor types exhibited lower incidences and the dose-response relationships were generally unclear upon visual inspection. EPA provides more information on why certain tumor types were not modeled in Appendix B of the supplemental file *Methylene Chloride Benchmark Dose and PBPK Modeling Report* (EPA, 2019h).

NTP (1986) showed a clear dose-response with lung and liver cancer, and these data were chosen for dose-response modeling (U.S. EPA, 2011). Furthermore, the study received a high data quality rating using the criteria specified in *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018b). Of the inhalation studies and tumor types considered, these tumors were most sensitive to methylene chloride exposure in mice, yielding responses of greater magnitude and more positive association than most other tumor data, other than the mostly benign mammary tumors results (see Section 3.2.3.2.2).

Table 3-16. Candidate Tumor Data for Dose-Response Modeling presents tumor results from the NTP (1986) and Aiso et al. (2014a) studies that were considered to be candidates for dose-response modeling.

Table 3-16. Candidate Tumor Data for Dose-Response Modeling

Reference	Strain and Species	Exposure route	Sex	Exposure levels	Tumor type	Significant dose-related trend	Significant pairwise comparison ^a	Exposure level with significant increase ^a	Data Quality Evaluation
<i>Hepatic Tumors</i>									
NTP (1986)	B6C3F1 mouse	Inhalation	M	0, 2000, 4000 ppm	Hepatocellular adenoma or carcinoma	✓	✓	4000 ppm	High
			F		Hepatocellular adenoma or carcinoma	✓	✓	≥ 2000 ppm	
Aiso et al. (2014b)	BDF1 mouse	Inhalation	M	0, 1000, 2000, 4000 ppm	Hepatocellular adenoma or carcinoma	✓	✓	≥ 2000 ppm	High
					Hepatic hemangioma	✓	✓	4000 ppm	
					Hepatic hemangioma or hemangiosarcoma	✓	-	-	
			F		Hepatocellular adenoma or carcinoma	✓	✓	≥ 1000 ppm	
					Hepatic hemangioma	✓	-	-	
					Hepatic hemangioma or hemangiosarcoma	✓	-	-	
<i>Lung Tumors</i>									
NTP (1986)	B6C3F1 mouse	Inhalation	M	0, 2000, 4000 ppm	Bronchoalveolar adenoma or carcinoma	✓	✓	≥ 2000 ppm	High
			F		Bronchoalveolar adenoma or carcinoma	✓	✓	≥ 2000 ppm	
Aiso et al. (2014b)	BDF1 mouse	Inhalation	M	0, 1000, 2000, 4000 ppm	Bronchoalveolar adenoma or carcinoma	✓	✓	≥ 1000 ppm	High
			F		Bronchoalveolar adenoma or carcinoma	✓	✓	≥ 2000 ppm	

Reference	Strain and Species	Exposure route	Sex	Exposure levels	Tumor type	Significant dose-related trend	Significant pairwise comparison ^a	Exposure level with significant increase ^a	Data Quality Evaluation
Mammary Tumors									
NTP (1986)	F344 rat	Inhalation	M	0, 1000, 2000, 4000 ppm	Mammary or subcutaneous tissue adenoma, fibroadenoma, or fibroma	✓	✓	4000 ppm	High
			F		Mammary adenoma, fibroadenoma, or adenocarcinoma	✓	✓	≥ 2000 ppm	
Aiso et al. (2014b)	F344/DuCrj	Inhalation	M	0, 1000, 2000, 4000 ppm	Mammary gland fibroadenoma	✓	✓	4000 ppm	High
					Mammary gland fibroadenoma or adenoma	✓	✓	4000 ppm	
					Mammary gland fibroadenoma or adenoma or adenocarcinoma	✓	-		
			F		Mammary gland fibroadenoma	✓	-		
					Mammary gland fibroadenoma or adenoma	✓	-		
					Mammary gland fibroadenoma or adenoma or adenocarcinoma	✓	-		
Subcutaneous Tumors									
Aiso et al. (2014b)	F344/ DuCrj	Inhalation	M	0, 1000, 2000, 4000 ppm	Subcutaneous fibroma	✓	✓	≥ 2000 ppm	High
					Subcutaneous fibroma or fibrosarcoma	✓	✓	≥ 2000 ppm	

^aAs reported in the cited reference

3.2.5.2 Derivation of PODs and UFs for Benchmark Margins of Exposures (MOEs)

3.2.5.2.1 PODs for Acute/Short-term Inhalation Exposure

Workers and consumers can be exposed to a single acute exposure to methylene chloride under various conditions of use via inhalation and dermal routes. EPA identified PODs for several acute inhalation exposure durations based on both hazard and exposure considerations. A duration of 8 hrs, a typical work shift, is used for occupational settings. For workers, EPA also evaluated a 15-minute exposure, which matches the duration used to set the STEL. Furthermore, some concentrations of methylene chloride in occupational settings are reported for 15 minutes or similar durations.

A 1-hr value is used for consumer settings, which is similar to the length of time (1.5 hrs) after which effects were observed by Putz et al. (1979).

Putz et al. (1979) is a well-conducted study of 12 volunteers that identified decreased visual peripheral performance after 1.5 hr of exposure to 195 ppm (200 ppm nominal). Results of EPA's qualitative data quality evaluation indicate that this study is of medium quality and unlike other key studies that have been evaluated, Putz et al. (1979) conducted his study in a double-blind manner. Because this study used a single concentration, it is not amenable to dose-response modeling, so EPA used the LOAEC of 195 ppm. Both OSHA and ACGIH cited the nominal value of 200 ppm as a LOAEC for CNS effects. ACGIH used this study with a safety factor of 4 to account for interindividual differences in sensitivity and use of a LOAEC rather than a NOAEC as the basis of its 8-hr TLV-TWA of 50 ppm.

The Office of Environmental Health Hazard Assessment (OEHHHA) from the state of California uses Putz et al. (1979) as the basis of their REL. OEHHHA (2008a) used a simplified equation, $C^n \times T = K$ with $n = 2$, to scale the LOAEC of 195 ppm (696 mg/m^3) for 1.5 hrs to values of 240 ppm (840 mg/m^3) and 80 ppm (290 mg/m^3) for 1 and 8 hours, respectively. This equation is a modification of Haber's rule, and $n = 2$ is based on an analysis by ten Berge et al. (1986), of concentration times time for lethality data from 20 acute inhalation studies of various compounds that resulted in an average value of 1.8 for n . OEHHHA (2008a) used a total UF of 60 based on an intraspecies UF of 10 to account for human variability and a LOAEL-to-NOAEL UF of 6 (Oehha, 2008a).

The NAC/AEGL has used $C^n \times T = K$ when setting AEGLs and has also used $n = 2$ when no exposure-versus-time data are available (NASEM (National Academies of Sciences, 2000)). Although there is uncertainty in using $n=2$ to extrapolate to longer time periods, ten Berge et al. (1986) identified the value of $n = 1.8$ from LC_{50} studies, which typically are 4 hours long. Thus, it was considered appropriate to use this for an 8-hour period.

For methylene chloride, exposure-versus-time data are limited. Therefore, EPA considers the ten Berge equation using $n = 2$ as a valid method to convert the 1.5 hour POD value from Putz et al. (1979) to the 15-minute, 1-hour and 8-hour PODs (see Table 3-17).

Although EPA considered using the PBPK model described by [Bos et al. \(2006\)](#), EPA believes that there are enough uncertainties regarding the assumptions, validation and precision of the model that don't warrant using it instead of the ten Berge equation. Although the model accounts for P-450 saturation and a switch to conjugation catalyzed by GSTT1, P450 saturation occurs at approximately 500 ppm, which is higher than the POD for the current evaluation. In addition, although the model includes the distribution of GSTT1 in the population, EPA considered this refinement less necessary when using human volunteers, especially at lower methylene chloride concentrations. Furthermore, the parent compound has been shown to result in CNS effects that are in excess of CO/COHb concentrations. However, [Bos et al. \(2006\)](#) acknowledge that there are no adequate data on methylene chloride in rat or human brains and also assume that at longer exposures, the more relevant endpoint is COHb only. OSHA, when considering a similar PBPK model for acute effects for derivation of the 1997 PEL, had similar concerns about the lack of experimental validation of the predicted brain MC concentrations ([OSHA, 1997a](#)). In addition, although EPA understands that the COHb concentrations may be maintained for several hours after exposure ceases (and a primary reason to consider this type of PBPK model), this effect is not as pronounced at lower concentrations. Finally, [Bos et al. \(2006\)](#) state that the model overpredicts methylene chloride and COHb concentrations by up to 50%. Thus, although the PBPK model has features that may be important for setting other limits set higher values, such as AEGLs, EPA considers the ten Berge equation to be appropriate for the current risk evaluation.

Table 3-17. Conversion of Acute PODs for Different Exposure Durations

Exposure Duration for Value	POD	UFs for Benchmark MOE ^{a,b}	Endpoint	References
15-min	478 ppm (1706 mg/m ³)	UF _H = 10 UF _L = 3	7% ↓ visual peripheral performance at 1.5 hrs	CNS data from Putz et al. (1979); Conversion of concentrations among exposure durations use ten Berge et al. (1986) equation $C_n \times T = K$, where $n = 2$
1-hr	240 ppm (840 mg/m ³)	Total UF = 30		
8-hr	80 ppm (290 mg/m ³)			

a. Margin of Exposure (MOE) = Non-cancer POD / Human exposure

b. UF_H = intraspecies uncertainty factor; UF_L = LOAEL-to-NOAEL uncertainty factor

EPA applied a composite UF of 30 for the acute inhalation benchmark MOE, based on the following considerations:

1) Interspecies uncertainty/variability factor (UF_A) of 1

Accounting for differences between animals and humans is not needed because the POD is based on data from humans

2) A default intraspecies uncertainty/variability factor (UF_H) of 10

To account for variation in sensitivity within human populations due to limited

information regarding the degree to which human variability may impact the disposition of or response to, methylene chloride.

a. Some of the specific variabilities/uncertainties for methylene chloride that can lead to greater risk and are accounted for with this UF_H include toxicokinetic differences:

Fetuses

Fetuses are at higher risk for CO toxicity and resulting CNS effects because of higher CO affinity for hemoglobin and slower CO elimination ([Nrc, 2010](#)). There are no studies reporting effects on the unborn after a single acute exposure resulting in lower COHb levels ([Nrc, 2010](#); [U.S. EPA, 2000](#)).

Workers, consumers engaged in vigorous activity

It has been shown that greater metabolism to CO occurs in individuals who are exercising ([Nac/Aegl, 2008b](#)). This leads to increased COHb and subsequent effects that may exacerbate the CNS effects. Workers or consumers who are engaged in more vigorous activity would be expected to exhibit greater effects due to additional CNS effects of increased COHb. In addition, exercise increases the rates of respiration and cardiac output, both of which are important in increasing systemic uptake of VOCs such as methylene chloride.

Individuals with higher CYP2E1 enzyme levels

Several other chemicals, including alcohol, can induce CYP 2E1 and lead to greater metabolism that leads to increased CO and COHb levels. Thus, individuals who consume large amounts of alcohol may be at greater risk.

Smokers

Smokers have higher levels of COHb and therefore, additional increases in COHb from methylene chloride exposure may lead to increased CNS effects or increased angina in individuals with heart disease.

b. Some of the specific variabilities/uncertainties related to toxicodynamic differences based on potentially susceptible subpopulations are as follows:

Individuals with heart disease/cardiac patients

At COHb levels of 2 or 4%, patients with coronary artery disease may experience a reduced time until onset of angina (chest pain) during physical exertion ([Allred et al., 1991](#); [Allred et al., 1989a](#); [Allred et al., 1989b](#)). Other studies have also confirmed a reduced time to onset of exercise-induced chest pain at a COHb between 2.5 and 4.5 percent ([Kleinman et al., 1998](#); [Kleinman et al., 1989](#); [Sheps et al., 1987](#); [Anderson et al., 1973](#); [Aronow et al., 1972](#)). The SMAC ([Nrc, 1996](#)) identified a NOAEC of 100 ppm for a 3% COHb level and because decreased time to angina may occur at even lower levels, this UF is considered important to account for this susceptible subpopulation. These values are lower than the value from Putz et al. ([1979](#)) used for the acute endpoint; the COHb level was measured as 5.1%.

c. Furthermore, additional differences among individuals that may result from either toxicokinetic or toxicodynamic differences may be of concern:

Bystanders of different ages

Residential bystanders for consumer uses are expected to be indirectly exposed to methylene chloride and may be of any age. For example, elderly individuals who may have other health concerns (e.g., those related to nervous system effects) may be more susceptible to the effects of methylene chloride from acute exposure.

3) A LOAEC-to-NOAEC uncertainty factor (UFL) of 3

This factor was applied to account for the lack of NOAEC in the critical study. A value of 3 rather than a more conservative value of 10 is applied because the effects observed by Putz et al. (1979) after one and one-half hours are of a small magnitude (decreased 7% in one measure – visual peripheral changes).

3.2.5.2.2 PODs for Chronic Inhalation Exposure

Chronic exposure was defined for occupational settings as exposure reflecting a 40-hour work week. A set of dichotomous dose-response models that are consistent with a variety of potentially underlying biological processes were applied to empirically model the dose-response relationship in the range of the observed data. The models in EPA's BMDS were applied to selected studies. Consistent with EPA's *Benchmark Dose Technical Guidance Document* (EPA, 2012a), the BMD and 95% lower confidence limit on the BMD (BMDL) were estimated using a benchmark response (BMR) to represent a minimal, biologically significant level of change, referred to as relative deviation (RD). In the absence of information regarding the level of change that is considered biologically significant, a BMR of 10% extra risk (ER) for dichotomous data is used to estimate the BMD and BMDL, and to facilitate a consistent basis of comparison across endpoints and studies. The estimated BMDLs were used as PODs; the PODs are summarized in Table 3-19 for non-cancer liver effects and in Table 3-20 includes information for cancer endpoints. Details on derivation of the IUR for cancer and the non-cancer HEC are included in Appendix I. More information and the full suite of models, model outputs and graphical results for the model selected for each endpoint can be found in *Supplemental File: Methylene Chloride Benchmark Dose and PBPK Modeling Report* (EPA, 2019h).

Non-Cancer Liver Effects

U.S. EPA (2011) modeled the dose response relationships for liver vacuolation in female rats using a modified PBPK model from Andersen et al. (1991). Female rats were used based on a higher response and because data were available for the lower dose groups. The PBPK model was used to calculate average daily internal liver doses.

U.S. EPA (1980) investigated four dose metrics (hepatic metabolism through the CYP pathway, GST pathway or combined hepatic metabolism through both pathways, and the concentration (AUC) of methylene chloride in the liver). Adequate model fits were observed for GST, CYP and AUC for inhalation data. However, the GST and AUC metrics produced inconsistencies in dose-response relationship depending on route of exposure. However, these inconsistencies were not observed using the CYP metric. Therefore, EPA used the internal dose metric based on total

hepatic metabolism through the CYP2E1 pathway (as mg methylene chloride metabolized via CYP pathway/L liver/day).

U.S. EPA (2011) used seven dichotomous dose-response models in EPA BMDS version 2.0 to fit to liver lesions incidence and PBPK model-derived internal dose data to obtain rat internal BMD₁₀ and BMDL₁₀ values. As noted above, a BMR of 10% was used given a lack of information on the magnitude of change thought to be minimally biologically significant. The log-probit model was the best fitting model. The comparison of BMDL₁₀s of internal doses from all seven models are presented in Table 3-18. More details are provided in U.S. EPA (2019h).

Table 3-18. Results of BMD Modeling of Internal Doses Associated with Liver Lesions in Female Rates from Nitschke et al. (1988a)

Model	BMD ₁₀	BMDL ₁₀	X ² Goodness of fit p-value	AIC
Gamma	622.10	227.29	0.48	367.24
Logistic	278.31	152.41	0.14	369.77
Log-logistic	706.50	506.84	0.94	365.90
Multistage (3)	513.50	155.06	0.25	368.54
Probit	279.23	154.52	0.14	369.76
Log-probit	737.93	531.82	0.98	365.82
Weibull	715.15	494.87	0.95	365.88

Source: U.S. EPA (2011), Table 5-6, pg. 193

AIC = Akaike information criterion

EPA obtained the human-equivalent internal BMDL₁₀ by dividing the internal rat dose metric by a pharmacokinetic scaling factor based on the ratio of BW^{3/4} (scaling factor of 4.09) because EPA lacked information on methylene chloride's pharmacokinetic differences between rats and humans. Use of BW^{3/4} represents EPA's general understanding that metabolic clearance scales allometrically across species. A probabilistic PBPK model for methylene chloride in humans was adapted from David et al. (2006) and used with Monte Carlo sampling to calculate distributions of chronic HECs (mg/m³) associated with the internal BMDL₁₀.

EPA used the 1st percentile to account for susceptibility from the toxicokinetic variability among humans related to differences in metabolism. Using the 1st percentile, EPA reduced the intraspecies uncertainty factor (UF_H) from 10 to 3. The remaining UF_H of 3 accounts for any toxicodynamic differences among humans. EPA's use of the human toxicokinetics data distribution is similar to using data-derived extrapolation factors (DDEFs) because it uses information more specific to methylene chloride hazard. DDEFs are suggested by agency guidance as preferable to default UFs (EPA, 2014b). The 5th percentile is very similar (21.3 mg/m³) to the 1st percentile (17.2 mg/m³). The mean is 48.5 mg/m³ (within an order of magnitude of 3 times higher than the 1st percentile).

Although EPA chose to use the HEC value modeled from Nitschke et al. (1988a), the HEC modeled from Aiso et al. (2014a) for basophilic cell foci is essentially the same as the value for vacuolation from Nitschke et al. (1988a) using the same PBPK models and similar assumptions. See Table 3-19 for the comparison of the modeled values.

Table 3-19. BMD Modeling Results and HECs Determined for 10% Extra Risk, Liver Endpoints from Two Studies

Internal dose metric ^a	Sex, Species	Endpoint	BMD model ^b	Animal BMDL ₁₀ ^{a,c}	Human BMDL ₁₀ ^{a,d}	Resulting HEC (mg/m ³) ^e	Reference
Liver CYP metabolism	Female rat	Vacuolation	log-probit	531.8	130.0	17.2 mg/m ³ [First percentile] ^f	Nitschke et al. (1988a) ^g
		Acidophilic cell foci	gam-r	645.5	157.4	98.2 mg/m ³	Aiso et al. (2014a)
		Basophilic cell foci	log	114.2	27.85	17.3 mg/m ³	

^a mg methylene chloride metabolized via CYP pathway /Liter of liver tissue /day

^b See BMD modeling report for model definitions and details.

^c Animal BMDL₁₀ refers to the BMD-model-predicted rat internal dose and its 95% lower confidence limit, associated with a 10% ER for the incidence of tumors; units are those for the identified dose metric, described in footnote “a”.

^d When the dose metric is the rate of production of the presumed toxic metabolite (mg/kg/d or mg/L/day), allometric scaling is applied to adjust for the fact that humans are expected to detoxify the metabolite more slowly than rats. A rat BMDL₁₀ divided by $(BW_{\text{human}}/BW_{\text{rat}})^{0.25} = 4.1$. Units are the same as for the Animal BMDL₁₀.

^e HEC is the 1st percentile of a distribution obtained by determining the exposure concentration for each individual in a simulated population that is predicted to yield an internal dose equal to the (internal) Human BMDL₁₀; with use of the 1st percentile the intra-human UF can be reduced from a standard value of 10 to 3, to account for remaining variability in pharmacodynamic sensitivity.

^f For comparison with 1st percentile the fifth percentile and mean values are 21.3 and 48.5 mg/m³, respectively.

^gResults of BMD modeling for this study are presented in U.S. EPA (2011).

EPA applied a composite UF of 10 for the chronic inhalation benchmark MOE, based on the following considerations:

1) Interspecies uncertainty/variability factor (UF_A) of 3

to account for species differences in animal to human extrapolation an interspecies uncertainty/variability factor of 3 (UF_A) was applied for toxicodynamic differences between species. This UF is comprised of two separate areas of uncertainty to account for differences in the toxicokinetics and toxicodynamics of animals and humans. In this assessment, the toxicokinetic uncertainty was accounted for by the PBPK modeling. As the toxicokinetic differences are thus accounted for, only the toxicodynamic uncertainties in extrapolating from animals to humans remain, and an UF_A of 3 is retained to account for this uncertainty.

2) Intraspecies uncertainty/variability factor (UF_H) of 3

to account for variation in sensitivity within human populations an intraspecies uncertainty/variability factor of 3 (UF_H) was applied for toxicodynamic differences in the human population. This UF is comprised of two separate areas of uncertainty to account

for variation in the toxicokinetics and toxicodynamics of the human population because humans of varying gender, age, health status, or genetic makeup might vary in response to methylene chloride. In this assessment, the toxicokinetic variation in humans was accounted for by the probabilistic PBPK model using Monte Carlo sampling of distributions for the following variables: physiological, tissue volume, partition coefficient and metabolism (including CYP 2E1) parameters. EPA selected the HEC associated with the first percentile among humans. As the toxicokinetic differences are thus accounted for, only the toxicodynamic variability in the human population remains, and an UF_A of 3 is retained to account for this variability.

3) A LOAEC-to-NOAEC uncertainty factor (UF_L) of 1

A BMDL, considered to be equivalent to a NOAEL(C) was calculated from Nitschke et al. (1988a) and therefore an UF of 1 is applied.

Cancer

EPA modeled dose-response relationships for tumor incidence in rodents observed in two studies, Aiso et al. (2014a) and NTP (1986), using the mouse PBPK model of Marino et al. (2006). Because metabolites of methylene chloride produced by the GST pathway are primarily responsible for methylene chloride carcinogenicity in mouse liver and lungs and based on the assumption that metabolites are reactive enough that they don't have substantial distribution outside the liver, the internal tissue-dose metrics used were daily mass of methylene chloride metabolized via the GST pathway per unit volume of liver and lung, respectively. When lung and liver tumors were combined to calculate BMDs and BMDLs for a holistic combination of tumors, a whole-body GST metric was used that essentially combined the lung and liver internal doses. Using species-specific information on GST activity in the PBPK models accounts for differences in GST and GSTT1 activity between mice and humans and among humans. Although the CYP pathway is considered important at lower concentrations, EPA assumed that there is some non-zero GSTT1 activity even at low concentrations because there is a possibility of reaction between methylene chloride and GST/GSH when these molecules are present.

For other tissues (subcutis and mammary gland), there is too little information to determine the relevant dose metric. For example, genotoxicity and mechanistic studies have not included mammary tissues. Therefore, these tumors were modeled using the estimated area under the curve (AUC) of methylene chloride from the Aiso et al. (2014a) data.

U.S. EPA (2011) also modeled the dose response from mammary tumors observed in NTP (1986) and details are presented in U.S. EPA (2011). Both NTP (1986) and Aiso et al. (2014a) observed mostly benign mammary tumors.

EPA obtained the human-equivalent internal $BMDL_{10}$ by dividing the internal mouse dose metric by a pharmacokinetic scaling factor based on the ratio of $BW^{3/4}$ (scaling factor of 7) because EPA lacked information on methylene chloride's pharmacokinetic differences between mice and humans. Use of $BW^{3/4}$ represents EPA's general understanding that metabolic clearance scales allometrically across species. A probabilistic PBPK model for methylene chloride in humans was adapted from David et al. (2006) and used with Monte Carlo sampling to calculate distributions of chronic HECs (mg/m^3) associated with the internal $BMDL_{10}$.

Table 3-20 presents the best model fits for several tumor types for multiple cancer endpoints from Aiso et al. (2014a) and for lung and liver tumors from NTP (1986). BMDL_{10S} of internal doses are presented along with IURs. In addition, the HECs for terminal bronchiole hyperplasia are also presented for context. Hyperplasia occurred at concentrations higher than lung tumors and is not expected to be a precursor to the tumors observed. See U.S. EPA (2019h) for other model results of the tumor types identified below.

Based on the results of these model fits, EPA chose to use the IUR of 1.38×10^{-9} per $\mu\text{g}/\text{m}^3$ based on NTP (1986) in the current risk evaluation because EPA determined that the combined liver and lung tumor response is relevant for humans and it is the most sensitive of the best-fitting models for the malignant tumors. Modeling the same tumor types using Aiso et al. (2014a) results in a very similar IUR of 1.30×10^{-9} per $\mu\text{g}/\text{m}^3$. Although mammary gland and subcutis tumors yielded higher IURs, there is less certainty about these tumors. The chosen IUR differs from the IUR of 1×10^{-8} per $\mu\text{g}/\text{m}^3$ recommended in the IRIS assessment (U.S. EPA, 2011) for two reasons. First, the current IUR is used only in the occupational assessment, and therefore, the value was adjusted from a 24-hr value to one applicable to a workweek of 8 hours per day, 5 days per week. Second, because the IUR is based on the lower 95% confidence limit, EPA considers the value to adequately include risk for the GSTT1 +/+ population and that the previous IUR was more conservative than necessary because it combined both the GSTT1 +/+ population and the lower 95% confidence limit.

Appendix I presents additional information regarding the dose-response modeling steps used to estimate the cancer slope, and the supplemental document *Methylene Chloride Benchmark Dose and PBPK Modeling Report* (EPA, 2019h) presents more details on the models used.

Table 3-20. BMD Modeling Results and Tumor Risk Factors/HECs Determined for 10% Extra Risk, Various Endpoints From Aiso et al. (2014a) and NTP (1986)

Internal dose metric ^a	Sex, Species	Endpoint (Asio study, unless “(NTP)”))	BMD model ^b	Animal BMDL ₁₀ ^{a,c}	Human BMDL ₁₀ ^{a,d}	Human tumor risk factor ^c	Mean human internal dose from 1 µg/m ³ exposure ^a		Resulting human IUR (µg/m ³) ⁻¹ or HEC (mg/m ³) ^f	
							Mixed population	GST +/-	Mixed population	GST +/-
Slowly perfused AUC (methylene chloride)	Male rat	Subcutis	lnp-ur	27.626	27.626	3.62×10^{-3}	1.59×10^{-5}	Not significantly different from mixed population	5.76×10^{-8}	Not significantly different from mixed population
			mst2-r	106.73	106.73	9.37×10^{-4}			1.49×10^{-8}	
		Mammary Gland (F/A)	log	266.06	266.06	3.76×10^{-4}			5.98×10^{-9}	
			mst1-r	205.35	205.35	4.87×10^{-4}			7.74×10^{-9}	
		Mammary Gland (F/A/AC)	log	267.16	267.16	3.74×10^{-4}			5.95×10^{-9}	
			mst1-r	222.31	222.31	4.50×10^{-4}			7.15×10^{-9}	
	Subcutis or Mammary Gland (F/A)	multi-tumor	78.802	78.802	1.27×10^{-3}	2.02×10^{-8}				
		Subcutis or Mammary Gland (F/A/AC)	multi-tumor	81.265	81.265	1.23×10^{-3}			1.96×10^{-8}	
	Female rat	Subcutis or Mammary Gland (F/A/AC)	pro	166.68	166.68	6.00×10^{-4}			9.54×10^{-9}	
			mst1-r	123.7	123.7	8.08×10^{-4}			1.29×10^{-8}	

Internal dose metric ^a	Sex, Species	Endpoint (Asio study, unless “(NTP)”))	BMD model ^b	Animal BMDL ₁₀ ^{a,c}	Human BMDL ₁₀ ^{a,d}	Human tumor risk factor ^e	Mean human internal dose from 1 µg/m ³ exposure ^a		Resulting human IUR (µg/m ³) ⁻¹ or HEC (mg/m ³) ^f	
							Mixed population	GST +/-	Mixed population	GST +/-
Liver GST	Male mice	Liver tumor	lnl-r	413.06	59.01	1.70×10^{-3}	6.65×10^{-7}	1.17×10^{-6}	1.13×10^{-9}	1.98×10^{-9}
			mst2-r	593.21	84.74	1.18×10^{-3}			7.58×10^{-10}	1.38×10^{-9}
		Liver tumor (NTP)	lnl-r	740.82	105.8	9.45×10^{-4}			6.28×10^{-10}	1.11×10^{-9}
			mst1-r	544.51	77.79	1.29×10^{-3}			8.55×10^{-10}	1.50×10^{-9}
	Female mice	Liver tumor	pro	1332.8	190.40	5.25×10^{-4}			3.49×10^{-10}	6.14×10^{-10}
			mst2-r	762.31	108.90	9.18×10^{-4}			6.11×10^{-10}	1.07×10^{-9}
Lung GST	Male mice	Lung tumor	pro	115.93	16.56	6.04×10^{-3}	4.39×10^{-8}	7.75×10^{-8}	2.65×10^{-10}	4.68×10^{-10}
			mst1-r	55.91	7.987	1.25×10^{-2}			5.50×10^{-10}	9.70×10^{-10}
		Lung tumor (NTP)	mst1-r	48.646	6.949	1.44×10^{-2}			6.32×10^{-10}	1.12×10^{-9}
	Female mice	Lung tumor	mst2-r	223.47	31.92	3.13×10^{-3}	4.39×10^{-8}	7.75×10^{-8}	1.38×10^{-10}	2.43×10^{-10}
		TB hyperplasia	mst3-r	411.28	58.75	n/a			7.75×10^4 mg/m ³	5.73×10^4 mg/m ³
Whole body GST	Male mice	Liver or lung tumor	multi-tumor	8.217	1.174	8.52×10^{-2}	1.53×10^{-8}	2.68×10^{-8}	1.30×10^{-9}	2.28×10^{-9}
		Liver or lung (NTP)		7.753	1.108	9.03×10^{-2}			1.38×10^{-9}	2.42×10^{-9}
	Female mice	Liver or lung tumor		25.302	3.615	2.77×10^{-2}			4.23×10^{-10}	7.41×10^{-10}

^a Tissue-specific dose-units = mg dichloromethane metabolized via GST pathway/L tissue (liver or lung)/day; whole-body dose units = mg dichloromethane metabolized via GST pathway in lung and liver/kg-day; AUC(methylene chloride) = mg-h/L tissue; all metrics are daily averages given a - week exposure per bioassay conditions (animal dosimetry) or 8 h/d, 5 d/w workplace exposure scenario (human dosimetry).

^b Models cited in the table include: lnl-r = Log-Logistic-restricted; lnp-ur = log-Probit-unrestricted; log = Logistic; mst1, 2 or 3 -r = Multistage-restricted (mst-r); from degree 1 to degree 3 (# dose groups – 1); multi-tumor = Multi-tumor (MS combo); pro = Probit; See the supplemental file *Methylene Chloride Benchmark Dose and PBPK Modeling Report (EPA, 2019h)* for additional details.

^c Animal BMDL₁₀ refers to the BMD-model-predicted mouse or rat internal dose and its 95% lower confidence limit, associated with a 10% ER for the incidence of tumors; units are those for the identified dose metric, described in footnote “a”.

^d When the dose metric is the rate of production of the presumed toxic metabolite (mg/kg/d), allometric scaling is applied to adjust for the fact that humans are expected to detoxify the metabolite more slowly than mice and rats. A mouse BMDL₁₀ is divided by $(BW_{\text{human}}/BW_{\text{mouse}})^{0.25} = 7$ and a rat BMDL₁₀ divided by $(BW_{\text{human}}/BW_{\text{rat}})^{0.25} = 4.1$. When the metric is the concentration (AUC) of a chemical, no adjustment is made. Units are the same as for the Animal BMDL₁₀.

^e Dichloromethane tumor risk factor (extra risk per unit internal dose) derived by dividing the BMR (0.1) by the allometric-scaled human BMDL₁₀. Units are 1/(BMDL₁₀ units) for corresponding tissues/endpoints.

^f Human inhalation risk is the product of the mean internal dose and the tumor risk factor. The HEC for the non-cancer response (hyperplasia) is the 1st percentile of a distribution obtained by determining the exposure concentration for each individual in a simulated population that is predicted to yield an internal dose equal to the (internal) Human BMDL₁₀.

3.2.5.2.3 Route to Route Extrapolation for Dermal PODs

EPA did not identify toxicity studies by the dermal route that were adequate for dose-response assessment. Dermal candidate values, therefore, were derived by route-to-route extrapolation from the inhalation PODs as introduced under Section 3.2.5.2 (Approach and Methodology). Inhalation studies were used because the toxic moieties are metabolites of methylene chloride; inhalation and dermal routes are similar because neither one includes a first pass through the liver (a site of high metabolic activity) before entering the general circulation. Furthermore, the inhalation studies are already used to calculate risks for the inhalation route.

Inhalation PODs were extrapolated using models that incorporate volatilization, penetration and absorption and use a methylene chloride permeability coefficient from an *in vitro* study ([Schenk et al., 2018](#)) using pig skin. See Section 2.4.2.3.1 and *Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment (EPA, 2019b)* for details regarding the models used.

The inhalation PODs were extrapolated using a POD based on either human data (i.e., acute exposures) or the BMDL_{HEC} (a value from animals adjusted to account for animal to human extrapolation using the PBPK model). The equations for extrapolating from inhalation PODs to the dermal route then must account for human inhalation and body weight:

For non-cancer effects:

$$\text{dermal POD} = \text{inhalation POD [mg/m}^3] \times \text{inhaled volume (m}^3) \div \text{body weight (kg)}$$

For cancer:

$$\text{dermal slope factor} = \text{IUR [per mg/m}^3] \div \text{inhaled volume (m}^3) \times \text{body weight (kg)}$$

where the inhaled volume was the ventilation rate 1.25 m³/hr (slightly higher than light activity) ([Niosh, 1976](#)) multiplied by the appropriate exposure duration (1.5 hours from Putz et al. ([1979](#))) for acute endpoints, or 20 m³ per day for the chronic endpoint) and a body weight of 80 kg ([EPA, 2011b](#)). Note that assuming a higher inhalation rate based on moderate intensity work for the purposes of route-to-route POD extrapolation would result in a higher POD that may not be appropriate or adequately health protective for all exposure scenarios.

PODs were derived from Putz et al. ([1979](#)) for a range of inhalation exposure durations. However, EPA used the duration from the experimental study (1.5 hrs) and the associated air concentration (a LOAEC of 195 ppm or 696 mg/m³) for extrapolation to the dermal route.

There is uncertainty in extrapolating the hazard endpoints across routes. Although some neurotoxicity may result from absorption through nasal passages to the brain, EPA does expect that dermal exposure can also result in neurotoxicity. Furthermore, there is uncertainty regarding the likelihood that dermal exposure will result in lung cancer, but because humans may experience different cancers than rodents, EPA has assumed that the slope factor of the combined tumor types can be considered generally representative of the potential for cancers of other types.

EPA has also identified irritation and burns from dermal contact. Although these are not quantitatively assessed in the risk evaluation, they are an important consideration for risk characterization and are noted in Section 4.3 (Human Health Risk).

3.2.5.3 PODs for Human Health Hazard Endpoints and Confidence Levels

Table 3-21 summarizes the PODs derived for evaluating human health hazards from acute and chronic inhalation scenarios. Table 3-22 summarizes the PODs extrapolated from inhalation studies to evaluate human health hazards from acute and chronic dermal scenarios. EPA has also determined confidence levels for the acute, non-cancer chronic and cancer chronic values used in the risk evaluation. These confidence levels consider the data quality ratings of the study chosen as the basis of dose-response modeling and also consider the strengths and limitations of the body of evidence including the strengths and limitations of the human, animal and MOA information to support the endpoint both qualitatively and quantitatively.

Confidence Levels

For the acute inhalation endpoint, the value used for this risk evaluation is from Putz et al. (1979), a medium quality double-blind study. In addition, there is consistency in observing CNS effects in humans, which is supported by several studies in animals. However, the study used a single concentration and there is uncertainty in converting among exposure durations. Overall, there is medium confidence in this endpoint.

For the chronic non-cancer endpoint, there is limited information in humans regarding liver endpoints but a consistent and full set of studies of liver effects in animals. The dose-response modeling is based on a chronic study given a high data quality rating with a chronic POD that is supported by a second high-quality study. Thus, EPA has medium confidence in the chronic non-cancer endpoint based on liver effects.

For the chronic cancer endpoint, there are some inconsistencies in the epidemiological data and uncertainty in concordance of cancers between animals and humans. However, there is good consistency of results in animals across multiple studies and support from genotoxicity studies that identify effects in the presence of GSTT1. Furthermore, use of PBPK models account for differences in GST and GSTT1 activity between mice and humans and among humans. Furthermore, a high-quality chronic cancer bioassay is used as the basis of the dose-response modeling. Thus, EPA has medium confidence in the chronic cancer endpoint and dose-response model used in this risk evaluation.

Table 3-21. Summary of PODs for Evaluating Human Health Hazards from Acute and Chronic Inhalation Scenarios

Exposure Duration for Risk Analysis	Hazard Value	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference
CHRONIC EXPOSURE	IUR 40 hrs/wk: 1.38×10^{-6} per mg/m ³	Liver and lung tumors	Not applicable	NTP (1986)
	1 st percentile HEC i.e., the HEC ₉₉ 24 hrs/day: 17.2 mg/m ³ (4.8 ppm)	Liver effects	UF _A =3; UF _H =3; UF _L =1 Total UF=10	Nitschke et al. (1988a)
ACUTE EXPOSURE	15-min: 478 ppm (1706 mg/m ³) 1-hr: 240 ppm (840 mg/m ³) 8-hrs: 80 ppm (290 mg/m ³)	Impairment of CNS 7% ↓ visual peripheral performance at 1.5 hrs (p < 0.01)	UF _A =1; UF _H =10; UF _L =3 Total UF=30	CNS data from Putz et al. (1979); Conversion of PODs based on ten Berge et al. (1986)

Table 3-22. Summary of PODs for Evaluating Human Health Hazards from Acute and Chronic Dermal Exposure Scenarios

Exposure Duration for Risk Analysis	Hazard Value Used in Risk Assessment	Effect	Total Uncertainty Factor (UF) for Benchmark MOE
CHRONIC EXPOSURE	Dermal Slope Factor extrapolated from the IUR: 1.1×10^{-5} per mg/kg	Liver and lung tumors	Not applicable
	1 st percentile human equivalent dermal dose (HEDD) i.e., the HEDD ₉₉ extrapolated from inhalation: 2.15 mg/kg	Liver effects	UF _A =3; UF _H =3; UF _L =1 Total UF=10
ACUTE EXPOSURE	Extrapolated from inhalation POD = 16 mg/kg	Impairment of the CNS	UF _A =1; UF _H =10; UF _L =3 Total UF=30

4 RISK CHARACTERIZATION

Environmental and human health risk estimate approaches and results for specific exposure scenarios are presented in sections 4.2 and 4.3, respectively. The aforementioned sections describe the basis for the risk conclusions presented in section 4.1.

4.1 Risk Conclusions

4.1.1 Summary of Environmental Risk

EPA's analysis of environmental risk, in Section 4.2, identified risk to aquatic organisms and sediment-dwelling species (acute $RQ \geq 1$, or a chronic $RQ \geq 1$ and 20 days or more of exceedance for the chronic COC). EPA identified risk to aquatic organisms near four recycling and disposal facilities and one WWTP and identified risk to sediment-dwelling species near one recycling and disposal facility. These facilities are presented in Table 4-1.

EPA's analysis, did not identify risk (acute $RQ < 1$, and chronic $RQ < 1$ or chronic $RQ \geq 1$ with less than 20 days of exceedance) for facilities in other conditions of use including manufacturing, import and repackaging, processing as a reactant, processing and formulation, use in polyurethane foam, use in plastics manufacturing, CTA film manufacturing, lithographic printer cleaning, spot cleaning, "other" unspecified conditions of use, and Department of Defense uses.

In ambient water, EPA's analysis did not identify risk (acute $RQ < 1$, and chronic $RQ < 1$ or chronic $RQ \geq 1$ with less than 20 days of exceedance) to aquatic organisms or sediment-dwelling species from acute or chronic exposures; therefore, the risks identified for the five facilities mentioned above are likely localized to surface water near the facility.

Recycling and Disposal

Four out of 16 recycling and disposal facilities had releases of methylene chloride to surface water that indicate risk to aquatic organisms. One out of these 16 facilities also had a release that indicated risk to sediment-dwelling species. Veolia es Technical Solutions, which transfers methylene chloride to Clean Harbors POTW, had an indirect release to surface water indicating risk from acute exposure with an acute RQ of 6.88. Veolia es Technical Solutions also had risks from chronic exposure for multiple taxonomic groups, with a chronic RQ for amphibians of 201 with 250 days of exceedance, for fish of 119 with 250 days of exceedance, and for aquatic invertebrates of 10.1 with 200 days of exceedance, respectively. Additionally, the data showed that there is risk to sediment dwelling organisms near Clean Harbors POTW due to chronic exposure with $RQ = 10.1$ with 200 days of exceedance. Johnson Matthey West Deptford and Clean Harbors Deer Park both had indirect releases to Clean Harbors Baltimore with chronic RQs for amphibians of 1.32 with 53 days of exceedance and 1.32 with 53 days of exceedance, respectively. Clean Water of New York Inc Staten Island, which may be releasing methylene chloride into an estuarian environment, had chronic RQs for amphibians of 3.92 and for fish of 2.34, both with 20 days of exceedance.

Wastewater Treatment Plants (WWTP)

One out of 29 WWTPs had a release of methylene chloride to surface water that indicated risk to aquatic organisms. Long Beach WPCP Long Beach had a direct release to an estuarian

environment that indicated risk for fish from chronic exposure, with RQs of 2.00 with 365 days of exceedance.

Table 4-1. Final Summary of Facilities Showing Risk from Acute and/or Chronic Exposure from the Release of Methylene Chloride; RQ Greater Than One are Shown in Bold

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Annual Release (kg)	Days of release ^e	Daily Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC Type	COC (ppb)	Days of Exceedance (days/yr) ^h	RQ
OES: Recycling and Disposal											
JOHNSON MATTHEY WEST DEPTFORD, NJ NPDES: NJ0115843	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	620	250	2	118.56	Chronic Amphib.	90	53	1.32
								Chronic Fish	151	27	0.79
								Chronic Invert.	1,800	0	0.07
								Acute Amphib.	2,630	N/A	0.05
CLEAN HARBORS DEER PARK LLC LA PORTE, TX NPDES: TX0005941	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	522	250	2	118.56	Chronic Amphib	90	53	1.32
								Chronic Fish	151	27	0.79
								Chronic Invert.	1,800	0	0.07
								Acute Amphib.	2,630	N/A	0.05
VEOLIA ES TECHNICAL SOLUTIONS LLC MIDDLESEX, NJ NPDES: NJ0127477	Non-POTW WWT	Receiving Facility: Clean Harbors; POTW (Ind.)	Surface water	76,451	250	306	18100	Chronic Amphib.	90	250	201
								Chronic Fish	151	250	119
								Chronic Invert.	1,800	200	10.1
								Acute Amphib.	2,630	N/A	6.88
CLEAN WATER OF NEW YORK INC STATEN ISLAND, NY NPDES: NY0200484	Surface Water	Active Releaser (Surrogate): NPDES NJ0000019	Still body	2.38	250	0.01	27.94	Chronic Amphib	90	250	0.31
								Chronic Fish	151	0	0.19
								Chronic Invert.	1,800	0	0.02
					20	0.12	352.94	Acute Amphib	2,630	N/A	0.01
								Chronic Amphib	90	20	3.92
								Chronic Fish	151	20	2.34
Chronic Invert.	1800	0	0.20								

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Annual Release (kg)	Days of release ^e	Daily Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC Type	COC (ppb)	Days of Exceedance (days/yr) ^h	RQ
								Acute Amphib	2,630	N/A	0.13
OES: WWTP											
LONG BEACH (C) WPCP LONG BEACH, NY NPDES: NY0020567	Surface Water	Active Releaser: NPDES NY0020567	Still water	2,730	365	7	301.46	Chronic Amphib.	90	365	3.35
								Chronic Fish	151	365	2.00
								Chronic Invert.	1,800	0	0.17
								Acute Amphib	2,630	N/A	0.11
					20	136.49	5878.12	Chronic Amphib	-	-	-
								Chronic Fish	-	-	-
								Chronic Invert.	-	-	-
								Acute Amphib.	-	-	-
<p>a. Facilities actively releasing methylene chloride were identified via DMR and TRI databases for the 2016 reporting year.</p> <p>b. Release media are either direct (release from active facility directly to surface water) or indirect (transfer of wastewater from active facility to a receiving POTW or non-POTW WWTP facility). A wastewater treatment removal rate of 57% is applied to all indirect releases, as well as direct releases from WWTPs.</p> <p>c. If a valid NPDES of the direct or indirect releaser was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.</p> <p>d. EFAST uses either the “surface water” model, for rivers and streams, or the “still water” model, for lakes, bays, and oceans.</p> <p>e. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.</p> <p>f. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.</p> <p>g. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.</p> <p>h. To determine the PDM days of exceedance for still bodies of water, the estimated number of release days should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.</p>											

4.1.2 Summary of Risk Estimates for Inhalation and Dermal Exposures to Workers

Table 4-2 summarizes the risk estimates for inhalation and dermal exposures for all occupational exposure scenarios. Risk estimates that exceed the benchmark (i.e., MOEs less than the benchmark MOE or cancer risks greater than the cancer risk benchmark) are highlighted by bolding the number and shading the cell. U.S. EPA shaded the cells for risk estimates that are not calculated i.e., short-term exposures estimates for chronic endpoints and that are not assessed i.e., PPE use for ONUs. The risk characterization is described in more detail in Sections 2.4.1 and 4.3.2 and specific links to the exposure and risk characterization sections are listed in Table 4-2 in the column headed Occupational Exposure Scenario.

Table 4-2 Summary of Risk Estimates for Inhalation and Dermal Exposures to Workers by Condition of Use

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
Manufacturing/ Domestic manufacturing	Manufacturing	Section 2.4.1.2.1 and 4.3.2.1.2 - Manufacturing Exposure	Worker	Inhalation 8-hr TWA	Central Tendency	795	207	2.00E-07	19878 (APF 25)	5164 (APF 25)	8.00E-07 (APF 25)
					High-End	63	16	3.26E-06	1575 (APF 25)	409 (APF 25)	1.30-07 (APF 25)
			Worker	Inhalation 15-min TWA *	Central Tendency	179	N/C	N/C	4465 (APF 25)	N/C	N/C
					High-End	9.3	N/C	N/C	232 (APF 25)	N/C	N/C
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	795	207	2.00E-07	N/A	N/A	N/A
ONU	Inhalation 15-min TWA *	Central Tendency	179	N/C	N/C	N/A	N/A	N/A			
Manufacturing/ Import	Import	Section 2.4.1.2.4 and 4.3.2.1.5 - Repackaging	Worker	Inhalation 8-hr TWA	Central Tendency	33	8.54	4.84E-06	822 (APF 25)	213 (APF 25)	-
					High-End	2.1	0.55	9.74E-05	53 (APF 25)	14 (APF 25)	-
			Worker	Inhalation 1-hr TWA*	Central Tendency	4.7	N/C	N/C	118 (APF 25)	N/C	N/C
					High-End	2.6	N/C	N/C	64 (APF 25)	N/C	N/C
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	33	8.54	4.84E-06	N/A	N/A	N/A
ONU	Inhalation 1-hr TWA*	Central Tendency	4.7	N/C	N/C	N/A	N/A	N/A			
Processing/ Processing as a reactant	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants)	Section 2.4.1.2.2 and 4.3.2.1.3 - Processing as a Reactant	Worker	Inhalation 8-hr TWA	Central Tendency	178	46	8.95E-07	4441 (APF 25)	1154 (APF 25)	-

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE				
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)		
					High-End	2.7	0.7	7.63E-05	67 (APF 25)	17 (APF 25)	-		
			Worker	Inhalation 15-min TWA *	Point Estimate	4.9	N/C	N/C	122 (APF 25)	N/C	N/C		
	Intermediate for pesticide, fertilizer, and other agricultural chemical manufacturing		Worker	Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)		
			ONU	Inhalation 8-hr TWA	Central Tendency	178	46	8.95E-07	N/A	N/A	N/A		
					High-End	2.7	0.7	7.63E-05	N/A	N/A	N/A		
	Petrochemical manufacturing		ONU	Inhalation 15-min TWA *	Point Estimate	4.9	N/C	N/C	N/A	N/A	N/A		
	Intermediate for other chemicals												
Processing/ Incorporated into formulation, mixture, or reaction product	Solvents (for cleaning or degreasing), including manufacturing of: · All other basic organic chemical · Soap, cleaning compound and toilet preparation	Section 2.4.1.2.3 and 4.3.2.1.4 - Processing - Incorporation into Formulation, Mixture, or Reaction Product	Worker	Inhalation 8-hr TWA	Central Tendency	2.9	0.74	5.58E-05	143 (APF 50)	37 (APF 50)	2.23E-06 (APF 25)		
					High-End	0.54	0.14	3.81E-04	27 (APF 50)	7.0 (APF 50)	1.52E-05 (APF 25)		
			Worker	Inhalation 15-min TWA *	Point Estimate	9.5	N/C	N/C	237 (APF 25)	N/C	N/C		
				Worker	Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)	
			Solvents (which become part of product formulation or mixture), including manufacturing of: · All other chemical product and preparation · Paints and coatings		ONU	Inhalation 8-hr TWA	Central Tendency	2.9	0.74	5.58E-05	N/A	N/A	N/A
							High-End	0.54	0.14	3.81E-04	N/A	N/A	N/A
					ONU	Inhalation 15-min TWA *	Point Estimate	9.5	N/C	N/C	N/A	N/A	N/A
			Propellants and blowing agents for all other chemical product and preparation manufacturing										

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE			
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	
	Propellants and blowing agents for plastics product manufacturing											
	Paint additives and coating additives not described by other codes											
	Laboratory chemicals for all other chemical product and preparation manufacturing											
	Laboratory chemicals for other industrial sectors											
	Processing aid, not otherwise listed for petrochemical manufacturing											
	Adhesive and sealant chemicals in adhesive manufacturing											
	Oil and gas drilling, extraction, and support activities											
											See the rows above for risk estimates	
Processing/ Repackaging	Solvents (which become part of product formulation or mixture) for all other chemical product and preparation manufacturing	Section 2.4.1.2.4 and 4.3.2.1.5 - Repackaging	Worker	Inhalation 8-hr TWA	Central Tendency	33	8.54	4.84E-06	822 (APF 25)	213 (APF 25)	-	
					High-End	2.1	0.55	9.74E-05	53 (APF 25)	14 (APF 25)	-	
			Worker	Inhalation 1-hr TWA*	Central Tendency	4.7	N/C	N/C	118 (APF 25)	N/C	N/C	
					High-End	2.6	N/C	N/C	64 (APF 25)	N/C	N/C	
	All other chemical product and preparation manufacturing		Worker	Dermal	High-End		7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
						Central Tendency	33	8.54	4.84E-06	N/A	N/A	N/A
	All other chemical product and preparation manufacturing		ONU	Inhalation 8-hr TWA	High-End		2.1	0.55	9.74E-05	N/A	N/A	N/A
						Central Tendency	4.7	N/C	N/C	N/A	N/A	N/A
All other chemical product and preparation manufacturing	ONU	Inhalation 1-hr TWA*	Central Tendency		4.7	N/C	N/C	N/A	N/A	N/A		
Processing/ Recycling	Recycling	Section 2.4.1.2.5 and 4.3.2.1.6 - Waste	Worker	Inhalation 8-hr TWA	Central Tendency	124	32	1.29E-06	3092 (APF 25)	803 (APF 25)	-	

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
		Handling, Disposal, Treatment, and Recycling			High-End	15	4.0	1.38E-05	382 (APF 25)	99 (APF 25)	-
			Worker	Dermal	High-End	3.6	0.93	5.71E-05	90 (APF 25)	23 (APF 25)	-
			ONU	Inhalation 8-hr TWA	Central Tendency	124	32	1.29E-06	N/A	N/A	N/A
					High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
Distribution in commerce	Distribution	Distribution	Please see Section 5.2.1.7								
Industrial and commercial use/ Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)	Section 2.4.1.2.5 and 4.3.2.1.7 - Batch Open-Top Vapor Degreasing	Worker	Inhalation 8-hr TWA	Central Tendency	1.7	0.45	9.23E-05	43 (APF 25)	11 (APF 25)	3.69E-06 (APF 25)
					High-End	0.39	0.10	5.27E-04	19 (APF 50)	5.1 (APF 50)	2.11E-05 (APF 25)
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
					ONU	Inhalation 8-hr TWA	Central Tendency	3	0.87	4.74E-05	N/A
			ONU	High-End	0.64		0.2	3.22E-04	N/A	N/A	N/A
					In-line vapor degreaser (e.g., conveyorized, web cleaner)	Section 2.4.1.2.6 and 4.3.2.1.8 - Conveyorized Vapor Degreasing	Worker	Inhalation 8-hr TWA	Central Tendency	0.60	0.15
	High-End	0.21	0.05	9.87E-04					10.4 (APF 50)	2.7 (APF 50)	2.97E-05 (APF 25)
	Worker	Dermal	High-End	7.1			1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
			ONU	Inhalation 8-hr TWA			Central Tendency	1	0.30	1.39E-04	N/A
	High-End	0.32			0.1	6.37E-04	N/A	N/A	N/A		
Cold cleaner		Section 2.4.1.2.7 and 4.3.2.1.9 - Cold Cleaning	Worker	Inhalation 8-hr TWA	Central Tendency	1.04	0.27	1.54E-04	52 (APF 50)	13 (APF 50)	6.14E-6 (APF 25)
	High-End				0.29	0.08	7.08E-04	15 (APF 50)	3.8 (APF 50)	2.83E-05 (APF 25)	

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	1.04	0.27	1.54E-04	N/A	N/A	N/A
					High-End	0.29	0.08	7.08E-04	N/A	N/A	N/A
	Aerosol spray degreaser/cleaner	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
					High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06
			Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
					High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A
			Industrial and commercial use/ Adhesives and sealants	Single component glues and adhesives and sealants and caulks	Section 2.4.1.2.9 and 4.3.2.1.11 - Adhesives and Sealants (spray)	Worker	Inhalation 8-hr TWA	Central Tendency	7.4	1.93	2.14E-05
High-End	0.52	0.14						3.95E-04	26 (APF 50)	6.8 (APF 50)	1.58E-05 (APF 25)
Worker	Dermal	High-End				7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
ONU	Inhalation 8-hr TWA	Central Tendency				7.4	1.93	2.14E-05	N/A	N/A	N/A
		High-End				0.52	0.14	3.95E-04	N/A	N/A	N/A
Section 2.4.1.2.9 and 4.3.2.1.11 - Adhesives and Sealants (non-spray)	Worker	Inhalation 8-hr TWA				Central Tendency	28	7.2	5.74E-06	692 (APF 25)	180 (APF 25)
				High-End	0.98	0.25	2.10E-04	49 (APF 50)	13 (APF 50)	8.37E-06 (APF 25)	
	Worker	Dermal		High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)	
ONU	Inhalation 8-hr TWA	Central Tendency		28	7.2	5.80E-06	N/A	N/A	N/A		

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE			
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	
					High-End	0.52	0.25	3.95E-04	N/A	N/A	N/A	
Industrial and commercial use/ Paints and coatings including commercial paint and coating removers	Paints and coatings use and paints and coating removers, including furniture refinisher	Section 2.4.1.2.10 and 4.3.2.1.12 - Paints and Coatings	Worker	Inhalation 8-hr TWA	Central Tendency	4.15	1.08	3.83E-05	104 (APF 25)	27 (APF 25)	1.53E-06 (APF 25)	
					High-End	0.80	0.21	2.58E-04	40 (APF 50)	10.3 (APF 50)	1.03E-05 (APF 25)	
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)	
			ONU	Inhalation 8-hr TWA	Central Tendency	4.15	1.08	3.83E-05	N/A	N/A	N/A	
	High-End	0.80			0.21	2.58E-04	N/A	N/A	N/A			
		Paint and Coating Removers	Please see Appendix L.									
	Adhesive/caulk removers	Section 2.4.1.2.11 and 4.3.2.1.13 - Adhesive and Caulk Removers	Worker	Inhalation 8-hr TWA	Central Tendency	0.19	0.05	8.34E-04	9.5 (APF 50)	2.5 (APF 50)	3.33E-05 (APF 25)	
					High-End	0.10	0.03	2.11E-03	4.9 (APF 50)	1.3 (APF 50)	8.44E-05 (APF 25)	
			Worker	Dermal	High-End	4.9	0.97	1.26E-05	49 (PF 10)	9.7 (PF 10)	2.51E-06 (PF 5)	
			ONU	Inhalation 8-hr TWA	Central Tendency	0.19	0.05	8.34E-04	N/A	N/A	N/A	
High-End					0.10	0.03	2.11E-03	N/A	N/A	N/A		
Industrial and commercial use/ Metal products not covered elsewhere			Degreasers – aerosol and non-aerosol degreasers and cleaners (e.g., coil cleaners)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
	High-End	1.3				0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06		
	Worker	Dermal		High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)		
	ONU	Inhalation 8-hr TWA		Central Tendency	48	12	3.31E-06	N/A	N/A	N/A		
				High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A		

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
		Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)
					High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)
			Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
					Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A
			ONU	Inhalation 8-hr TWA	High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A
					Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A
Industrial and commercial use/ Fabric, textile and leather products not covered elsewhere	Textile finishing and impregnating/surface treatment products (e.g., water repellent)	Section 2.4.1.2.12 and 4.3.2.1.15 - Fabric Finishing	Worker	Inhalation 8-hr TWA	Central Tendency	37	9.6	4.29E-06	928 (APF 25)	241 (APF 25)	1.71E-07 (APF 25)
					High-End	2.1	0.56	9.60E-05	53 (APF 25)	14 (APF 25)	3.84E-06 (APF 25)
			Worker	Dermal	High-End	4.7	0.93	1.30E-05	47 (PF 10)	9.3 (PF 10)	2.61E-06 (PF 5)
					Central Tendency	37	9.6	4.29E-06	N/A	N/A	N/A
			ONU	Inhalation 8-hr TWA	High-End	2.1	0.56	9.60E-05	N/A	N/A	N/A
					Central Tendency	37	9.6	4.29E-06	N/A	N/A	N/A
Industrial and commercial use/ Automotive care products	Function fluids for air conditioners: refrigerant, treatment, leak sealer	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)
					High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)
			Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
					Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A
			ONU	Inhalation 8-hr TWA	High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A
					Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A
	Interior car care – spot remover	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
					High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
	Degreasing, Aerosol Lubricants, Automotive Care Products)	Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
					High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A
	Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
					High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06
			Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
					High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A
			Industrial and commercial use/ Apparel and footwear care products	Post-market waxes and polishes applied to footwear (e.g., shoe polish)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)
	High-End	1.3					0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06
Dermal	High-End	4.6					0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
ONU	Inhalation 8-hr TWA	Central Tendency			48	12	3.31E-06	N/A	N/A	N/A	
		High-End			1.3	0.33	1.61E-04	N/A	N/A	N/A	
Industrial and commercial use/ Laundry and dishwashing products	Spot remover for apparel and textiles	Worker	Inhalation 8-hr TWA	Central Tendency	436	113	3.66E-07	10896 (APF 25)	2830 (APF 25)	1.46E-08 (APF 25)	
				High-End	1.6	0.41	1.31E-04	39 (APF 25)	10 (APF 25)	5.25E-06 (APF 25)	
				Dermal	High-End	4.9	0.97	1.26E-05	49 (PF 10)	9.7 (PF 10)	2.51E-06 (PF 5)
		ONU	Inhalation 8-hr TWA	Central Tendency	436	113	3.66E-07	N/A	N/A	N/A	

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
					High-End	1.6	0.41	1.31E-04	N/A	N/A	N/A
Industrial and commercial use/ Lubricants and greases	Liquid and spray lubricants and greases	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
					High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06
			Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
					Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
			ONU	Inhalation 8-hr TWA	High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A
					Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)	
				High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)	
		Worker	Dermal	High-End	7.1	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
				Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A	
		ONU	Inhalation 8-hr TWA	High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A	
				Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A	
Degreasers – aerosol and non-aerosol degreasers and cleaners	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07	
				High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06	
		Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
				Central Tendency	48	12	3.31E-06	N/A	N/A	N/A	
		ONU	Inhalation 8-hr TWA	High-End	1.3	0.33	1.61E-04	N/A	N/A	N/A	
				Central Tendency	48	12	3.31E-06	N/A	N/A	N/A	
Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)			

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
		Miscellaneous Non-Aerosol Industrial and Commercial Uses			High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)
			Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A
		High-End			0.31	0.08	6.58E-04	N/A	N/A	N/A	
Industrial and commercial use/ Building/ construction materials not covered elsewhere	Cold pipe insulation	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
					High-End	1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06
			Worker	Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A	N/A	N/A
High-End	1.3	0.33			1.61E-04	N/A	N/A	N/A			
Industrial and commercial use/ Solvents (which become part of product formulation or mixture)	All other chemical product and preparation manufacturing	Section 2.4.1.2.3 and 4.3.2.1.4 - Processing - Incorporation into Formulation, Mixture, or Reaction Product	Worker	Inhalation 8-hr TWA	Central Tendency	2.9	0.74	5.58E-05	143 (APF 50)	37 (APF 50)	2.23E-06 (APF 25)
					High-End	0.54	0.14	3.81E-04	27 (APF 50)	7.0 (APF 50)	1.52E-05 (APF 25)
			Worker	Inhalation 15-min TWA *	Point Estimate	9.5	N/C	N/C	237 (APF 25)	N/C	N/C
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
			ONU	Inhalation 15-min TWA *	Point Estimate	2.9	0.74	5.58E-05	N/A	N/A	N/A
					Central Tendency	0.54	0.14	3.81E-04	N/A	N/A	N/A
		ONU	Inhalation 8-hr TWA	High-End	9.5	N/C	N/C	N/A	N/A	N/A	

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
Industrial and commercial use/ Processing aid not otherwise listed	In multiple manufacturing sectors	Section 2.4.1.2.14 and 4.3.2.1.17 - Cellulose Triacetate Film Production	Worker	Inhalation 8-hr TWA	Central Tendency	0.28	0.07	5.68E-04	14 (APF 50)	3.6 (APF 50)	2.27E-05 (APF 25)
					High-End	0.21	0.05	7.67E-04	10 (APF 50)	2.7 (APF 50)	3.07E-05 (APF 25)
			Worker	Dermal	High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
					ONU	Inhalation 8-hr TWA	Central Tendency	0.28	0.07	5.68E-04	N/A
			High-End	0.21			0.05	7.67E-04	N/A	N/A	N/A
			Industrial and commercial use/ Propellants and blowing agents	Flexible polyurethane foam manufacturing	Section 2.4.1.2.15 and 4.3.2.1.19 - Flexible Polyurethane Foam Manufacturing	Worker	Inhalation 8-hr TWA	Central Tendency	1.5	0.39	1.16E-04
High-End	0.29	0.08						7.08E-04	15 (APF 50)	3.8 (APF 50)	2.83E-05 (APF 25)
Worker	Dermal	High-End				7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
		ONU				Inhalation 8-hr TWA	Central Tendency	1.5	0.39	1.16E-04	N/A
High-End	0.29						0.08	7.08E-04	N/A	N/A	N/A
Industrial and commercial use/ Other Uses	Laboratory chemicals - all other chemical product and preparation manufacturing	Section 2.4.1.2.16 and 4.3.2.1.20 - Laboratory Use				Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06
			High-End	2.8	0.74			7.21E-05	77 (APF 25)	18 (APF 25)	2.89E-06 (APF 25)
			Worker	Inhalation 15-min TWA *	Central Tendency	256	N/C	N/C	6394 (APF 25)	N/C	N/C
					High-End	22	N/C	N/C	549 (APF 25)	N/C	N/C
			Worker	Dermal	High-End	4.6	0.9	1.35E-05	91 (PF 20)	18 (PF 20)	2.70E-06 (PF 5)
					ONU	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A
			High-End	2.8			0.74	7.21E-05	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
			ONU	Inhalation 15-min TWA *	Central Tendency	256	N/C	N/C	N/A	N/C	N/C
					High-End	22	N/C	N/C	N/A	N/C	N/C
	Electrical equipment, appliance, and component manufacturing	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.33	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)
					High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)
			Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)
			ONU	Inhalation 8-hr TWA	Central Tendency	5.1	1.33	3.11E-05	N/A	N/A	N/A
					High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A
			Plastic and rubber products	Section 2.4.1.2.17 and 4.3.2.1.18 - Plastic Product Manufacturing	Worker	Inhalation 8-hr TWA	Central Tendency	34	8.9	4.66E-06	853 (APF 25)
	High-End	1.4					0.37	1.46E-04	30 (APF 25)	18 (APF 50)	5.83E-06 (APF 25)
	Worker	Inhalation 15-min TWA *			Central Tendency	21	N/C	N/C	517 (APF 25)	N/C	N/C
					High-End	13	N/C	N/C	328 (APF 25)	N/C	N/C
	Worker	Dermal			High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
	ONU	Inhalation 8-hr TWA			Central Tendency	30	7.3	5.31E-06	N/A	N/A	N/A
					High-End	28	7.8	7.28E-06	N/A	N/A	N/A
Section 2.4.1.2.14 and 4.3.2.1.17 - Cellulose Triacetate Film Production	Worker	Inhalation 8-hr TWA			Central Tendency	0.28	0.07	5.68E-04	14 (APF 50)	3.6 (APF 50)	2.27E-05 (APF 25)
					High-End	0.21	0.05	7.67E-04	10 (APF 50)	2.7 (APF 50)	3.07E-05 (APF 25)
		Dermal			High-End	7.1	1.4	8.69E-06	36 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)
			ONU	Inhalation 8-hr TWA	Central Tendency	0.28	0.07	5.68E-04	N/A	N/A	N/A
					High-End	0.21	0.05	7.67E-04	N/A	N/A	N/A
	Anti-adhesive agent - anti-spatter welding aerosol	Section 2.4.1.2.8 and 4.3.2.1.10 - Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Worker	Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	1201 (APF 25)	312 (APF 25)	1.32E-07
High-End					1.3	0.33	1.61E-04	32 (APF 25)	17 (APF 50)	6.44E-06	
Worker			Dermal	High-End	4.6	0.9	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
ONU			Inhalation 8-hr TWA	Central Tendency	48	12	3.31E-06	N/A	N/A	N/A	
	High-End	1.3		0.33	1.61E-04	N/A	N/A	N/A			
Oil and gas drilling, extraction, and support activities	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)	
				High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)	
		Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
		ONU	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A	
				High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A	
		Toys, playground, and sporting equipment - including novelty articles (toys, gifts, etc.)	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.3	3.11E-05	128 (APF 25)	33 (APF 25)
High-End	0.31					0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)	
Worker	Dermal			High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
ONU	Inhalation 8-hr TWA			Central Tendency	5.1	1.3	3.11E-05	N/A	N/A	N/A	
		High-End	0.31	0.08	6.58E-04	N/A	N/A	N/A			

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE			
						Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	Acute Non-cancer (benchmark MOE = 30)	Chronic Non-cancer (benchmark MOE = 10)	Cancer (benchmark = 10 ⁻⁴)	
	Lithographic printing cleaner	Section 2.4.1.2.18 and 4.3.2.1.22 - Lithographic Printing Plate Cleaning	Worker	Inhalation 8-hr TWA	Central Tendency	33	8.7	4.78E-06	832 (APF 25)	216 (APF 25)	1.91E-07 (APF 25)	
					High-End	1.8	0.47	1.13E-04	45 (APF 50)	12 (APF 25)	4.54E-06 (APF 25)	
			Worker	Dermal	High-End	5.1	1.0	1.21E-05	51 (PF 10)	10 (PF 10)	2.41E-06 (PF 5)	
					ONU	Inhalation 8-hr TWA	Central Tendency	33	8.7	4.78E-06	N/A	N/A
	High-End	1.8	0.47	1.13E-04	N/A		N/A	N/A				
	Carbon remover, Wood floor cleaner, and Brush cleaner	Section 2.4.1.2.19 and 4.3.2.1.14 - Miscellaneous Non-Aerosol Industrial and Commercial Uses	Worker	Inhalation 8-hr TWA	Central Tendency	5.1	1.33	3.11E-05	128 (APF 25)	33 (APF 25)	1.24E-06 (APF 25)	
					High-End	0.31	0.08	6.58E-04	16 (APF 50)	4.0 (APF 50)	2.63E-05 (APF 25)	
			Worker	Dermal	High-End	4.6	0.90	1.35E-05	46 (PF 10)	9.0 (PF 10)	2.70E-06 (PF 5)	
ONU					Inhalation 8-hr TWA	Central Tendency	5.1	1.33	3.11E-05	N/A	N/A	N/A
	High-End	0.31	0.08	6.58E-04		N/A	N/A	N/A				
Disposal/ Disposal	Industrial pre-treatment	Section 2.4.1.2.20 and 4.3.2.1.6 - Waste Handling, Disposal, Treatment, and Recycling	Worker	Inhalation 8-hr TWA	Central Tendency	124	32	1.29E-06	3092 (APF 25)	803 (APF 25)	-	
	High-End				3.6	0.93	5.71E-05	90 (APF 25)	23 (APF 25)	-		
	Worker				Dermal	High-End	7.1	1.4	8.69E-06	356 (PF 5)	28 (PF 20)	1.74E-06 (PF 5)
						ONU	Inhalation 8-hr TWA	Central Tendency	124	32	1.29E-06	N/A
	High-End		3.6	0.93	5.71E-05			N/A	N/A	N/A		
	Industrial wastewater treatment											
	Publicly owned treatment works (POTW)											
	Underground injection											
Municipal landfill												
Hazardous landfill												
Other land disposal												
Municipal waste incinerator												
Off-site waste transfer												

N/C = not calculated because 15-min TWAs are not used for assessing chronic non-cancer or cancer risks

* risk estimates for the 15-min TWA are shown for COUs that had available exposure data and when risks from acute exposure indicated were different from 8-hr TWA, see Section 4.2.2.1 for details of 15-min TWAs for each OES. N/A = not assessed because ONUs are not assumed to be wearing PPE

- = cancer risks assuming PPE are not shown when the cancer risk without PPE was above the cancer risk benchmark of 10⁻⁴

4.1.3 Summary of Risk Estimates for Inhalation and Dermal Exposures to Consumers and Bystanders

Table 4-3 summarizes the risk estimates for CNS effects from acute inhalation and dermal exposures for all consumer exposure scenarios. Risk estimates that exceed the benchmark (i.e., MOEs less than the benchmark MOE) are highlighted by bolding the number and shading the cell. The risk characterization is described in more detail in Sections 2.4.2 and 4.3.2.3 and specific links to the exposure and risk characterization sections are listed in Table 4-3 in the column headed Consumer Condition of Use Scenario.

Table 4-3 Summary of Risk Estimates for CNS effects from Acute Inhalation and Dermal Exposures to Consumers by Conditions of Use

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
Solvents (for cleaning and degreasing)	Aerosol spray degreaser/cleaner	Section 2.4.2.4.5 and Section 4.3.2.3.1 - Brake Cleaner	Inhalation 1-hr	Low Intensity User	24	202
				Medium Intensity User	1.7	14
				High Intensity User	0.43	2.3
			Inhalation 8-hr	Low Intensity User	50	218
				Medium Intensity User	3.6	15
				High Intensity User	0.56	2.0
			Dermal	Low Intensity User	234	N/A
				Medium Intensity User	4.4	N/A
				High Intensity User	0.32	N/A
		Section 2.4.2.4.7 and Section 4.3.2.3.2 - Carbon Remover	Inhalation 1-hr	Low Intensity User	9.5	103
				Medium Intensity User	0.94	9.7
				High Intensity User	0.18	1.0
			Inhalation 8-hr	Low Intensity User	22	119
				Medium Intensity User	2.1	11
				High Intensity User	0.23	0.93
			Dermal	Low Intensity User	38	N/A
				Medium Intensity User	2.9	N/A
				High Intensity User	0.36	N/A
		Section 2.4.2.4.8 and Section 4.3.2.3.3 - Carburetor Cleaner	Inhalation 1-hr	Low Intensity User	13	110
				Medium Intensity User	1.4	12
				High Intensity User	0.30	2.0
Inhalation 8-hr	Low Intensity User		27	118		
	Medium Intensity User		3.0	13		
	High Intensity User		0.55	2.0		

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
		Section 2.4.2.4.9 and Section 4.3.2.3.4 - Coil Cleaner	Dermal	Low Intensity User	158	N/A
				Medium Intensity User	10	N/A
				High Intensity User	1.0	N/A
			Inhalation 1-hr	Low Intensity User	5.5	60
				Medium Intensity User	0.57	5.9
				High Intensity User	0.11	0.61
			Inhalation 8-hr	Low Intensity User	13	69
				Medium Intensity User	1.3	6.8
				High Intensity User	0.14	0.57
		Dermal	Low Intensity User	22	N/A	
			Medium Intensity User	1.8	N/A	
			High Intensity User	0.22	N/A	
		Section 2.4.2.4.11 and Section 4.3.2.3.5 - Electronics Cleaner	Inhalation 1-hr	Low Intensity User	1171	8027
				Medium Intensity User	91	633
				High Intensity User	6.5	31
			Inhalation 8-hr	Low Intensity User	2492	10794
				Medium Intensity User	195	854
				High Intensity User	13	46
			Dermal	Low Intensity User	1208	N/A
				Medium Intensity User	328	N/A
				High Intensity User	64	N/A
Section 2.4.2.4.12 and Section 4.3.2.3.6 - Engine Cleaner	Inhalation 1-hr	Low Intensity User	5.4	47		
		Medium Intensity User	0.62	5.1		
		High Intensity User	0.16	0.88		
	Inhalation 8-hr	Low Intensity User	12	50		
		Medium Intensity User	1.3	5.4		

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)		
			Dermal	High Intensity User	0.22	0.77		
				Low Intensity User	32	N/A		
				Medium Intensity User	4.7	N/A		
				High Intensity User	0.38	N/A		
		Section 2.4.2.4.13 and Section 4.3.2.3.7 - Gasket Remover	Inhalation 1-hr	Low Intensity User	5.9	51		
				Medium Intensity User	1.1	9.1		
				High Intensity User	0.22	1.4		
			Inhalation 8-hr	Low Intensity User	13	55		
				Medium Intensity User	2.3	9.7		
				High Intensity User	0.42	1.4		
			Dermal	Low Intensity User	29	N/A		
				Medium Intensity User	2.9	N/A		
				High Intensity User	0.72	N/A		
		Adhesives and Sealants	Single component glues and adhesives and sealants and caulk	Section 2.4.2.4.1 and Section 4.3.2.3.8 – Adhesives	Inhalation 1-hr	Low Intensity User	199	2188
						Medium Intensity User	12	130
High Intensity User	0.53					4.2		
Inhalation 8-hr	Low Intensity User				452	2535		
	Medium Intensity User				27	150		
	High Intensity User				1.1	4.7		
Dermal	Low Intensity User				372	N/A		
	Medium Intensity User				27	N/A		
	High Intensity User				6.3	N/A		
Section 2.4.2.4.14 and Section 4.3.2.3.14 - Sealant	Inhalation 1-hr			Low Intensity User	35	304		
				Medium Intensity User	2.9	24		
				High Intensity User	0.59	3.8		
Inhalation 8-hr	Low Intensity User			75	327			

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
				Medium Intensity User	6.1	26
				High Intensity User	1.1	3.6
			Dermal	Low Intensity User	198	N/A
				Medium Intensity User	16	N/A
				High Intensity User	12	N/A
Paints and coatings including paint and coating removers	Paint and Coating Removers	Section 2.4.2.4.6 and Section 4.3.2.3.10 - Brush Cleaner	Inhalation 1-hr	Low Intensity User	3956	44077
				Medium Intensity User	786	6209
				High Intensity User	462	1293
			Inhalation 8-hr	Low Intensity User	8981	50216
				Medium Intensity User	1653	6916
				High Intensity User	191	919
			Dermal	Low Intensity User	396	N/A
				Medium Intensity User	33	N/A
				High Intensity User	4.7	N/A
	Adhesive/caulk removers	Section 2.4.2.4.2 and Section 4.3.2.3.11 - Adhesives Remover	Inhalation 1-hr	Low Intensity User	255	2869
				Medium Intensity User	17	134
				High Intensity User	11	14
			Inhalation 8-hr	Low Intensity User	581	3269
				Medium Intensity User	36	150
				High Intensity User	4.3	16
			Dermal	Low Intensity User	21	N/A
				Medium Intensity User	0.71	N/A
				High Intensity User	0.090	N/A
Metal products not covered elsewhere	Degreasers - aerosol and non-aerosol degreasers	Section 2.4.2.4.7 and Section 4.3.2.3.2 - Carbon Remover	Inhalation 1-hr	Low Intensity User	9.5	103
				Medium Intensity User	0.94	9.7
				High Intensity User	0.18	1.0

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
			Inhalation 8-hr	Low Intensity User	22	119
				Medium Intensity User	2.1	11
				High Intensity User	0.23	0.93
			Dermal	Low Intensity User	38	N/A
				Medium Intensity User	2.9	N/A
				High Intensity User	0.36	N/A
		Section 2.4.2.4.9 and Section 4.3.2.3.4 - Coil Cleaner	Inhalation 1-hr	Low Intensity User	5.5	60
				Medium Intensity User	0.57	5.9
				High Intensity User	0.11	0.61
			Inhalation 8-hr	Low Intensity User	13	69
				Medium Intensity User	1.3	6.8
				High Intensity User	0.14	0.57
			Dermal	Low Intensity User	22	N/A
				Medium Intensity User	1.8	N/A
				High Intensity User	0.22	N/A
		Section 2.4.2.4.11 and Section 4.3.2.3.5 - Electronics Cleaner	Inhalation 1-hr	Low Intensity User	1171	8027
				Medium Intensity User	91	633
				High Intensity User	6.5	31
			Inhalation 8-hr	Low Intensity User	2492	10794
				Medium Intensity User	195	854
				High Intensity User	13	46
Dermal	Low Intensity User		1208	N/A		
	Medium Intensity User		328	N/A		
	High Intensity User		64	N/A		

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
Automotive care products	Function fluids for air conditioners: refrigerant, treatment, leak sealer	Section 2.4.2.4.3 and Section 4.3.2.3.9 - Automotive AC Leak Sealer	Inhalation 1-hr	Low Intensity User	120	1031
				Medium Intensity User	123	1015
				High Intensity User	210	1117
			Inhalation 8-hr	Low Intensity User	255	1107
				Medium Intensity User	259	1077
				High Intensity User	274	980
		Dermal	Low Intensity User	10	N/A	
			Medium Intensity User	5.0	N/A	
			High Intensity User	3.9	N/A	
		Section 2.4.2.4.4 and Section 4.3.2.3.12 - Automotive AC Refrigerant	Inhalation 1-hr	Low Intensity User	102	875
				Medium Intensity User	8.8	72
				High Intensity User	3.6	19
	Inhalation 8-hr		Low Intensity User	216	939	
			Medium Intensity User	18	76	
			High Intensity User	4.7	17	
	Dermal		Low Intensity User	1482	N/A	
			Medium Intensity User	164	N/A	
			High Intensity User	21	N/A	
	Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Inhalation 1-hr	Low Intensity User	24	202	
			Medium Intensity User	1.7	14	
			High Intensity User	0.43	2.3	
Inhalation 8-hr		Low Intensity User	50	218		
		Medium Intensity User	3.6	15		
		High Intensity User	0.56	2.0		
Dermal		Low Intensity User	234	N/A		
		Medium Intensity User	4.4	N/A		

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
				High Intensity User	0.32	N/A
		Section 2.4.2.4.8 and Section 4.3.2.3.3 - Carburetor Cleaner	Inhalation 1-hr	Low Intensity User	13	110
				Medium Intensity User	1.4	12
				High Intensity User	0.28	2.0
			Inhalation 8-hr	Low Intensity User	27	118
				Medium Intensity User	3.0	13
				High Intensity User	0.55	2.0
			Dermal	Low Intensity User	158	N/A
				Medium Intensity User	10	N/A
				High Intensity User	1.0	N/A
		Section 2.4.2.4.12 and Section 4.3.2.3.6 - Engine Cleaner	Inhalation 1-hr	Low Intensity User	5.4	47
				Medium Intensity User	0.60	5.1
				High Intensity User	0.20	0.88
			Inhalation 8-hr	Low Intensity User	12	50
				Medium Intensity User	1.3	5.4
				High Intensity User	0.20	0.77
			Dermal	Low Intensity User	32	N/A
				Medium Intensity User	4.7	N/A
				High Intensity User	0.38	N/A
		Section 2.4.2.4.13 and Section 4.3.2.3.7 - Gasket Remover	Inhalation 1-hr	Low Intensity User	5.9	51
				Medium Intensity User	1.1	9.1
				High Intensity User	0.22	1.4
			Inhalation 8-hr	Low Intensity User	13	55
				Medium Intensity User	2.3	9.7
				High Intensity User	0.42	1.4
			Dermal	Low Intensity User	29	N/A

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
				Medium Intensity User	2.9	N/A
				High Intensity User	0.72	N/A
Lubricants and greases	Degreasers - Aerosol and non-aerosol degreasers and cleaners	Section 2.4.2.4.5 and Section 4.3.2.3.1 - Brake Cleaner	Inhalation 1-hr	Low Intensity User	24	202
				Medium Intensity User	1.7	14
				High Intensity User	0.43	2.3
			Inhalation 8-hr	Low Intensity User	50	218
				Medium Intensity User	3.6	15
				High Intensity User	0.56	2.0
			Dermal	Low Intensity User	234	N/A
				Medium Intensity User	4.4	N/A
				High Intensity User	0.32	N/A
		Section 2.4.2.4.8 and Section 4.3.2.3.3 - Carburetor Cleaner	Inhalation 1-hr	Low Intensity User	13	110
				Medium Intensity User	1.4	12
				High Intensity User	0.28	2.0
			Inhalation 8-hr	Low Intensity User	27	118
				Medium Intensity User	3.0	13
				High Intensity User	0.55	2.0
			Dermal	Low Intensity User	158	N/A
				Medium Intensity User	10	N/A
				High Intensity User	1.0	N/A
		Section 2.4.2.4.12 and Section 4.3.2.3.6 - Engine Cleaner	Inhalation 1-hr	Low Intensity User	5.4	47
				Medium Intensity User	0.62	5.1
				High Intensity User	0.16	0.88
Inhalation 8-hr	Low Intensity User		12	50		
	Medium Intensity User		1.3	5.4		
	High Intensity User		0.22	0.77		

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
			Dermal	Low Intensity User	32	N/A
				Medium Intensity User	4.7	N/A
				High Intensity User	0.38	N/A
		Section 2.4.2.4.13 and Section 4.3.2.3.7 - Gasket Remover	Inhalation 1-hr	Low Intensity User	5.9	51
				Medium Intensity User	1.1	9.1
				High Intensity User	0.22	1.4
			Inhalation 8-hr	Low Intensity User	13	55
				Medium Intensity User	2.3	9.7
				High Intensity User	0.42	1.4
			Dermal	Low Intensity User	29	N/A
				Medium Intensity User	2.9	N/A
				High Intensity User	0.72	N/A
Building/ construction materials not covered elsewhere	Cold pipe insulation	Section 2.4.2.4.10 and Section 4.3.2.3.13 - Cold Pipe Insulating Spray	Inhalation 1-hr	Low Intensity User	16	167
				Medium Intensity User	1.6	17
				High Intensity User	0.28	2.2
			Inhalation 8-hr	Low Intensity User	35	194
				Medium Intensity User	3.6	20
				High Intensity User	0.59	2.4
			Dermal	Low Intensity User	325	N/A
				Medium Intensity User	20	N/A
				High Intensity User	8.2	N/A
Arts, crafts, and hobby materials	Crafting glue and cement/concrete	Section 2.4.2.4.1 and Section 4.3.2.3.8 - Adhesives	Inhalation 1-hr	Low Intensity User	199	2188
				Medium Intensity User	12	130
				High Intensity User	0.53	4.2
			Inhalation 8-hr	Low Intensity User	452	2535
				Medium Intensity User	27	150

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
Other Uses			Dermal	High Intensity User	1.1	4.7
				Low Intensity User	372	N/A
				Medium Intensity User	27	N/A
				High Intensity User	6.3	N/A
	Anti-adhesive agent - anti-spatter welding aerosol	Section 2.4.2.4.15 and Section 4.3.2.3.15 - Weld Spatter Protectant	Inhalation 1-hr	Low Intensity User	4.6	51
				Medium Intensity User	0.94	10
				High Intensity User	0.16	1.3
			Inhalation 8-hr	Low Intensity User	11	59
				Medium Intensity User	2.1	12
				High Intensity User	0.35	1.5
			Dermal	Low Intensity User	65	N/A
				Medium Intensity User	8.2	N/A
High Intensity User				3.3	N/A	
Brush Cleaner		Section 2.4.2.4.6 and Section 4.3.2.3.10 - Brush Cleaner	Inhalation 1-hr	Low Intensity User	3956	44077
				Medium Intensity User	786	6209
				High Intensity User	462	1293
	Inhalation 8-hr		Low Intensity User	8981	50216	
			Medium Intensity User	1653	6916	
			High Intensity User	191	919	
	Dermal		Low Intensity User	396	N/A	
			Medium Intensity User	33	N/A	
			High Intensity User	4.7	N/A	
Carbon Remover	Section 2.4.2.4.7 and Section 4.3.2.3.2 - Carbon Remover	Inhalation 1-hr	Low Intensity User	9.5	103	
			Medium Intensity User	0.94	9.7	
			High Intensity User	0.18	1.0	
		Inhalation 8-hr	Low Intensity User	22	119	

Category	Sub Category	Consumer Condition of Use Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 30)	Bystander MOE (benchmark MOE=30)
				Medium Intensity User	2.1	11
				High Intensity User	0.23	0.93
			Dermal	Low Intensity User	38	N/A
				Medium Intensity User	2.9	N/A
				High Intensity User	0.36	N/A

4.2 Environmental Risk

EPA considered fate, exposure, and environmental hazard to characterize environmental risk of methylene chloride. As stated in Section 2.1 Fate and Transport, methylene chloride is not expected to bioconcentrate in biota or accumulate in wastewater biosolids, soil, sediment, or biota. Releases of methylene chloride to the environment, are likely to volatilize to the atmosphere, where it will slowly photooxidize. It may migrate to groundwater, where it will slowly hydrolyze. Additionally, the bioconcentration potential of methylene chloride is low. EPA modeled environmental exposure with surface water concentrations of methylene chloride ranging from almost 0 to 18,100 ppb from facilities releasing the chemical to surface water. Measured surface water concentrations in ambient water range from below the detection limit to 29 ppb. The modeled data represents estimated concentrations near facilities that are actively releasing methylene chloride to surface water, while the reported measured concentrations represent sampled ambient water concentrations of methylene chloride. Differences in magnitude between modeled and measured concentrations may be due to measured concentrations not being geographically or temporally close to known releasers of methylene chloride.

EPA concludes that methylene chloride poses a hazard to environmental aquatic receptors (Section 3.1.5). Amphibians are the most sensitive taxa for both acute and chronic exposures. For acute exposures, a hazard value of 26.3 mg/L was established for amphibians using data on teratogenesis leading to lethality in frog embryos and larvae. For acute exposures, methylene chloride also has toxicity values for fish as low as 99 mg/L and for freshwater aquatic invertebrates as low as 135.8 mg/L. For chronic exposures, methylene chloride has a hazard value for amphibians of 0.9 mg/L, based on teratogenesis and lethality in frog embryos and larvae. For chronic exposures to fish, methylene chloride has hazard values as low as 1.5 mg/L. For chronic exposure to aquatic invertebrates, methylene chloride has a toxicity value of 18 mg/L. In algal species, methylene chloride has toxicity values ranging from 33.1 mg/L to 242 mg/L (with the more sensitive value of 33.1 mg/L used to represent algal species as a whole).

A total of 14 acceptable aquatic environmental hazard studies were identified for methylene chloride. EPA's evaluation of these studies was mostly high or medium during data quality evaluation (see Table 3-1 in Section 3.1.2 and "*Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies CASRN: 75-09-2*"). The *Methylene Chloride (75-09-2) Systematic Review: Supplemental File for the TSCA Risk Evaluation Document* presents details of the data evaluations for each study, including scores for each metric and the overall study score.

Given methylene chloride's conditions of use under TSCA outlined in problem formulation ([U.S. EPA, 2018c](#)), EPA determined that environmental exposures are expected for aquatic species, and risk estimation is discussed in Section 4.2.2.

4.2.1 Risk Estimation Approach

To assess environmental risk, EPA evaluates environmental hazard and exposure data. EPA used modeled exposure data from E-FAST, as well as monitored data from the WQP (www.waterqualitydata.us), to characterize the exposure of methylene chloride to aquatic

species. Environmental risks are estimated by calculating a risk quotients (RQ). As stated previously, modeled data was used to represent surface water concentrations near facilities actively releasing methylene chloride to surface water, while the monitored concentrations were used to represent ambient water concentrations of methylene chloride. RQs were calculated using surface water concentrations and the COCs calculated in the hazard section of this document (Section 3.1.4). The RQ is defined as:

$$\text{RQ} = \text{Predicted Environmental Concentration} / \text{Effect Level or COC}$$

RQs equal to 1 indicate that environmental exposures are the same as the COC. If the RQ is above 1, the exposure is greater than the COC. If the RQ is below 1, the exposure is less than the COC. The COCs for aquatic organisms shown in Table 3-2 and the environmental concentrations described in Section 2.3.2 were used to calculate RQs ([EPA, 1998](#)).

EPA considered the biological relevance of the species that the COCs were based on when integrating the COCs with the location of surface water concentration data to produce RQs. For example, certain biological factors affect the potential for adverse effects in aquatic organisms. Life-history and the habitat of aquatic organisms influences the likelihood of exposure in an aquatic environment. In general, amphibian distribution is limited to freshwater environments. More specifically, those amphibian (*Rana* sp.) species evaluated for hazards resulting from chronic exposure (see Section 3.1.2) generally occupy shallow, vegetated, low-flow, freshwater habitats. In contrast, fish generally occupy a much wider breadth of water body types and habitats. If hazard benchmarks are exceeded by both amphibians and fish from estimated chronic exposures, it provides evidence that the site-specific releases could affect that specific aquatic environment.

Frequency and duration of exposure also affects potential for adverse effects in aquatic organisms. Therefore, the number of days that a COC was exceeded was also calculated using E-FAST as described in Section 2.3.2. The days of exceedance modeled in E-FAST are not necessarily consecutive and could occur sporadically throughout the year. For methylene chloride, continuous aquatic exposures are more likely for the longer exposure scenarios (i.e., 100-365 days/yr of exceedance of a COC), and more of an interval or pulse exposure for shorter exposure scenarios (i.e., 1-99 days/yr of exceedances of a COC). Due to the volatile properties of methylene chloride, it is more likely that a chronic exposure duration will occur when there are long-term consecutive days of release versus an interval or pulse exposure which would more likely result in an acute exposure duration.

4.2.2 Risk Estimation for Aquatic Environment

To characterize potential risk from exposures to methylene chloride, EPA calculated RQs based on modeled data from E-FAST for sites that had surface water discharges of methylene chloride according to DMR and TRI data (see Table 4-4 and Appendix H.2). EPA modeled surface water concentrations of methylene chloride for 121 releases from facilities that manufacture, import and repackage, process, use, and dispose of methylene chloride. Direct releasing facilities (releases from an active facility directly to surface water) were modeled with two scenarios based on a high-end and low-end days of release. Indirect facilities (transfer of wastewater from an active facility to a receiving POTW or non-POTW WWTP facility) were only modeled with a

high-end days of release scenario because it was assumed that the actual release to surface water would mostly occur at receiving treatment facilities, which were assumed to typically operate greater than 20 days/yr. As stated in Section 2.3.1.2.2, the maximum release frequency (250 to 365 days) is based on estimates specific to the facility's condition of use and the low-end release frequency of 20 days of release per year is based on estimated releases that could lead to risk from chronic exposure.

All facilities were modeled in E-FAST and RQs are listed in Appendix H.2. Facilities with RQs and days of exceedance that indicate risk for aquatic organisms (facilities with an acute $RQ \geq 1$, or a chronic $RQ \geq 1$ and 20 days or more of exceedance for the chronic COC) are presented in Table 4-4. There are four recycling and disposal facilities and one WWTP that indicate risk for aquatic organisms. Facilities in other conditions of use had acute and chronic $RQs < 1$, indicating they do not present acute or risk to aquatic organisms from chronic exposure.

Recycling and Disposal

Of the 16 recycling and disposal facilities, there were 4 sites with releases indicating risk to aquatic organisms (either the acute $RQ \geq 1$, or the chronic $RQ \geq 1$ with 20 days or more of exceedance for the chronic COC). One of these facilities had an acute $RQ \geq 1$, indicating risk from acute exposure. This RQ was associated with indirect releases from a recycling and disposal facility, Veolia ES Technical Solutions LLC. The facility transferred methylene chloride for the purpose of wastewater treatment to Clean Harbors POTW. The acute RQ associated with this release was 6.88, indicating the surface water concentration was almost seven times higher than the acute COC. Veolia ES Technical Solutions LLC also transferred methylene chloride to three other facilities; however, those receiving facilities indicated exposures that are less than the concentration of concern. Middlesex County Utilities Authority had an acute $RQ < 1$ (indicating acute exposure is less than the COC), and it was determined after further analysis that Safety-Kleen Systems Inc and Ross Incineration receiving facilities did not release methylene chloride to surface water.

Among the recycling and disposal facilities, there were 4 with releases indicating risk from chronic exposure (where the chronic $RQs \geq 1$ and there were 20 days or more of exceedance). These four facilities had both direct releases to surface water and indirect releases, where waste was transferred to another facility before it was released. The facility with the highest RQ for this OES (chronic $RQ = 201.11$) had an indirect release, the result of a transfer from Veolia ES Technical Solutions LLC to Clean Harbors POTW for wastewater treatment, as mentioned above. It is unclear whether Clean Harbors POTW releases methylene chloride to freshwater or an estuarian environment; however, chronic RQs are greater than or equal to one with 20 days or more of exceedance for amphibians ($RQ = 201.11$ with 250 days of exceedance), fish ($RQ = 119.87$ with 250 days of exceedance), and invertebrates ($RQ = 10.06$ with 200 days of exceedance). Two other indirect releases from Johnson Matthey West and Clean Harbors Deer Park LLC also resulted in chronic $RQs \geq 1$ and involved transfers to Clean Harbors Baltimore (chronic $RQ = 1.63$ and 1.38 , respectively). One direct release from a recycling and disposal facility resulted in an $RQ \geq 1$; Clean Water of New York Inc, had a chronic RQ of 3.92.

As stated previously, the highest modeled release originated from Veolia ES Technical Solutions LLC. The release was transferred to Clean Harbors of Baltimore (modeled concentration of 18,100 ppb). This concentration is many times higher than the next highest surface water

concentration modeled. To calculate this surface water concentration, EPA used TRI data indicating that methylene chloride was transferred to Clean Harbors POTW for wastewater treatment. In the absence of information about how methylene chloride waste was managed or possibly released at Clean Harbors POTW, EPA used a reasonable default assumption for assessing releases to surface water. Because the TRI data indicate methylene chloride was transferred to Clean Harbors Baltimore for wastewater treatment, EPA assumed 54% removal of methylene chloride before it was released to surface water (the assumption EPA uses for the POTW industry sector). Site-specific flow data was not available, so instream flow information representative of industrialized POTWs was used to model subsequent surface water concentrations. It was not indicated in the TRI data whether the chemical was incinerated on-site or underwent some other treatment activity.

Wastewater Treatment Plant (WWTP)

For WWTPs, 1 facility, Long Beach (C) WPCP in Long Beach, NY, had an acute RQ ≥ 1 at 2.23 from a direct release of methylene chloride to surface water. This facility releases methylene chloride into an estuarian environment. Because amphibians reside in freshwater environments, risk for Long Beach (C) WPCP was based on fish. Additionally, a WWTP is likely to be operating at greater than 20 days of release, therefore the RQ associated with the high-end days of release scenario (365 days) is likely more representative of actual conditions. The acute RQ associated with the high-end days of release scenario (365 days) for this site was 0.12, indicating acute exposure is less than the COC. However, RQs from chronic exposure indicated risk with a fish RQ of 2.13 and 365 days of exceedance.

Table 4-4. Modeled Facilities Showing Risk from Acute and/or Chronic Exposure from the Release of Methylene Chloride; RQ Greater Than One are Shown in Bold

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Annual Release (kg)	Days of release ^e	Daily Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC Type	COC (ppb)	Days of Exceedance (days/yr) ^h	RQ
OES: Processing: Formulation											
EUROFINS MWG OPERON LLC LOUISVILLE, KY TRI: 4029WRFNSM1 271P	POTW	Receiving Facility: VEOLIA ENVIRONMENTAL SERVICES TECH SOLUTIONS LLC; Inorganic Chemicals Manuf.	Surface water	5,785	300	19	1659.44	Chronic Amphib.	90	221	18.44
								Chronic Fish	151	181	10.99
								Chronic Invert.	1,800	21	0.92
								Acute Amphib.	2,630	N/A	0.63
SOLVAY - HOUSTON PLANT HOUSTON, TX NPDES: TX0007072	Surface Water	Active Releaser: NPDES TX0007072	Surface water	12	300	0.04	7.15	Chronic Amphib	90	0	0.079
								Chronic Fish	151	0	0.047
								Chronic Invert.	1,800	0	0.004
								Acute Amphib.	2,630	N/A	0.0027
					20	0.58	107.41	Chronic Amphib	90	0	1.19
								Chronic Fish	151	0	0.71
								Chronic Invert.	1,800	0	0.06
								Acute Amphib.	2,630	N/A	0.041
OES: Recycling and Disposal											
JOHNSON MATTHEY WEST DEPTFORD, NJ NPDES: NJ0115843	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	620	250	2	147.01	Chronic Amphib.	90.0	68	1.63
								Chronic Fish	151.0	36	0.97
								Chronic Invert.	1800.0	0	0.08
								Acute Amphib.	2,630	N/A	0.056
CLEAN HARBORS DEER PARK LLC LA PORTE, TX NPDES: TX0005941	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	522	250	2	123.89	Chronic Amphib	90.0	56	1.38
								Chronic Fish	151.0	28	0.82
								Chronic Invert.	1800.0	0	0.07
								Acute Amphib.	2,630	N/A	0.047

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Annual Release (kg)	Days of release ^e	Daily Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC Type	COC (ppb)	Days of Exceedance (days/yr) ^h	RQ
VEOLIA ES TECHNICAL SOLUTIONS LLC MIDDLESEX, NJ NPDES: NJ0127477	Non-POTW WWT	Receiving Facility: MIDDLESEX COUNTY UTILITIES AUTHORITY; NPDES: NJ0020141	Still body	4.40	250	0.018	0.00504	Chronic Amphib.	90	0	5.60E-05
								Chronic Fish	151	0	3.34E-05
								Chronic Invert.	1,800	0	2.80E-06
								Acute Amphib.	2,630	N/A	1.92E-06
		Receiving Facility: Clean Harbors; POTW (Ind.)	Surface water	76,450.66	250	306	18100	Chronic Amphib.	90	250	201.11
								Chronic Fish	151	250	119.87
								Chronic Invert.	1,800	200	10.06
								Acute Amphib.	2,630	N/A	6.88
		Receiving Facility: ROSS INCINERATION SERVICES INC; POTW (Ind.)	NA	NA	NA	NA	NA	Chronic Amphib.	-	-	-
								Chronic Fish	-	-	-
								Chronic Invert.	-	-	-
								Acute Amphib.	-	-	-
		Receiving Facility: SAFETY-KLEEN SYSTEMS INC; POTW (Ind.)	NA	NA	NA	NA	NA	Chronic Amphib.	-	-	-
								Chronic Fish	-	-	-
								Chronic Invert.	-	-	-
								Acute Amphib.	-	-	-
CLEAN WATER OF NEW YORK INC STATEN ISLAND, NY NPDES: NY0200484	Surface Water	Active Releaser (Surrogate): NPDES NJ0000019	Still body	2.38	250	0.01	28.00	Chronic Amphib	90	0	0.31
								Chronic Fish	151	0	0.19
								Chronic Invert.	1,800	0	0.02
								Acute Amphib	2,630	N/A	0.01
				20	0.12	352.94	Chronic Amphib	90	20	3.92	
							Chronic Fish	151	20	2.34	
							Chronic Invert.	1800	0	0.20	
							Acute Amphib	2,630	N/A	0.13	
OILTANKING HOUSTON INC	Surface Water	Active Releaser (Surrogate):	Surface water	1	250	0.003	7.22	Chronic Amphib	90	0	8.02E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Annual Release (kg)	Days of release ^e	Daily Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC Type	COC (ppb)	Days of Exceedance (days/yr) ^h	RQ
HOUSTON, TX NPDES: TX0091855		NPDES TX0065943						Chronic Fish	151	0	4.78E-02
								Chronic Invert.	1,800	0	4.01E-03
								Acute Amphib	2,630	N/A	2.75E-03
								Chronic Amphib	90	0	1.00
								Chronic Fish	151	0	0.60
								Chronic Invert.	1,800	0	0.05
								Acute Amphib	2,630	N/A	0.03
									20	0.041	90.00
OES: WWTP											
LONG BEACH (C) WPCP LONG BEACH, NY NPDES: NY0020567	Surface Water	Active Releaser: NPDES NY0020567	Still water	2,730	365	7	322.14	Chronic Amphib.	90	365	3.58
								Chronic Fish	151	365	2.13
								Chronic Invert.	1,800	0	0.18
								Acute Amphib	2,630	N/A	0.12
								Chronic Amphib	90	20	65.08
								Chronic Fish	151	20	38.79
								Chronic Invert.	1,800	20	3.25
								Acute Amphib.	2,630	N/A	2.23
	20	136.49	5857.02								
<p>i. Facilities actively releasing methylene chloride were identified via DMR and TRI databases for the 2016 reporting year.</p> <p>j. Release media are either direct (release from active facility directly to surface water) or indirect (transfer of wastewater from active facility to a receiving POTW or non-POTW WWTP facility). A wastewater treatment removal rate of 57% is applied to all indirect releases, as well as direct releases from WWTPs.</p> <p>k. If a valid NPDES of the direct or indirect releaser was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.</p> <p>l. EFAST uses either the “surface water” model, for rivers and streams, or the “still water” model, for lakes, bays, and oceans.</p> <p>m. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.</p> <p>n. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.</p> <p>o. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.</p> <p>p. To determine the PDM days of exceedance for still bodies of water, the estimated number of release days should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.</p>											

EPA also used surface water monitoring data from the WQP and from the peer reviewed publicly available literature and grey literature to characterize the risk of methylene chloride to aquatic organisms in ambient water. From the WQP, EPA's STORET data and USGS's NWIS data show an average concentration of methylene chloride of 0.78 ± 1.5 $\mu\text{g/L}$ in surface water. These data reflect 2,286 measurements taken throughout 10 U.S. states between 2013 and 2017. The highest concentration recorded was 29 $\mu\text{g/L}$, measured once in 2016. Very few monitors were positioned downstream of facilities releasing methylene chloride to surface water, and the monitors that were downstream were not close. As stated in Section 2.3.2, three of the monitoring sites were 7.5 to 15.8 miles downstream of two facilities. The remaining monitoring sites were not collocated with facilities. Therefore, the monitored data from these locations reflect concentrations of methylene chloride in ambient water, rather than concentrations near facilities. The monitored data generally show ambient concentrations much lower than the concentrations modeled close to facilities releasing methylene chloride from the E-FAST results. This indicates that risk to aquatic organisms from methylene chloride exposure is more likely proximal to facilities, than in locations farther downstream. Environmental conditions, like wind speed, water depth, and temperature, will affect how long methylene chloride remains in the surface water. As stated previously, the estimated volatilization half-life of methylene chloride is 1.1 hours in a model river and less than 4 days in a model lake.

Table 4-5 shows acute and chronic RQs calculated using the mean surface water concentration from monitoring data. It also shows an acute RQ of 0.0 (with rounding) and chronic RQs of 0.3, 0.2, and 0.0 calculated using the maximum surface water concentration from the monitored data. These data indicate that levels less than the COC were identified in ambient water for amphibians, fish, and aquatic invertebrates exposed to methylene chloride for a chronic duration.

Table 4-5. RQs Calculated using Monitored Environmental Concentrations from WQP

Monitored Surface Water Concentrations (ppb) from 2013-2017	RQ using Acute COC of 2,630 ppb	RQ using Chronic COC of 90 ppb	RQ using Chronic COC of 151 ppb	RQ using Chronic COC of 1,800 ppb
Mean (SD): 0.78 (1.5) ppb	0.0	0.0	0.0	0.0
Maximum: 29 ppb	0.0	0.3	0.2	0.0

To show where facilities releasing methylene chloride to surface water are in relation to monitored data, EPA used the geospatial analysis outlined in Section 2.3 to conduct a watershed analysis. This analysis combined predicted concentrations from modeled facility releases with monitored data from WQP. Overall, there are 28 U.S. states/territories with either a measured concentration (n=10) or a predicted concentration (n=23). At the watershed level, there are 125 HUC-8 areas and 196 HUC-12 areas with either measured or predicted concentrations (Table_Apx E-1 and Table_Apx E-2). The surface water concentrations were compared to the COCs.

Figure 4-1 through Figure 4-5 show where monitored and modeled surface water concentrations exceeded the COCs for amphibians, fish, and invertebrates. Figure 4-1 and Figure 4-2 show exceedances for a maximum days of release scenario, and Figure 4-3 and Figure 4-4 show

exceedances for a 20-days of release scenario. Figure 4-5 shows an area where some monitoring information was co-located with facilities that release methylene chloride to surface water. However, the monitoring samples were not down-stream of the facilities and did not detect methylene chloride in the ambient water.

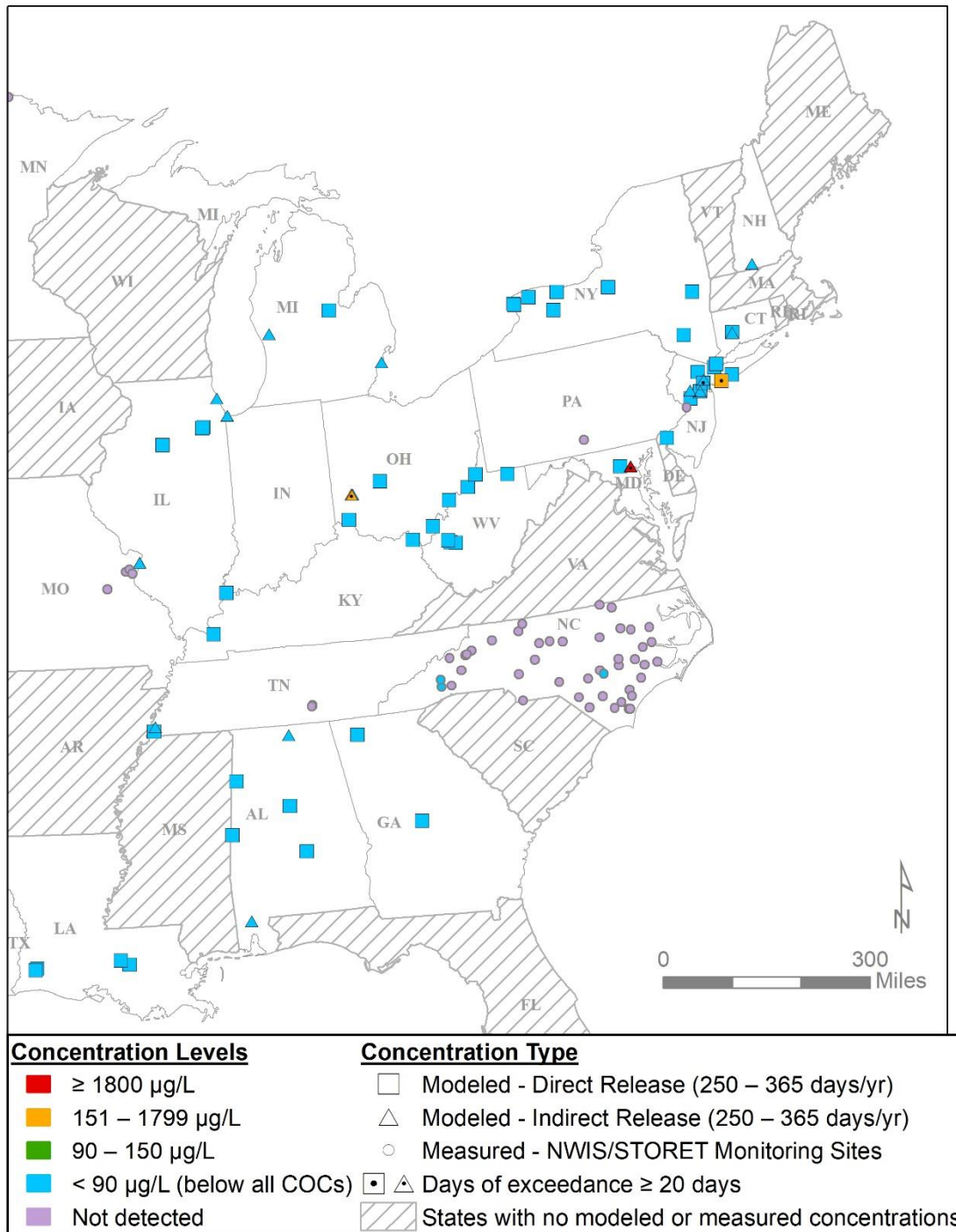


Figure 4-1. Surface Water Concentrations of Methylene Chloride from Releasing Facilities (Maximum Days of Release Scenario) and WQX Monitoring Stations: Year 2016, East U.S.

All indirect releases are mapped at the receiving facility unless the receiving facility is unknown. Puerto Rico and U.S. Virgin Islands not shown due to no modeled releases or measured monitoring information.

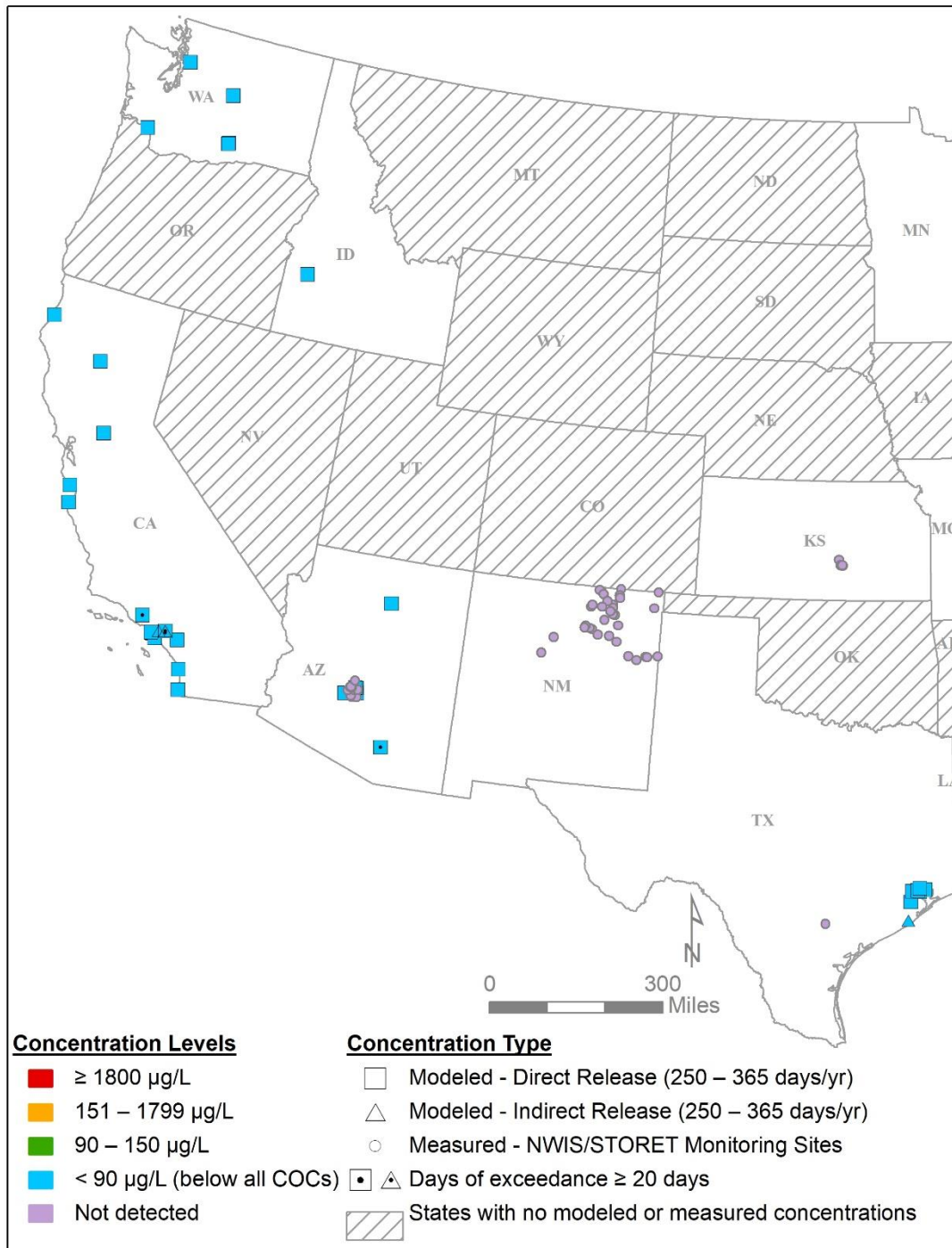


Figure 4-2. Surface Water Concentrations of Methylene Chloride from Releasing Facilities (Maximum Days of Release Scenario) and WQX Monitoring Stations: Year 2016, West U.S.

All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.

Alaska, Hawaii, Guam, N. Mariana Islands and American Somoma not shown due to no modeled releases or measured monitoring information.

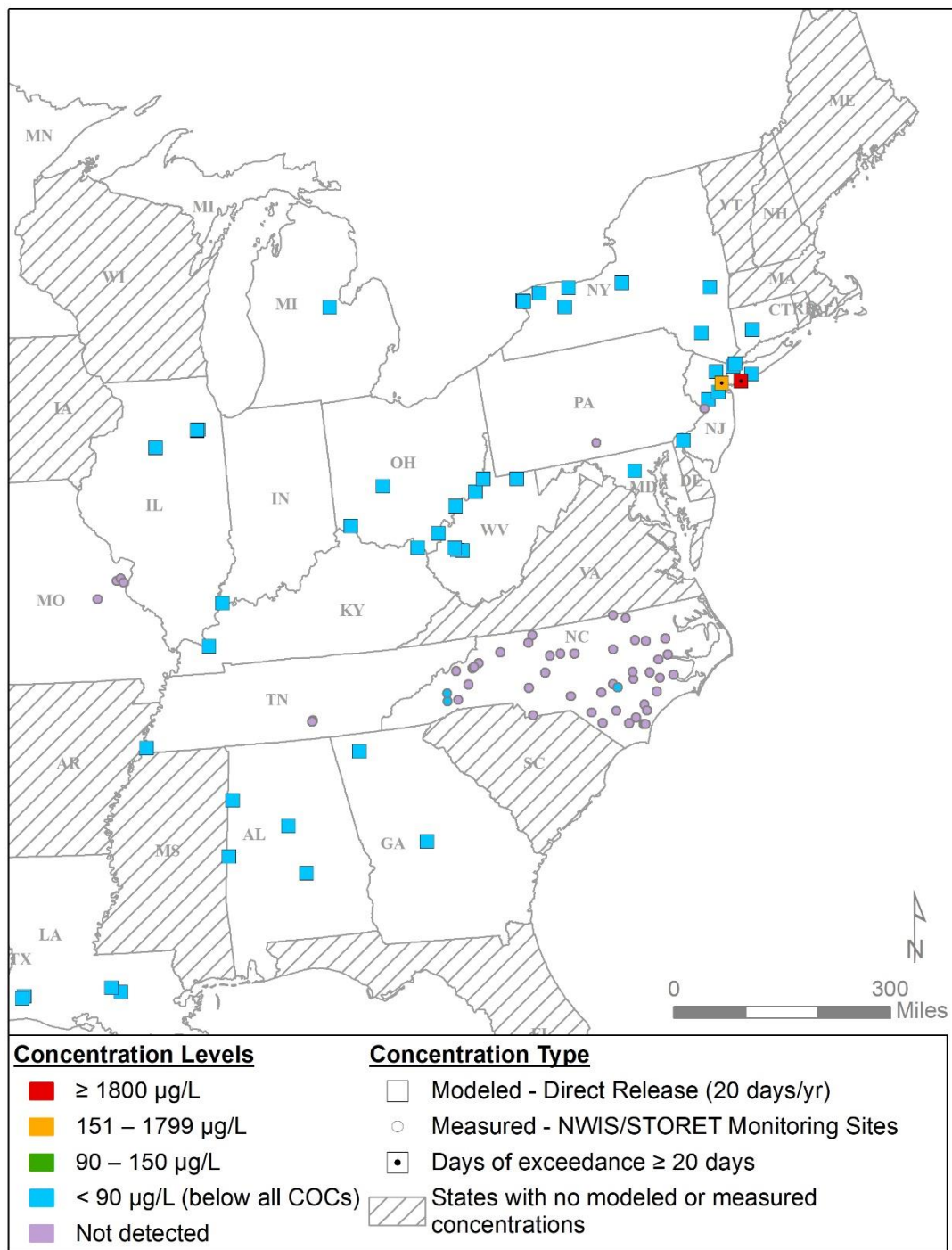


Figure 4-3. Concentrations of Methylene Chloride from Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, East U.S.

Puerto Rico and U.S. Virgin Islands not shown due to no modeled releases or measured monitoring information.

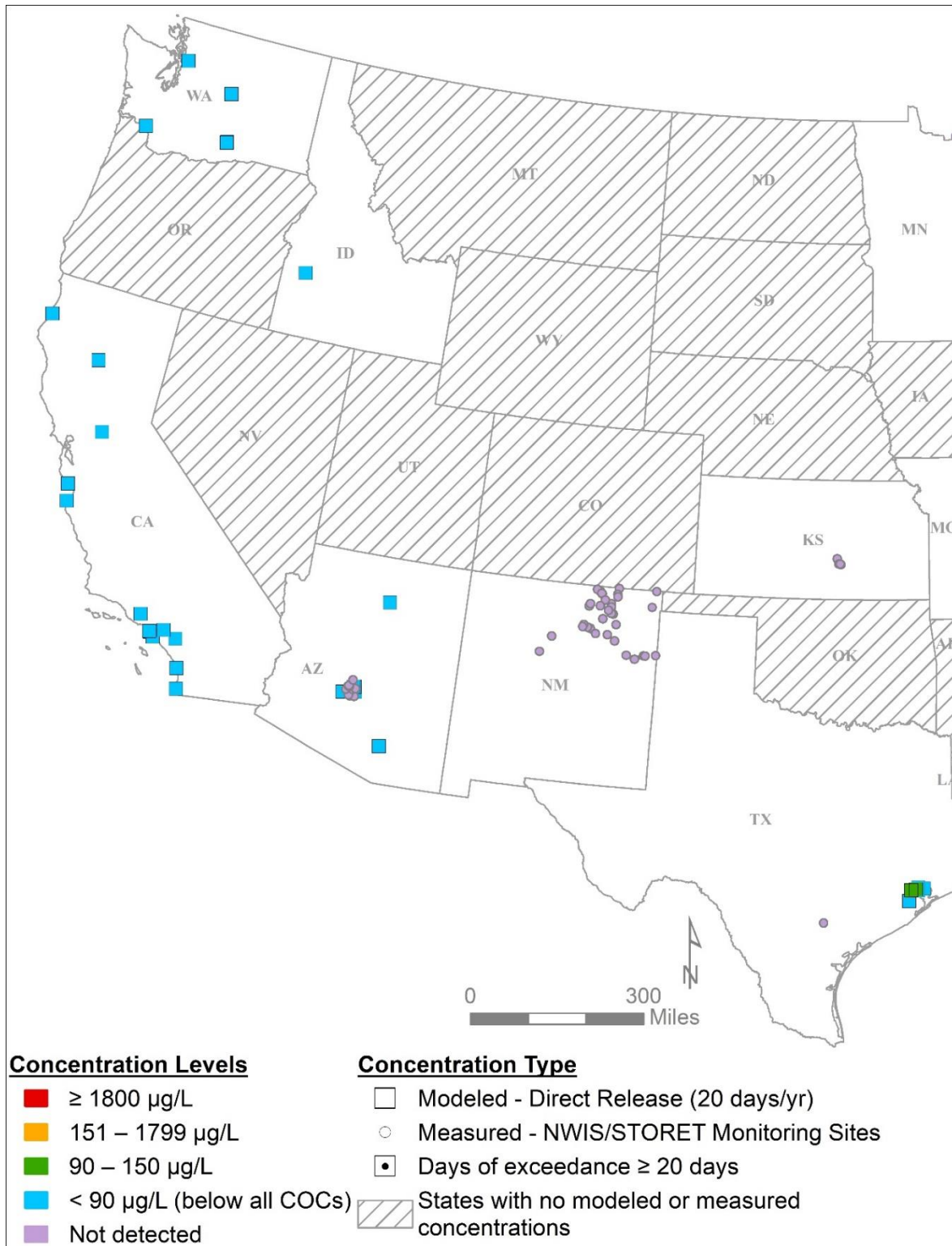


Figure 4-4. Concentrations of Methylene Chloride from Methylene Chloride-Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, West U.S.

Alaska, Hawaii, Guam, N. Mariana Islands and American Somoa not shown due to no modeled releases or measured monitoring information.

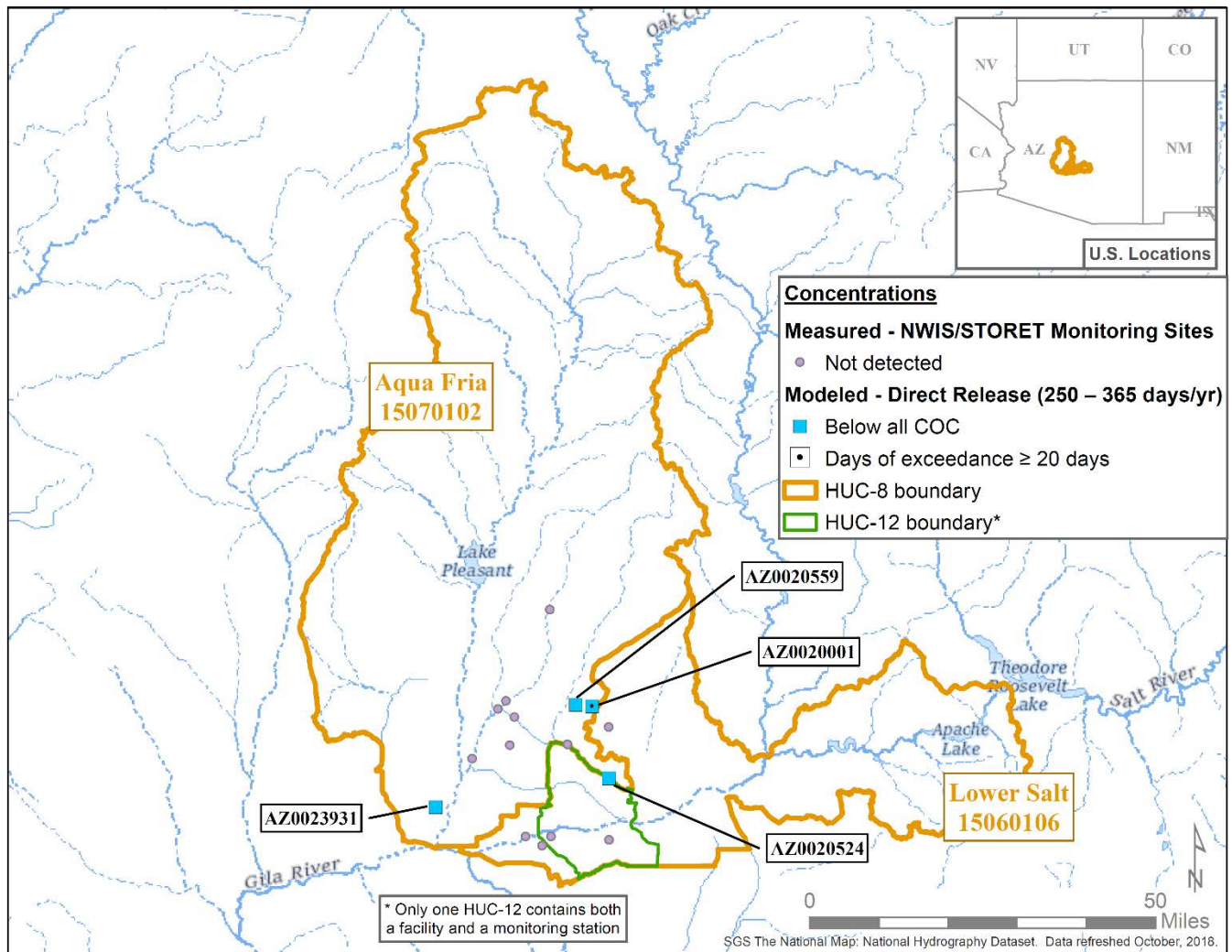


Figure 4-5. Co-location of Methylene Chloride Releasing Facilities and WQX Monitoring Stations at the HUC 8 and HUC 12 Level

4.2.3 Risk Estimation for Sediment

EPA also quantitatively analyzed exposure to sediment organisms. While no ecotoxicity studies were available for sediment-dwelling organisms (e.g., *Lumbriculus variegatus*, *Hyalella azteca*, *Chironomus riparius*), aquatic invertebrates were used as a surrogate species. EPA is uncertain whether methylene chloride is more or less toxic to daphnia than sediment-dwelling species. However, because methylene chloride is not expected to sorb to sediment and will instead remain in pore water, daphnia which feed through the entire water column were deemed to be an acceptable surrogate species for sediment invertebrates. EPA calculated an acute aquatic invertebrate COC of 36,000 ppb, and a chronic aquatic invertebrate COC of 1,800 ppb to address hazards to sediment organisms. Methylene chloride is expected to be in sediment and pore water with concentrations similar to or less than the overlying water due to its water solubility (13 g/L), low partitioning to organic matter ($\log K_{OC} = 1.4$), and biodegradability in anaerobic environments. Thus, methylene chloride concentrations in sediment and pore water are expected to be similar to or less than the concentrations in the overlying water, and concentrations of methylene chloride in the deeper part of sediment, where anaerobic conditions prevail, are expected to be lower.

Therefore, EPA used modeled surface water concentrations to estimate the concentration of methylene chloride in pore water near facilities. EPA also used monitored data to estimate the concentration of methylene chloride in pore water in the ambient water. Comparing aquatic invertebrate data to these exposure numbers, the data showed that there is risk to sediment dwelling organisms near one facility due to chronic exposure. Table 4-4 shows an RQ from chronic exposure near Clean Harbors POTW at $RQ = 10.1$ with 200 days of exceedance for aquatic invertebrates. In ambient water, for both acute and chronic exposures to methylene chloride, the RQs are 0.00 and 0.016, based on the highest ambient surface water concentration of 29 ppb, indicating exposures are less than the COC ($RQs < 0$) to sediment organisms from acute or chronic exposures.

4.2.4 Risk Estimation for Terrestrial

During Problem Formulation EPA conducted a screening level analysis to consider whether pathways of exposure for terrestrial organisms should be further analyzed and determined that terrestrial organism exposures to methylene chloride was not of concern partially based on estimates of soil concentrations several orders of magnitude below concentrations observed to cause effects in terrestrial organisms. EPA did not assess exposure to terrestrial organisms through soil, land-applied biosolids, or ambient air in this Risk Evaluation. Methylene chloride is not expected to partition to or accumulate in soil; rather, it is expected to volatilize to air or migrate through soil into groundwater based on its physical-chemical properties ($\log K_{OC} = 1.4$, Henry's Law constant = $0.00325 \text{ atm}\cdot\text{m}^3/\text{mole}$, vapor pressure = 435 mmHg at 25°C). A screening of hazard data for terrestrial organisms shows potential hazard; however, physical chemical properties do not support an exposure pathway through water and soil pathways to terrestrial organisms. In addition, soil concentrations from the WQP were several orders of magnitude below concentrations observed to cause effects in terrestrial organisms.

Methylene chloride is not anticipated to be retained in biosolids (processed sludge) obtained through wastewater treatment. Most methylene chloride present in the water portion of biosolids following wastewater treatment, processing, and land application would be expected to volatilize into air. Furthermore, methylene chloride is not anticipated to remain in soil, as it is expected to

either volatilize into air or migrate through soil into groundwater. Therefore, the land application of biosolids was not analyzed as a pathway for environmental exposure.

Methylene chloride is expected to volatilize to air, based on physical-chemical properties. However, EPA did not include the emission pathways to ambient air from commercial and industrial stationary sources or associated inhalation exposure of terrestrial species, because stationary source releases of methylene chloride to ambient air are covered under the jurisdiction of the Clean Air Act (CAA). The CAA contains a list of hazardous air pollutants (HAP) and provides EPA with the authority to add to that list pollutants that present, or may present, a threat of adverse human health effects or adverse environmental effects. For stationary source categories emitting HAP, the CAA requires issuance of technology-based standards and, if necessary, additions or revisions to address developments in practices, processes, and control technologies, and to ensure the standards adequately protect public health and the environment. The CAA thereby provides EPA with comprehensive authority to regulate emissions to ambient air of any hazardous air pollutant.

Methylene chloride is a HAP. EPA has issued a number of technology-based standards for source categories that emit methylene chloride to ambient air and, as appropriate, has reviewed, or is in the process of reviewing remaining risks. Because stationary source releases of methylene chloride to ambient air are addressed under the CAA, EPA is not evaluating emissions to ambient air from commercial and industrial stationary sources or associated inhalation exposure of the general population or terrestrial species in this TSCA risk evaluation.

Additionally, based on the Guidance for Ecological Soil Screening Levels ([EPA, 2003a, b](#)) document, for wildlife, relative exposures associated with inhalation and dermal exposure pathways are insignificant compared to direct ingestion of food or water contaminated with methylene chloride (by approximately 1,000-fold). Therefore, volatilization from surface water and biosolids to air of methylene chloride is not a concern for wildlife.

4.3 Human Health Risk

Methylene chloride exposure is associated with a variety of cancer and non-cancer adverse effects deemed relevant to humans for risk estimations for the scenarios and populations addressed in this risk evaluation. Based on a weight-of-evidence analysis of the available toxicity studies from animals and humans, the non-cancer effects selected for risk estimation because of their robustness and sensitivity were neurotoxicity (i.e., CNS depression) from acute exposure and liver toxicity from chronic exposures. The evaluation of cancer includes estimates of risk of lung and liver tumors. Although irritation and burns may result from exposure to methylene chloride, air concentrations leading to eye and respiratory tract irritation are not well established, nor are concentrations resulting in direct contact burns to skin or eyes.

4.3.1 Risk Estimation Approach

Table 4-6, Table 4-7, and Table 4-8 show the use scenarios, populations of interest and toxicological endpoints used for acute exposures for workers, acute exposure for consumers and chronic exposure for workers, respectively.

Table 4-6. Use Scenarios, Populations of Interest and Toxicological Endpoints for Assessing Occupational Risks Following Acute Exposures to Methylene Chloride

Populations and Toxicological Approach	Occupational Use Scenarios of Methylene Chloride
<p>Population of Interest and Exposure Scenario:</p>	<p><i>Users:</i> Adults and youth of both sexes (>16 years old) exposed to methylene chloride during an 8-hr workday^{1, 2}</p> <p><i>Occupational Non-user:</i> Adults and youth of both sexes (>16 years old) indirectly exposed to methylene chloride while being in the same building during product use and further information when available is included in section 2.4.1.2 listed by OES. Workers include 16-year olds because of OSHA work permits.</p>
<p>Health Effects of Concern, Concentration and Time Duration</p>	<p><u>Non-Cancer Health Effects:</u> Acute toxicity CNS depression.</p> <p><i>Hazard Values (PODs) for Occupational Scenarios:</i>^{3,4}</p> <ul style="list-style-type: none"> • 15-min: 478 ppm (1706 mg/m³) • 1-hr: 240 ppm (840 mg/m³) • 8-hrs: 80 ppm (290 mg/m³) <p><u>Cancer Health Effects:</u> Cancer risks following acute exposures were not estimated. Relationship is not known between a single short-term exposure to methylene chloride and the induction of cancer in humans.</p>
<p>Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations</p>	<p>Total UF = 30 (10X UF_H * 3X UH_L)⁵</p>
<p>Notes:</p> <p>¹ It is assumed no substantial buildup of methylene chloride in the body between exposure events due to methylene chloride's short biological half-life (~40 min).</p> <p>² EPA believes that the users of these products are generally adults.</p> <p>³ Exposure estimates were made for 8 hr TWAs for all the conditions of use and when exposure estimates for times shorter than 8 hrs were made the additional PODs (identified above) were used.</p> <p>⁴ In addition to the PODs identified, EPA also compared higher exposure values (≥ 4000 mg/m³) with the NIOSH IDLH value of 7981 mg/m³, which is the value identified as immediately dangerous to life or health (NIOSH, 1994); individuals should not be exposed to this level for any length of time.</p> <p>⁵ UF_H=intraspecies UF; UFL=LOAEL to NOAEL UF</p>	

Table 4-7. Use Scenarios, Populations of Interest and Toxicological Endpoints for Assessing Consumer Risks Following Acute Exposures to Methylene Chloride

Use Scenarios Populations and Toxicological Approach	CONSUMER USES
Population of Interest and Exposure Scenario: <i>Users</i>	Adults of both sexes (>16 years old) typically exposed to methylene chloride.
Population of Interest and Exposure Scenario: <i>Bystander</i>	Individuals of any age indirectly exposed to methylene chloride while being in the rest of the house during product use see Section 2.4.2 for more information.
Health Effects of Concern, Concentration and Time Duration	<p><u>Non-Cancer Health Effects:</u> CNS effects</p> <p><i>Hazard Values (PODs) for Consumer Scenarios³:</i></p> <ul style="list-style-type: none"> • 15-min: 478 ppm (1706 mg/m³) • 1-hr: 240 ppm (840 mg/m³) • 8-hrs: 80 ppm (290 mg/m³) <p><u>Cancer Health Effects:</u> Cancer risks following acute exposures were not estimated.</p>
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	Total UF = 30 (10X UF _H * 3X UH _L) ⁴
<p>Notes:</p> <p>¹ It is assumed no substantial buildup of methylene chloride in the body between exposure events due to methylene chloride's short biological half-life (~40 min).</p> <p>² EPA believes that the users of these products are generally adults, but younger individuals may be users of methylene chloride products</p> <p>³ In addition to the PODs identified, EPA also compared higher exposure values (≥ 4000 mg/m³) with the NIOSH IDLH value of 7981 mg/m³, which is the value identified as immediately dangerous to life or health (NIOSH, 1994); individuals should not be exposed to this level for any length of time.</p> <p>⁴ UF_H= intraspecies UF; UFL=LOAEL to NOAEL UF</p>	

Table 4-8. Use Scenarios, Populations of Interest and Toxicological Endpoints for Assessing Occupational Risks Following Chronic Exposures to Methylene Chloride

Use Scenarios Populations And Toxicological Approach	OCCUPATIONAL USE	
Population of Interest and Exposure Scenario: <i>Users</i>	Adults of both sexes (>16 years old) exposed to methylene chloride during an 8-hr workday for up to 250 days/yr for as many as 40 working years depending on the occupational scenario ^{1, 2, 3}	
Population of Interest and Exposure Scenario: <i>Non-user</i>	Adults of both sexes (>16 years old) indirectly exposed to methylene chloride while being in the same building during product use. ³	
Health Effects of Concern, Concentration and Time Duration	<i>Hazard Value (PODs) for Non-Cancer Effects (liver effects):</i> 1 st percentile HEC i.e., the HEC ₉₉ : HEC i.e., the HEC ₉₉ : 17.2 mg/m ³ (4.8 ppm) for 24 hr/day exposure	<i>Hazard Value (PODs) for Cancer Effects (liver and lung tumors):</i> IUR: 1.38 x 10 ⁻⁶ per mg/m ³ for 40 hr work week
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	UF for the HEC ₉₉ = 10 (3X UF _A * 3X UH _H) UF is not applied for the cancer risk calculations.	
Notes: ¹ It is assumed no substantial buildup of methylene chloride in the body between exposure events due to methylene chloride's short biological half-life (~40 min). ² EPA believes that the users of these products are generally adults. ³ A range of working years were evaluated from 31 – 40 years, see Section 2.4.1.1. ⁴ Data sources did not often indicate whether exposure concentrations were for occupational users or non-users. Therefore, EPA assumed that exposures were for a combination of users and non-users. Some non-users may have lower exposures than users, especially when they are further away from the source of exposure.		

Acute or chronic MOEs (MOE_{acute} or $MOE_{chronic}$) were used in this assessment to estimate non-cancer risks using Eq. 4-1

(Eq. 4-1)

Equation to Calculate Non-Cancer Risks Following Acute or Chronic Exposures Using MOEs

$$MOE_{acute\ or\ chronic} = \frac{\text{Non - cancer Hazard value (POD)}}{\text{Human Exposure}}$$

Where:

MOE = Margin of exposure (unitless)

Hazard value (POD) = POD or HEC (mg/m^3 or $mg/kg/day$)

Human Exposure = Exposure estimate (mg/m^3 or $mg/kg/day$) from occupational or consumer exposure assessment (see Section 2.4).

EPA used MOEs²² to estimate risks from acute and chronic exposure for non-cancer effects based on the following:

1. the endpoint/study-specific UFs applied to the HECs per EPA Guidance ([EPA, 2002](#)); and
2. the exposure estimates calculated for methylene chloride uses examined in this risk evaluation (see Section 2.4).

MOEs allow for the presentation of a range of risk estimates. The OES considered both acute and chronic exposures. All consumer uses considered only acute exposure scenarios. Different adverse endpoints were determined to be appropriate based on the expected exposure durations. For non-cancer effects, risks for acute effects (neurotoxicity) were evaluated for acute (short-term) exposures, whereas risks for liver toxicity were evaluated for repeated (chronic) exposures to methylene chloride. For cancer, risks for chronic effects are based on lung and liver tumors. EPA discusses other effects in Sections 3.2.3 and 3.2.4.

For occupational exposure calculations, the 8 hr TWA was used to calculate MOEs for risk estimates for acute and chronic exposures. When shorter duration exposure estimates were available (e.g., 15 minutes or 1 hr), these were used to calculate MOEs for risk estimates for acute exposures. EPA selected exposure durations of 15 mins and 1 hr, in addition to the 8-hr duration to represent a reasonable range of acute exposure durations. Also, in one fatality case report, the exposed individual was found dead 20-30 mins after the individual had been observed alive ([Nac/Aegl, 2008b](#)). Even though the individual may have been exposed for some time prior to being still observed alive, additional information was not available and thus, the total exposure time could have been limited. Finally, 15 mins matches the duration of the OSHA STEL. For these reasons, EPA is presenting this range of acute durations when exposure data are available to calculate such risks.

²² Margin of Exposure (MOE) = (Non-cancer hazard value, POD) ÷ (Human Exposure). Equation 4-1. The benchmark MOE is used to interpret the MOEs and consists of the total UF shown in Table 4-3, Table 4-4 and Table 4-5.

The total UF for each non-cancer POD was developed as the benchmark MOE used to interpret the MOE risk estimates for each use scenario. The MOE estimate was interpreted as a human health risk if the MOE estimate was less than the benchmark MOE (i.e., the total UF). On the other hand, the MOE estimate indicated negligible concerns for adverse human health effects if the MOE estimate was equal to or exceeded the benchmark MOE. Typically, the larger the MOE, the more unlikely it is that a non-cancer adverse effect would occur.

Extra cancer risks for chronic exposures to methylene chloride were estimated using Eq 4-2. Estimates of extra cancer risks should be interpreted as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e., incremental or extra individual lifetime cancer risk).

(Eq. 4-2)

Equation to Calculate Extra Cancer Risks

$$\text{Risk} = \text{Human Exposure} \times \text{Slope Factor}$$

Where:

Risk = Extra cancer risk (unitless)

Human exposure = Exposure estimate (mg/m³ or mg/kg/day) from occupational exposure assessment

Slope Factor = Inhalation unit risk (1.38E-06 per mg/m³) or
Dermal slope factor (1.1 x 10⁻⁵ per mg/kg/day)

Exposures to methylene chloride were evaluated by inhalation and dermal routes separately. Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers.

4.3.2 Risk Estimation for Inhalation and Dermal Exposures

The acute inhalation and dermal risk assessment used CNS effects to evaluate the risks from acute exposure for consumer and occupational use of methylene chloride. Both non-cancer liver effects and cancer liver and lung tumors were used to evaluate risk from chronic exposure. Non-cancer risk estimates were calculated with equation 4-1 and cancer risks were calculated with equation 4-2.

4.3.2.1 Risk Estimation for Inhalation Exposures to Workers

4.3.2.1.1 Occupational Inhalation Exposure Summary and PPE Use Determination by OES

EPA considered all reasonably available data for estimating exposures for each OES. EPA also determined whether air-supplied respirator use up to APF = 50 was plausible for those OES based on expert judgement and reasonably available information. Table 4-9 presents this information below, which is considered in the risk characterization for each OES in the following sections.

Table 4-9. Inhalation Exposure Data Summary and Respirator Use Determination

Occupational Exposure Scenario	Inhalation Exposure Approach	Number of Data Points	Model Used	Approach for ONUs	Respirator Use	Industrial or Commercial OES
Manufacturing	Monitoring data	438 (15 min, 30 min, 1-hr, 8-hr and 12-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Processing as a Reactant	Monitoring data	30 (15 min, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Processing – Incorporation into Formulation, Mixture, or Reaction Product	Monitoring data	55 (8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Repackaging	Monitoring data	9 (30 min, 1-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Waste Handling, Disposal, treatment, and Recycling	Monitoring data	30 (30 min, 2-hr, 3-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial

Occupational Exposure Scenario	Inhalation Exposure Approach	Number of Data Points	Model Used	Approach for ONUs	Respirator Use	Industrial or Commercial OES
Batch Open-Top Vapor Degreasing	Model	N/A – model only	Batch Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	May use respirators	Industrial
Conveyorized Vapor Degreasing	Model	N/A – model only	Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	May use respirators	Industrial
Cold Cleaning	Monitoring data supplemented by model	≥3 (8-hr TWA)	Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	Monitoring data supplemented by model	21 (8-hr TWA)	Aerosol Degreasing Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	May use respirators	Commercial
Adhesives and Sealants	Monitoring data	103 for non-spray (15 min, 8-hr), 25 for spray (15 min, 1-hr, 8-hr TWA), and 468 for unknown application (8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial

Occupational Exposure Scenario	Inhalation Exposure Approach	Number of Data Points	Model Used	Approach for ONUs	Respirator Use	Industrial or Commercial OES
Paint and Coatings	Monitoring data	36 for spray (15 min, 30 min, 8-hr TWA) and 271 for unknown application (15 min, 30 min, 1-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial/ Commercial
Paint and Coating Removers	Monitoring data	>1,342 (15 min, 30 min, 1-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial/ Commercial
Adhesives and Caulk Removers	Surrogate Monitoring data for Paint Stripping by Professional Contractors	>42 (\leq 1-hr, 2-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Commercial
Miscellaneous Non-Aerosol Commercial and Industrial Uses	Monitoring data	108 (8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial/ Commercial
Fabric Finishing	Monitoring data	41 (3-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift); 1 ONU data point	May use respirators	Industrial/ Commercial

Occupational Exposure Scenario	Inhalation Exposure Approach	Number of Data Points	Model Used	Approach for ONUs	Respirator Use	Industrial or Commercial OES
Spot Cleaning	Monitoring data	18 (8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Commercial
Cellulose Triacetate Film Production	Monitoring data	>166 (8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Plastic Product Manufacturing	Monitoring data	85 (83 workers and 2 ONUs, 15 min, 30 min, 8-hr TWA)	N/A – monitoring data only	ONU monitoring data available	May use respirators	Industrial
Flexible Polyurethane Foam Manufacturing	Monitoring data	92 (30 min, 6-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Laboratory Use	Monitoring data	103 (15 min, 30 min, 1-hr, 2-hr, 3-hr, 4-hr, 8-hr)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Industrial
Lithograph Printing Plate Cleaning	Monitoring data	>130 (4-hr, 8-hr TWA)	N/A – monitoring data only	Equal to workers (assumes employees may be workers or ONUs throughout their shift)	May use respirators	Commercial

4.3.2.1.2 Manufacturing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for manufacturing are presented in Table 4-10, Table 4-11, and Table 4-12, respectively. For manufacturing exposure estimates for TWAs of 15 mins, 1 hr, and 8 hrs, are available based on personal monitoring data samples, including 136 data points from 2 sources ([Halogenated Solvents Industry Alliance, 2018](#)). The 15 mins and 1 hr TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins and 1 hr TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride manufacturing. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.1. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to high. Section 2.4.1.2.1 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-10. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Manufacturing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	63	1575	30
		Central Tendency	795	19878	
15-minute	1706	High End	9.3	232	30
		Central Tendency	179	4465	
1-hr	840	High End	53	1314	30
		Central Tendency	197	4935	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-11. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Manufacturing

Endpoint ³	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver effects	17.2	High End	16	409	10
		Central Tendency	207	5164	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-12. Risk Estimation for Chronic, Cancer Inhalation Exposures for Manufacturing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	3.26E-06	1.30E-07	10 ⁻⁴
		Central Tendency	2.00E-07	8.00E-09	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

For acute inhalation exposures, MOEs are greater than benchmark MOEs for workers when respirators are not worn for all exposure scenarios except for the 15-minute estimate without a respirator for high end exposures and the consistency across multiple exposure durations adds further support to identifying MOEs greater than benchmark MOEs. The OSHA STEL is 433 mg/m³ as a 15-min TWA. In an alternative approach, EPA calculated central tendency and high end values for the measurements lower than the STEL. Since, only one sample of 486 mg/m³ among the 148 15-min samples exceeded the STEL, the high-end concentration values changed, from 184 to 183 mg/m³ and risk estimate did not change for the 15-min exposure.

For chronic inhalation exposures, the MOEs are greater than benchmark MOEs for all exposure scenarios.

For chronic inhalation exposures, cancer risks are less than 10⁻⁴ for all exposure scenarios.

Overall, there is medium confidence in the exposure and hazard estimates that make up the risk estimates and the risk estimates for acute, chronic and cancer indicate negligible concerns for adverse human health effects.

4.3.2.1.3 Processing as a Reactant

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for processing as a reactant are presented in Table 4-13, Table 4-14, and Table 4-15, respectively. For processing as a reactant exposure estimates for TWAs of 15 min and 8 hrs are available based on personal monitoring data samples, including 29 data points from two sources ([Halogenated Solvents Industry Alliance, 2018](#)); ([Finkel, 2017](#)). The 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride processing as a reactant. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.2. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to high. Section 2.4.1.2.2 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-13. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Processing as a Reactant

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ⁴	
8-hr	290	High End	2.7	67	30
		Central Tendency	178	4441	
15-min	1706	Point Estimate ³	4.9	122	30

¹ Data from Putz et al. ([1979](#))

² Exposures to ONUs were not able to be estimated separately from workers.

³ Exposure data were not available to characterize the central tendency and high-end exposures.

⁴ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-14. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Processing as a Reactant

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposure		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver Effects	17.2	High End	0.70	17	10
		Central Tendency	46	1154	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-15. Risk Estimation for Chronic, Cancer Inhalation Exposures for Processing as a Reactant

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates	Benchmark
			Worker & ONU ² No respirator ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	7.63E-05	10 ⁻⁴
		Central Tendency	8.95E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ Cancer risks with respirators not shown based on cancer risks without respirators are less than the benchmark cancer risk of 10⁻⁴.

4.3.2.1.4 Processing - Incorporation into Formulation, Mixture, or Reaction Product

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for processing - incorporation into formulation, mixture, or reaction product are presented in Table 4-16, Table 4-17, and Table 4-18, respectively. For processing - incorporation into formulation, mixture, or reaction product exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including a range of values for more than 55 samples from four sources (EPA, 1985); (Finkel, 2017). The 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride processing - incorporation into formulation, mixture, or reaction product. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.3. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational

inhalation estimates in this scenario is medium. Section 2.4.1.2.3 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-16. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Processing - Incorporation into Formulation, Mixture, or Reaction Product

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposure			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ⁴	Worker APF 50 ⁴	
8-hr	290	High End	0.54	13.5	27	30
		Central Tendency	2.9	71.3	143	
15-min	1706	Point Estimate ³	9.5	237	474	30

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ Exposure data were not available to characterize the central tendency and high-end exposures.

⁴ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

The MOEs are less than the benchmark MOE for high end exposures and the estimated 15-minute exposure when respirators are not worn. The MOEs are greater than benchmark MOEs when respirators APF 25 are worn except for high end exposure estimates, which are less than the benchmark at both APF 25 and 50.

Table 4-17. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Processing - Incorporation into Formulation, Mixture, or Reaction Product

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposure			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.14	3.5	7.0	10
		Central Tendency	0.74	18.5	37.0	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-18. Risk Estimation for Chronic, Cancer Inhalation Exposures for Processing - Incorporation into Formulation, Mixture, or Reaction Product

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	3.81E-04	1.52E-05	10 ⁻⁴
		Central Tendency	5.58E-05	2.23E-06	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴

4.3.2.1.5 Repackaging

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for repackaging are presented in Table 4-19, Table 4-20, and Table 4-21, respectively. For repackaging exposure estimates for TWAs of 1 hr and 8 hrs are available based on personal monitoring data samples, including 5 data points from 1 source (Unocal Corporation, 1986). The 1 hr TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 1 hr TWA exposures were used for characterization of the risk. EPA assessed the median value as the central tendency and the maximum reported value as the high-end exposure estimate. EPA has not identified data on potential ONU inhalation exposures from methylene chloride repackaging. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.4. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.1 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-19. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Repackaging

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	2.1	53	105	30
		Central Tendency	33	822	1643	
1-hr	840	High End	2.6	64	129	30
		Central Tendency	4.7	118	236	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-20. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Repackaging

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.55	14	27	10
		Central Tendency	8.54	213	427	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-21. Risk Estimation for Chronic, Cancer Inhalation Exposures for Repackaging

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates	Benchmark
			Worker & ONU ² No respirator ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	9.74E-05	10 ⁻⁴
		Central Tendency	4.84E-06	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. Cancer risks with respirators not shown based on cancer risks without respirators are less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.6 Waste Handling, Disposal, Treatment, and Recycling

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for waste handling, disposal, treatment and recycling are presented in Table 4-22, Table 4-23, and Table 4-24, respectively. For waste handling, disposal, treatment and recycling exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 22 data points from four sources ([Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\), 2018](#); [Finkel, 2017](#); [EPA, 1985](#)). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride waste handling, disposal, treatment and recycling. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.20. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a

high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.20 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-22. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	3.6	90	30
		Central Tendency	124	3092	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-23. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Workers APF 25 ³	
Liver Effects	17.2	High End	0.93	23	10
		Central Tendency	32	803	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-24. Risk Estimation for Chronic, Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator		
Cancer Risk Liver and lung tumors	1.38E-06	High End	5.71E-05		10 ⁻⁴
		Central Tendency	1.29E-06		

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE with this condition of use. Cancer risks with APF 25 or APF 50 are not shown based on cancer risks without respirators are less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.7 Batch Open-Top Vapor Degreasing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for batch open-top vapor degreasing are presented Table 4-25, Table 4-26, and Table 4-27, respectively. For batch open-top vapor degreasing exposure estimates for TWAs of 8 hrs are available based on modeling with a near-field and far-field approach. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA used the near-field air concentrations for worker exposures and the far-field air concentrations for potential ONU inhalation exposures from methylene chloride batch open-top vapor degreasing as described in more detail above in Section 2.4.1.2.5. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.5 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-25. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures						Benchmark MOE (= Total UF)
			No respirator		APF 25 ²		APF 50 ²		
			Workers	ONUs	Workers	ONUs	Workers	ONUs	
8-hr	290	High End	0.39	0.64	9.7	N/A	19	N/A	30
		Central Tendency	1.7	3	43	N/A	86	N/A	

¹ Data from Putz et al. (1979)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

N/A = not assessed because ONUs are not assumed to be wearing PPE

Table 4-26. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures						Benchmark MOE (= Total UF)
			Workers No respirator	ONUs No respirator	Workers APF 25 ²	ONUs APF 25 ²	Workers APF 50 ²	ONUs APF 50 ²	
Liver Effects	17.2	High End	0.10	0.2	2.5	N/A	5.1	N/A	10
		Central Tendency	0.45	0.87	11	N/A	22	N/A	

¹ Data from Nitschke et al. (1988a)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

N/A = not assessed because ONUs are not assumed to be wearing PPE

Table 4-27. Risk Estimation for Chronic, Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates				Benchmark
			Workers No respirator	ONUs No respirator	Workers APF 25 ²	ONUs APF 25 ²	
Cancer Risk Liver and lung tumors	1.38E-06	High End	5.27E-04	3.22E-04	2.11E-05	N/A	10 ⁻⁴
		Central Tendency	9.23E-05	4.74E-05	3.69E-06	N/A	

¹ Data from NTP (1986)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

N/A = not assessed because ONUs are not assumed to be wearing PPE

4.3.2.1.8 ConveyORIZED Vapor Degreasing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for conveyORIZED vapor degreasing are presented in Table 4-28, Table 4-29, and Table 4-30, respectively. For conveyORIZED vapor degreasing exposure estimates for TWAs of 8 hrs are available based on modeling with a near-field and far-field approach. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA used the near-field air concentrations for worker exposures and the far-field air concentrations for potential ONU inhalation exposures from methylene chloride conveyORIZED vapor degreasing as described in more detail above in Section 2.4.1.2.6. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.6 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-28. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for ConveyORIZED Vapor Degreasing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Workers No respirator	ONUs No respirator	Workers APF 50 ²	ONUs APF 50 ²	
8-hr	290	High End	0.21	0.32	10.4	N/A	30
		Central Tendency	0.60	1	30	N/A	

¹ Data from Putz et al. (1979)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

N/A = not assessed because ONUs are not assumed to be wearing PPE

Table 4-29. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for ConveyORIZED Vapor Degreasing

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures				Benchmark MOE (= Total UF)
			Workers No respirator	ONUs No respirator	Workers APF 50 ²	ONUs APF 50 ²	
Liver Effects	17.2	High End	0.05	0.1	2.7	N/A	10
		Central Tendency	0.15	0.30	7.7	N/A	

¹ Data from Nitschke et al. (1988a)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

N/A = not assessed because ONUs are not assumed to be wearing PPE

Table 4-30. Risk Estimation for Chronic, Cancer Inhalation Exposures for ConveyORIZED Vapor Degreasing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates				Benchmark
			Workers No respirator	ONUs No respirator	Workers APF 25 ²	ONUs APF 25 ²	
Cancer Risk Liver and lung tumors	1.38E-06	High End	9.87E-04	6.37E-04	2.97E-05	N/A	10 ⁻⁴
		Central Tendency	2.67E-04	1.39E-04	1.04E-05	N/A	

¹ Data from NTP (1986)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

N/A = not assessed because ONUs are not assumed to be wearing PPE

4.3.2.1.9 Cold Cleaning

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for cold cleaning are presented in Table 4-31, Table 4-32, and Table 4-33, respectively. For cold cleaning exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including a range of values from 1 source ([TNO \(CIVO\), 1999](#)). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride cold cleaning. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.7. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.7 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-31. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Cold Cleaning

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	0.29	7.3	15	30
		Central Tendency	1.04	26	52	

¹ Data from Putz et al. ([1979](#))

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-32. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Cold Cleaning

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.08	1.9	3.8	10
		Central Tendency	0.27	7	13	

¹ Data from Nitschke et al. ([1988a](#))

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-33. Risk Estimation for Chronic, Cancer Inhalation Exposures for Cold Cleaning

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	7.08E-04	2.83E-05	1.42E-05	10 ⁻⁴
		Central Tendency	1.54E-04	6.14E-06	3.07E-06	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

4.3.2.1.10 Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for commercial aerosol products are presented in Table 4-34, Table 4-35, and Table 4-36, respectively. For commercial aerosol products exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 21 data points from (Finkel, 2017). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.8 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-34. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Workers and ONUs No respirator	Workers APF 25 ²	
8-hr	290	High End	1.3	32	30
		Central Tendency	48	1201	

¹ Data from Putz et al. (1979)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-35. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Workers and ONU No respirator ²	Workers APF 25 ³	Workers APF 50 ³	
Liver Effects	17.2	High End	0.33	8.3	17	10
		Central Tendency	12	312	625	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-36. Risk Estimation for Chronic, Cancer Inhalation Exposures for Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Workers and ONUs No respirator ²	Workers APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	1.61E-04	6.44E-06	10 ⁻⁴
		Central Tendency	3.31E-06	1.32E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

4.3.2.1.11 Adhesives and Sealants

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for adhesives and sealants are presented in Table 4-37, Table 4-38, and Table 4-39, respectively. For both spray and non-spray industrial adhesive application exposure estimates for TWAs of 15 mins, and 8 hrs are available based on personal monitoring data samples, including 100 data points for non-spray adhesive use (NIOSH, 1985); (EPA, 1985), 16 data points for spray adhesive use from multiple data sources (TNO (CIVO), 1999); (WHO, 1996b); (EPA, 1985), and 468 personal monitoring samples for unknown application (Finkel, 2017). The 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride adhesives and sealants. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.9. EPA calculated risk estimates

assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.9 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer, the respective hazard values and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach. Overall EPA has medium confidence in the acute, chronic and cancer hazard endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-37. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesives and Sealants

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
SPRAY USES						
8-hr	290	High End	0.52	13	26	30
		Central Tendency	7.4	186	372	
15-min	1706	High End	2.6	64	129	30
		Central Tendency	6.0	150	299	
NON-SPRAY USES						
8-hr	290	High End	0.98	25	49	30
		Central Tendency	28	692	1385	
15-min	1706	High End	3.0	75	150	30
		Central Tendency	3.4	86	172	
UNKNOWN APPLICATION						
8-hr	290	High End	0.42	11	21	30
		Central Tendency	10.7	267	533	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-38. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Adhesives and Sealants

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
SPRAY USES						
Liver Effects	17.2	High End	0.14	3.38	6.8	10
		Central Tendency	1.93	48	97	
NON-SPRAY USES						
Liver Effects	17.2	High End	0.25	6.4	13	10
		Central Tendency	7.2	180	360	
UNKNOWN APPLICATION						
Liver Effects	17.2	High End	0.11	2.7	5.5	10
		Central Tendency	2.8	69	139	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-39. Risk Estimation for Chronic, Cancer Inhalation Exposures for Adhesives and Sealants

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
SPRAY						
Cancer Risk Liver and lung tumors	1.38E-06	High End	3.95E-04	1.58E-05	7.90E-6	10 ⁻⁴
		Central Tendency	2.14E-05	8.56E-07	4.28E-7	
NON-SPRAY						
Cancer Risk Liver and lung tumors	1.38E-06	High End	2.10E-04	8.37E-06	4.18E-6	10 ⁻⁴
		Central Tendency	5.74E-06	2.30E-07	1.15E-7	
UNKNOWN APPLICATION						
Cancer Risk Liver and lung tumors	1.38E-06	High End	4.88E-04	1.95E-05	9.75E-06	10 ⁻⁴
		Central Tendency	1.49E-05	5.97E-07	2.98E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

4.3.2.1.12 Paints and Coatings

Risk estimates for methylene chloride-based paint and coating removers were assessed in EPA's 2014 Risk Assessment on Paint Stripping Use for Methylene Chloride ([U.S. EPA, 2014](#)) and those results are included in Appendix L. Risk estimates for use of methylene chloride-based paints and coatings are described in this section.

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for paints and coatings are presented in Table 4-40, Table 4-41, and Table 4-42, respectively. For paints and coatings exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 27 data points from 2 sources ([OSHA, 2019](#)); ([EPA, 1985](#)) and 271 data points from two sources ([Finkel, 2017](#)); [Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\) \(2018\)](#). For paint and coating removers, exposure estimates for TWAs of 8 hrs are available from EPA's 2014 Risk Assessment on Paint Stripping Use for Methylene Chloride ([U.S. EPA, 2014](#)) and from DoD ([Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\), 2018](#)). The DoD data also included 15-min TWAs and these 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride paints and coatings. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.10. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to high. Section 2.4.1.2.10 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-40. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Paints and Coatings Including Commercial Paint and Coating Removers

HEC Time Period Endpoint = CNS Effects ¹ / Exposure Scenario	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Paints and Coatings (Spray)						
8-hr Paints and Coatings	290	High End	0.80	20	40	30
		Central Tendency	4.15	104	208	
Paints and Coatings (Unknown Application)						

HEC Time Period Endpoint = CNS Effects ¹ / Exposure Scenario	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr Paints and Coatings	290	High End	1.1	28	55	30
		Central Tendency	24	588	1176	
Paint and Coating Removers⁴						
Professional Contractors	290	High End ⁵	0.1	2	5	30 ⁶
		Central Tendency ⁵	0.2	5	10	
Automotive Refinishing	290	High End ⁵	0.7	17	35	30 ⁶
		Central Tendency ⁵	1	29	57	
Furniture Refinishing	290	High End ⁵	0.1	3	6	30 ⁶
		Central Tendency ⁵	0.3	6	13	
Art Restoration and Conservation	290	Point estimate ⁷	145	3625	7250	30 ⁶
Aircraft Paint Stripping	290	High End ⁵	0.1	2	4	30 ⁶
		Central Tendency ⁵	0.2	4	7	
Graffiti Removal	290	High End ⁵	0.2	6	12	30 ⁶
		Central Tendency ⁵	0.5	12	24	
Non-Specific Workplace Settings - Immersion Stripping of Wood	290	High End ⁵	0.04	1	2	30 ⁶
		Central Tendency ⁵	0.1	2	4	
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	290	High End ⁵	0.3	7	14	30 ⁶
		Central Tendency ⁵	0.4	9	18	
Non-Specific Workplace Settings - Unknown	290	High End ⁵	0.7	17	34	30 ⁶
		Central Tendency ⁵	0.8	20	41	
DoD Paint Removal 8-hr TWA	290	High End	6.2	154	308	30
		Central Tendency	58	1458	2916	
DoD Paint Removal 15-minute TWA	1706	High End	5.9	147	295	30
		Central Tendency	62	1557	3113	

¹ Data from Putz et al. (1979)² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25 or 50) with this condition of use.

⁴ See Appendix L for the description of exposure and risk estimates

⁵ High-End is the “High” exposure estimate and central tendency is the “midpoint” exposure estimate as described in the 2014 assessment there are not sufficient data to calculate a 50th and 95th percentile for more information see Appendix L and Table L-6.

⁶ While the benchmark used in the 2014 assessment was 60 the benchmark shown here is 30 for consistency with this current evaluation.

⁷ Exposure data were not available to characterize the central tendency and high-end exposures.

Table 4-41. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Paints and Coatings

Liver Effects Endpoint / Exposure Scenario ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Paints and Coatings						
Paints and Coatings	17.2	High End	0.21	5.2	10.3	10
		Central Tendency	1.08	27	54	
Paints and Coatings (Unknown Application)						
Paints and Coatings	17.2	High End	0.29	7.2	14	10
		Central Tendency	6.1	152	505	
Paint and Coating Removers⁴						
Professional Contractors	17.2	High End ⁵	0.025	1	2	10
		Central Tendency ⁵	0.05	1	2	
Automotive Refinishing	17.2	High End ⁵	0.2	5	10	10
		Central Tendency ⁵	0.3	7	14	
Furniture Refinishing	17.2	High End ⁵	0.03	0.8	1.6	10
		Central Tendency ⁵	0.1	2	4	10
Art Restoration and Conservation	17.2	Point estimate ⁶	34	860	1720	10
Aircraft Paint Stripping	17.2	High End ⁵	0.02	0.5	1	10
		Central Tendency ⁵	0.04	1	2	
Graffiti Removal	17.2	High End ⁵	0.1	2	4	10
		Central Tendency ⁵	0.1	3	6	
Non-Specific	17.2	High End ⁵	0.01	0.3	0.6	10

Liver Effects Endpoint / Exposure Scenario ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Workplace Settings - Immersion Stripping of Wood		Central Tendency ⁵	0.02	0.5	1	
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	17.2	High End ⁵	0.07	2	4	10
		Central Tendency ⁵	0.1	2	4	
Non-Specific Workplace Settings - Unknown	17.2	High End ⁵	0.18	4	8	10
		Central Tendency ⁵	0.21	5	10	
DoD Paint Removal	17.2	High End	1.6	40	80	10
		Central Tendency	15	379	757	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). ONUs are not expected to wear respirators.

⁴ See Appendix L for the description of exposure and risk estimates

⁵ High-End is the “High” exposure estimate and central tendency is the “midpoint” exposure estimate shown in Appendix L Tables 3-21 through 3-29

⁶ Exposure data were not available to characterize the central tendency and high-end exposures.

Table 4-42. Risk Estimation for Chronic, Cancer Inhalation Exposures for Paints and Coatings

Cancer Risk Liver and lung tumors ¹ / Exposure Scenario	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Paints and Coatings (Spray)						
Paints and Coatings	1.38E-06	High End	2.58E-04	1.03E-05	5.16E-6	10 ⁻⁴
		Central Tendency	3.83E-05	1.53E-06	7.66E-7	
Paints and Coatings (Unknown Application)						
Paints and Coatings	1.38E-06	High End	1.85E-04	7.40E-06	3.70E-06	10 ⁻⁴
		Central Tendency	6.76E-06	2.7E-07	1.35E-07	
Paint and Coating Removers⁴						
	1E-05 ⁵	High End ⁶	3.9E-3	1.6E-4	8.0E-5	10 ⁻⁴

Cancer Risk Liver and lung tumors ¹ / Exposure Scenario	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Professional Contractors		Central Tendency ⁶	2.0E-3	7.9E-5	4.0E-5	
Automotive Refinishing	1E-05 ⁵	High End ⁶	5.4E-4	2.2E-5	1.1E-5	10 ⁻⁴
		Central Tendency ⁶	3.3E-4	1.3E-5	6.5E-6	
Furniture Refinishing	1E-05 ⁵	High End ⁶	2.9E-3	1.2E-4	6.0E-5	10 ⁻⁴
		Central Tendency ⁶	1.5E-3	5.9E-5	3.0E-5	10 ⁻⁴
Art Restoration and Conservation	1E-05 ⁵	Point estimate ⁷				10 ⁻⁴
Aircraft Paint Stripping	1E-05 ⁵	High End ⁶	5.0E-3	2.0E-4	1.0E-4	10 ⁻⁴
		Central Tendency ⁶	2.5E-3	1.0E-4	5.0E-5	
Graffiti Removal	1E-05 ⁵	High End ⁶	1.6E-3	6.2E-5	3.1E-5	10 ⁻⁴
		Central Tendency ⁶	7.9E-4	3.2E-5	1.6E-5	
Non-Specific Workplace Settings - Immersion Stripping of Wood	1E-05 ⁵	High End ⁶	9.1E-3	3.7E-4	1.9E-4	10 ⁻⁴
		Central Tendency ⁶	4.6E-3	1.8E-4	9.0E-5	
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	1E-05 ⁵	High End ⁶	1.3E-3	5.3E-5	2.7E-5	10 ⁻⁴
		Central Tendency ⁶	1.1E-3	4.3E-5	2.2E-5	
Non-Specific Workplace Settings - Unknown	1E-05 ⁵	High End ⁶	5.6E-4	2.2E-5	1.1E-5	10 ⁻⁴
		Central Tendency ⁶	4.7E-4	1.9E-5	1.0E-5	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1).

⁴ See Appendix L for the description of exposure and risk estimates.

⁵ The IUR used in the 2014 assessment was derived assuming 24 hr/day, 7 day/week exposure and the air concentration exposure estimates were adjusted accordingly. The results of these calculations are shown in this table and described in Appendix L. The IUR used in this evaluation was derived assuming worker exposures of 8 hrs/day, 5 days/week exposure and the air concentration exposure estimates were adjusted accordingly.

⁶ High-End is the "High" exposure estimate and central tendency is the "midpoint" exposure estimate shown in Appendix L Tables 3-12 through 3-20

⁷ Exposure data were not available to characterize the central tendency and high-end exposures.

4.3.2.1.13 Adhesive and Caulk Removers

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for adhesive and caulk removers are presented in Table 4-43, Table 4-44, and Table 4-45,

respectively. EPA did not find specific industry information exposure data for adhesive and caulk removers, based on expected worker activities, EPA assumes that the use of adhesive and caulk removers is similar to paint stripping by professional contractors and used the air concentration data from the 2014 Risk Assessment on Paint Stripping Use for Methylene Chloride ([U.S. EPA, 2014](#)) where overall, four personal monitoring data samples were available. EPA calculated the 50th and 95th percentile 8-hr TWA concentrations to represent a central tendency and high-end estimate of potential occupational inhalation exposures, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride adhesive and caulk removers. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.11. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.11 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

The high-end short-term exposure identified in Section 2.4.1.2.11 (14,000 mg/m³) exceeds the NIOSH IDLH value of 7981 mg/m³ ([NIOSH, 1994](#)) described in Section 3.2.3.1.1. The short-term value identified in Section 2.4.1.2.11 (7100 mg/m³) approaches the IDLH value. The NIOSH IDLH value was set to avoid situations that are immediately dangerous and is a value above which individuals should not be exposed for any length of time.

Table 4-43. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesive and Caulk Removers

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	0.10	2.4	4.9	30
		Central Tendency	0.19	4.8	9.5	

¹ Data from Putz et al. ([1979](#))

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-44. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Adhesive and Caulk Removers

Endpoint ³	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.03	0.63	1.3	10

		Central Tendency	0.05	1.2	2.5	
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¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-45. Risk Estimation for Chronic, Cancer Inhalation Exposures for Adhesive and Caulk Removers

Endpoint, Tumor Types ⁴	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates Cancer Risk			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	2.11E-03	8.44E-05	4.22E-05	10 ⁻⁴
		Central Tendency	8.34E-04	3.33E-05	1.67E-05	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Overall, there is medium confidence in the exposure and hazard estimates that make up the risk estimates and the risk estimates for acute, chronic and cancer all indicate human health hazard concerns and acute and chronic non-cancer concerns even when an APF 50 respirator is used.

4.3.2.1.14 Miscellaneous Non-Aerosol Commercial and Industrial Uses

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for miscellaneous non-aerosol industrial and commercial settings are presented in Table 4-46, Table 4-47, and Table 4-48, respectively. For miscellaneous non-aerosol industrial and commercial settings exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 108 data points from 1 source (EPA, 1985). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride miscellaneous non-aerosol industrial and commercial settings. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.19. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.19 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-46. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Non-Aerosol Commercial and Industrial Uses

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	0.31	7.8	16	30
		Central Tendency	5.1	128	256	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-47. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Non-Aerosol Commercial and Industrial Uses

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.08	2.0	4.0	10
		Central Tendency	1.3	33	66	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-48. Risk Estimation for Chronic, Cancer Inhalation Exposures for Non-Aerosol Commercial and Industrial Uses

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	6.58E-04	2.63E-05	10 ⁻⁴
		Central Tendency	3.11E-05	1.24E-06	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.15 Fabric Finishing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for fabric finishing are presented in Table 4-49, Table 4-50, and Table 4-51, respectively. For fabric finishing exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 39 data points from two sources [OSHA \(2019\)](#); ([Finkel, 2017](#)). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride fabric finishing. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.12. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.12 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-49. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Fabric Finishing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	2.1	53	30
		Central Tendency	37	928	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-50. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Fabric Finishing

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver Effects	17.2	High End	0.56	14	10
		Central Tendency	9.6	241	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because

only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-51. Risk Estimation for Chronic, Cancer Inhalation Exposures for Fabric Finishing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	9.60E-05	3.84E-06	10 ⁻⁴
		Central Tendency	4.29E-06	1.71E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.16 Spot Cleaning

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for spot cleaning are presented in Table 4-52, Table 4-53, and Table 4-54, respectively. For spot cleaning exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 18 data points from 1 source (Finkel, 2017). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride spot cleaning. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.13. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.13 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-52. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Spot Cleaning

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	1.6	39	30
		Central Tendency	436	10,896	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-53. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Spot Cleaning

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver Effects	17.2	High End	0.41	10	10
		Central Tendency	113	2,830	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-54. Risk Estimation for Chronic, Cancer Inhalation Exposures for Spot Cleaning

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	1.31E-04	5.25E-06	10 ⁻⁴
		Central Tendency	3.66E-07	1.46E-08	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.17 Cellulose Triacetate Film Production

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for CTA film production are presented in Table 4-55, Table 4-56, and Table 4-57, respectively. For CTA film production exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including more than 100 data points from 6 studies compiled in 3 sources [Dell et al. \(1999\)](#); [TNO \(CIVO\) \(1999\)](#); [Ott et al. \(1983a\)](#). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride CTA film production. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described

in more detail above in Section 2.4.1.2.14. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.14 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-55. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Cellulose Triacetate Film Production

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures MOE			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	0.21	5.2	10	30
		Central Tendency	0.28	7.0	14	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-56. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Cellulose Triacetate Film Production

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.05	1.3	2.7	10
		Central Tendency	0.07	1.8	3.6	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-57. Risk Estimation for Chronic, Cancer Inhalation Exposures for Cellulose Triacetate Film Production

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	7.67E-04	3.07E-05	10 ⁻⁴
		Central Tendency	5.68E-04	2.27E-05	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10^{-4} .

4.3.2.1.18 Plastic Product Manufacturing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for plastic product manufacturing are presented in Table 4-58, Table 4-59, and Table 4-60, respectively. For plastic product manufacturing exposure estimates for TWAs of 15 mins, and 8 hrs are available based on personal monitoring data samples, including 62 data points from six sources [OSHA \(2019\)](#); [Halogenated Solvents Industry Alliance \(2018\)](#); [Fairfax and Porter \(2006\)](#); [WHO \(1996b\)](#); [General Electric Co \(1989\)](#); [Finkel \(2017\)](#). The 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Based on these strengths and limitations of the worker inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium. EPA has identified 1 data point on potential ONU inhalation exposures from methylene chloride plastic product manufacturing as described in more detail above in Section 2.4.1.2.17. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimate in this scenario is low for ONUs. Section 2.4.1.2.17 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-58. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Plastic Product Manufacturing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures ²			Benchmark MOE (= Total UF)
			Workers No respirator	ONUs No respirator	Workers APF 25 ³	
8-hr	290	High End	1.4	28	35	30
		Central Tendency	34	30	853	
15-minute	1706	High End	13	--	328	30
		Central Tendency	21		517	

¹ Data from Putz et al. (1979)

² This scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures for workers. For ONU 15-minute TWA exposure data were not available to characterize the central tendency and high end.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-59. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Plastic Product Manufacturing

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures ²				Benchmark MOE (= Total UF)
			Workers No respirator	ONUs No respirator	Workers APF 25 ³	Workers APF 50 ³	
Liver Effects	17.2	High End	0.37	7.3	9.1	18	10
		Central Tendency	8.9	7.8	221	443	

¹ Data from Nitschke et al. (1988a)

² This scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures for workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-60. Risk Estimation for Chronic, Cancer Inhalation Exposures for Plastic Product Manufacturing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker No respirator	ONUs No respirator	Worker APF 25 ²	
Cancer Risk Liver and lung tumors	1.38E-06	High End	1.46E-04	7.28E-06	5.83E-06	10 ⁻⁴
		Central Tendency	4.66E-06	5.31E-06	1.87E-07	

¹ Data from NTP (1986)

² APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.19 Flexible Polyurethane Foam Manufacturing

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for flexible polyurethane foam manufacturing are presented in Table 4-61, Table 4-62, and Table 4-63, respectively. For flexible polyurethane foam manufacturing exposure estimates for a TWA of 8 hrs are available based on personal monitoring data samples, including 84 data points from multiple sources ([IARC, 2016](#); [TNO \(CIVO\), 1999](#); [WHO, 1996b](#); [Vulcan Chemicals, 1991](#); [Reh and Lushniak, 1990](#); [EPA, 1985](#); [Cone Mills Corp, 1981a, b](#); [Olin Chemicals, 1977](#)). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride flexible polyurethane foam manufacturing. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.11. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.11 describes the justification for this

occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-61. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Flexible Polyurethane Foam Manufacturing

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
8-hr	290	High End	0.29	7.3	15	30
		Central Tendency	1.5	38	76	

¹Data from Putz et al. (1979)

²Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. ONUs are not expected to wear respirators.

There are short term exposure data that allow estimation of 30-minute exposures (7 data points) and 4-hr exposures (1 data point). Monitoring data to estimate a 15-min or 1-hr TWA exposure were not available.

Table 4-62. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Flexible Polyurethane Foam Manufacturing

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures			Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Liver Effects	17.2	High End	0.08	1.9	3.8	10
		Central Tendency	0.39	9.9	20	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

Table 4-63. Risk Estimation for Chronic, Cancer Inhalation Exposures for Flexible Polyurethane Foam Manufacturing

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates			Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	Worker APF 50 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	7.06E-04	2.83E-05	1.41E-05	10 ⁻⁴
		Central Tendency	1.05E-04	4.19E-06	2.10E-06	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use.

4.3.2.1.20 Laboratory Use

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for laboratory use are presented in Table 4-64, Table 4-65, and Table 4-66, respectively. For laboratory use exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including 76 data points from multiple sources [Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\) \(2018\)](#); [Texaco Inc \(1993\)](#); [Mccammon \(1990\)](#); [OSHA \(2019\)](#); [Finkel \(2017\)](#). The 15 mins TWAs are useful for characterizing exposures shorter than 8 hrs that could lead to adverse CNS effects. PODs specific to 15 mins TWA exposures were used for characterization of the risk. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride laboratory use. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.16. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium to low. Section 2.4.1.2.16 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-64. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Laboratory Use

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	2.8	71	30
		Central Tendency	48	1200	
15-min	1706	High End	22	549	30
		Central Tendency	256	6394	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures for workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-65. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Laboratory Use

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver Effects	17.2	High End	0.74	18	10
		Central Tendency	12	312	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures for workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on MOEs at APF 25 are all greater than the benchmark MOE.

Table 4-66. Risk Estimation for Chronic, Cancer Inhalation Exposures for Laboratory Use

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	7.21E-05	2.89E-06	10 ⁻⁴
		Central Tendency	3.31E-06	1.32E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures for workers.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride and are considered plausible for respirator use. APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10⁻⁴.

4.3.2.1.21 Lithographic Printing Plate Cleaning

Estimates of MOEs for acute and chronic exposures and cancer risks from inhalation for lithographic printing plate cleaning are presented in Table 4-67, Table 4-68, and Table 4-69, respectively. For lithographic printing plate cleaning exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including greater than 130 data points from 4 sources Ukai et al. (1998); EPA (1985); Ahrenholz (1980); (Finkel, 2017). EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified data on potential ONU inhalation exposures from methylene chloride lithographic printing plate cleaning. ONU inhalation exposures are expected to be lower than worker inhalation exposures however the relative exposure of ONUs to workers cannot be quantified as described in more detail above in Section 2.4.1.2.18. EPA calculated risk estimates assuming ONU exposures could be as high as worker exposures as a high-end estimate and there is large uncertainty in this assumption. Considering the overall strengths and limitations of the

data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.2.18 describes the justification for this occupational scenario confidence rating. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach and overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-67. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Lithographic Printing Plate Cleaning

HEC Time Period Endpoint = CNS Effects ¹	Acute HEC (mg/m ³)	Exposure Level	MOEs for Acute Exposures MOE		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
8-hr	290	High End	1.8	45	30
		Central Tendency	33	832	

¹ Data from Putz et al. (1979)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25 or 50) with this condition of use.

Table 4-68. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Lithographic Printing Plate Cleaning

Endpoint ¹	Chronic HEC (mg/m ³)	Exposure Level	MOEs for Chronic Exposures		Benchmark MOE (= Total UF)
			Worker & ONU ² No respirator	Worker APF 25 ³	
Liver Effects	17.2	High End	0.47	12	10
		Central Tendency	8.7	216	

¹ Data from Nitschke et al. (1988a)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25 or 50) with this condition of use.

Table 4-69. Risk Estimation for Chronic, Cancer Inhalation Exposures for Lithographic Printing Plate Cleaning

Endpoint, Tumor Types ¹	IUR (risk per mg/m ³)	Exposure Level	Cancer Risk Estimates		Benchmark
			Worker & ONU ² No respirator	Worker APF 25 ³	
Cancer Risk Liver and lung tumors	1.38E-06	High End	1.13E-04	4.54E-06	10 ⁻⁴
		Central Tendency	4.78E-06	1.91E-07	

¹ Data from NTP (1986)

² Exposures to ONUs were not able to be estimated separately from workers; also, this scenario covers a broad range of industries and processes, which may result in significant differences between central and high-end exposures.

³ APF 25 and APF 50 are the two lowest APF allowable under OSHA standards for methylene chloride. EPA does not assume routine use of PPE that would mitigate risk (respirator APF 25) with this condition of use in part because only supplied air respirators can be used (see section 2.4.1.1). APF 50 not shown based on cancer risks at APF 25 are all less than the cancer risk benchmark of 10^{-4} .

4.3.2.2 Risk Estimation for Dermal Exposures to Workers

Estimates of MOEs for acute and chronic exposures and cancer risks from dermal exposures for workers for all of the OESs are presented in Table 4-70, Table 4-71 and Table 4-72, respectively. EPA calculated exposure estimates as described in more detail above in Section 2.4.1.1.

Considering these primary strengths and limitations, the overall confidence of the dermal dose results is medium. The studies that support the health concerns of acute CNS effects, liver toxicity and cancer and the hazard value and benchmark MOEs are described above in Section 4.3.1 Risk Estimation Approach. EPA conducted route-to-route extrapolation to derive the dermal PODs and uncertainty factors. Overall EPA has medium confidence in the acute, chronic and cancer endpoints. Section 3.2.5.3 describes the justification for these confidence levels.

Table 4-70. MOEs for Acute Dermal Exposures to Workers, by Occupational Exposure Scenario for CNS Effects POD 16 mg/kg/day, Benchmark MOE 30

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs with Glove PFs			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Manufacturing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Processing as a Reactant	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Processing - Incorporation into Formulation, Mixture, or Reaction Product	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Repackaging	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Waste Handling, Disposal, Treatment, and Recycling	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Batch Open-Top Vapor Degreasing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Conveyorized Vapor Degreasing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs with Glove PFs			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Cold Cleaning	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Commercial Aerosol Product Uses	commercial	Central Tendency	1.2	14	68	136	NA
		High-End	3.5	4.5	23	45	NA
Adhesives and Sealants	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Paints and Coatings	industrial/ commercial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Paint and Coating Removers	industrial/ commercial	Central Tendency	1.2	14	68	136	NA
		High-End	3.5	4.5	23	45	NA
Adhesive and Caulk Removers	commercial	Central Tendency	1.1	15	75	151	NA
		High-End	3.2	5.0	25	50	NA
Miscellaneous Industrial Non-Aerosol Use	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Miscellaneous Commercial Non-Aerosol Use	commercial	Central Tendency	1.2	14	68	136	NA
		High-End	3.5	4.5	23	45	NA
Fabric Finishing	industrial/ commercial	Central Tendency	1.1	14	71	143	NA
		High-End	3.4	4.8	24	48	NA
Spot Cleaning	commercial	Central Tendency	1.1	15	75	151	NA
		High-End	3.2	5.0	25	50	NA
CTA Film Manufacturing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Plastic Product Manufacturing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Flexible Polyurethane Foam Manufacturing	industrial	Central Tendency	0.75	21	107	NA	426
		High-End	2.25	7.1	36	NA	142
Laboratory Use	industrial	Central Tendency	1.18	14	68	NA	271
		High-End	3.5	4.5	23	NA	90
	commercial	Central Tendency	1.0	15	77	153	NA

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs with Glove PFs			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Lithographic Printing Plate Cleaner		High-End	3.1	5.1	26	51	NA

NA not assessed because not all PFs are considered relevant to all conditions of use (COUs) and settings, see Section 2.4.1.1

MOEs are less than benchmark MOEs when gloves are not worn for all OESs. When gloves are used MOEs are greater than benchmark MOEs with PF 5 – 10 depending on the OES.

Table 4-71. MOEs for Chronic Dermal Exposures to Workers, by Occupational Exposure Scenario for Liver Effects POD 2.15 mg/kg/day, Benchmark MOE = 10

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs for Different PF			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Manufacturing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Processing as a Reactant	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Processing - Incorporation into Formulation, Mixture, or Reaction Product	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Repackaging	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Waste Handling, Disposal, Treatment, and Recycling	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Batch Open-Top Vapor Degreasing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Conveyorized Vapor Degreasing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Cold Cleaning	industrial	Central Tendency	0.75	3.0	15	NA	60

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs for Different PF			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
		High-End	2.25	1.0	5.0	NA	20
Commercial Aerosol Product Uses	commercial	Central Tendency	1.2	2.7	13	27	NA
		High-End	3.5	0.90	4.4	9.0	NA
Adhesives and Sealants	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Paints and Coatings	industrial/commercial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Paint and Coating Removers	industrial/commercial	Central Tendency	1.2	2.7	13	27	NA
		High-End	3.5	0.90	4.4	9.0	NA
Adhesive and Caulk Removers	commercial	Central Tendency	1.1	3.0	15	30	NA
		High-End	3.2	0.98	4.8	9.7	NA
Miscellaneous Industrial Non-Aerosol Use	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Miscellaneous Commercial Non-Aerosol Use	commercial	Central Tendency	1.2	2.7	13	27	NA
		High-End	3.5	0.90	4.4	9.0	NA
Fabric Finishing	industrial/commercial	Central Tendency	1.1	2.8	14	28	NA
		High-End	3.4	0.93	4.7	9.3	NA
Spot Cleaning	commercial	Central Tendency	1.1	3.0	15	30	NA
		High-End	3.2	0.97	4.8	9.7	NA
CTA Film Manufacturing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Plastic Product Manufacturing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Flexible Polyurethane Foam Manufacturing	industrial	Central Tendency	0.75	3.0	15	NA	60
		High-End	2.25	1.0	5.0	NA	20
Laboratory Use	industrial	Central Tendency	1.2	2.7	13	27	NA
		High-End	3.5	0.90	4.4	9.0	NA
	commercial	Central Tendency	1.0	3.0	15	30	NA

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	MOEs for Different PF			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Lithographic Printing Plate Cleaner		High-End	3.1	1.0	5.0	10	NA

NA not assessed because not all PFs are considered relevant to all COUs and settings, see Section 2.4.1.1

MOEs are less than benchmark MOEs when gloves are not worn for all OESs. When gloves are used MOEs are greater than benchmark MOEs for industrial uses with PF 20. MOEs are less than benchmark MOEs for commercial uses with PF 10.

Table 4-72. Cancer Risk for Chronic Dermal Exposures to Workers, by Occupational Exposure Scenario CSF 1.1×10^{-5} per mg/kg/day

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	Cancer Risk For Different PFs			
				No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10
Manufacturing	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Processing as a Reactant	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Processing - Incorporation into Formulation, Mixture, or Reaction Product	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Repackaging	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Waste Handling, Disposal, Treatment, and Recycling	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Batch Open-Top Vapor Degreasing	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
ConveyORIZED Vapor Degreasing	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	Cancer Risk For Different PFs			
			No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10	PF 20
Cold Cleaning	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Commercial Aerosol Product Uses	commercial	Central Tendency	1.2	4.5E-06	9.0E-07	4.5E-07	NA
		High-End	3.5	1.35E-05	2.70E-06	1.35E-06	NA
Adhesives and Sealants	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Paints and Coatings	industrial/ commercial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Paint and Coating Removers	industrial/ commercial	Central Tendency	1.2	4.5E-06	9.0E-07	4.5E-07	NA
		High-End	3.5	1.35E-05	2.70E-06	1.35E-06	NA
Adhesive and Caulk Removers	commercial	Central Tendency	1.1	4.3E-06	7.3E-07	4.3E-07	NA
		High-End	3.2	1.26E-05	2.51E-06	1.26E-06	NA
Miscellaneous Industrial Non-Aerosol Use	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Miscellaneous Commercial Non-Aerosol Use	commercial	Central Tendency	1.2	4.5E-06	9.0E-07	4.5E-07	NA
		High-End	3.5	1.35E-05	2.70E-06	1.35E-06	NA
Fabric Finishing	industrial/ commercial	Central Tendency	1.1	4.2E-06	8.4E-07	4.2E-07	NA
		High-End	3.4	1.30E-05	2.61E-06	1.30E-06	NA
Spot Cleaning	commercial	Central Tendency	1.1	4.3E-06	7.3E-07	4.3E-07	NA
		High-End	3.2	1.26E-05	2.51E-06	1.26E-06	NA
CTA Film Manufacturing	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Plastic Product Manufacturing	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07
		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
	industrial	Central Tendency	0.75	2.9E-06	5.8E-07	NA	1.45E-07

Occupational Exposure Scenario	Setting	Exposure Level	Exposure (mg/kg/day)	Cancer Risk For Different PFs			
			No Protective Gloves PF 1	No Protective Gloves PF 1	PF 5	PF 10	PF 20
Flexible Polyurethane Foam Manufacturing		High-End	2.25	8.69E-06	1.74E-06	NA	4.35E-07
Laboratory Use	industrial	Central Tendency	1.2	4.5E-06	9.0E-07	4.5E-07	NA
		High-End	3.5	1.35E-05	2.70E-06	1.35E-06	NA
Lithographic Printing Plate Cleaner	commercial	Central Tendency	1.0	3.9E-06	7.8E-07	3.9E-07	NA
		High-End	3.1	1.21E-05	2.41E-06	1.21E-06	NA

NA not assessed because not all PFs are considered relevant to all COUs and settings, see Section 2.4.1.1

Cancer risks are less than 10^{-4} when gloves are not worn for all OESs.

4.3.2.3 Risk Estimation for Inhalation and Dermal Exposures to Consumers

Estimates of MOEs for consumers were calculated for consumers for acute inhalation and dermal exposures, because the exposure frequencies were not considered sufficient to cause the health effects (i.e., liver effects and liver and lung tumors) that were observed in chronic animal studies typically defined as at least 10% of the animal's lifetime.

4.3.2.3.1 Brake Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the brake cleaner consumer use are presented in 4-72 and 4-73, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.5. Inhalation exposures were modeled for 27 different scenarios, and dermal exposure was evaluated for nine scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-73. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Brake Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	24	202	30
		Medium Intensity User	1.7	14	
		High Intensity User	0.43	2.3	
8-hr	290	Low Intensity User	50	218	30
		Medium Intensity User	3.6	15	
		High Intensity User	0.56	2.0	

¹ Data from Putz et al. (1979)

The MOEs are < benchmark MOE for the 1 hr and 8 hr value high end and medium exposure scenarios. Most MOEs are > benchmark MOE for the low exposures.

Table 4-74. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Brake Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.068	234	30
		Medium Intensity User	3.6	4.4	
		High Intensity User	49	0.32	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.2 Carbon Remover

Estimates of MOEs for acute inhalation and dermal exposures for the carbon remover consumer use are presented in Table 4-75 and Table 4-76, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.7. Inhalation exposures were modeled for 18 different scenarios and dermal exposure evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups)

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate, as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-75. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Carbon Remover Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	9.5	103	30
		Medium Intensity User	0.94	9.7	
		High Intensity User	0.18	1.0	
8-hr	290	Low Intensity User	22	119	30
		Medium Intensity User	2.1	11	
		High Intensity User	0.23	0.93	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low exposure bystanders.

The peak exposure value (4940 mg/m³) and the 1-hr maximum TWA (4750 mg/m³) for the high intensity user identified in Section 2.4.2.4.7 do not exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994) described in Section 3.2.3.1.1 but are greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

Table 4-76. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Carbon Remover Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.42	38	30
		Medium Intensity User	5.5	2.9	
		High Intensity User	43.9	0.36	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.3 Carburetor Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the carburetor cleaner consumer use are presented in Table 4-77 and Table 4-78, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.8. Inhalation exposures were modeled for 27 different scenarios and dermal exposure was evaluated for nine scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-77. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Carburetor Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	13	110	30
		Medium Intensity User	1.4	12	
		High Intensity User	0.28	2.0	
8-hr	290	Low Intensity User	27	118	30
		Medium Intensity User	3.0	13	
		High Intensity User	0.55	2.0	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low exposure bystanders.

The peak exposure value (4420 mg/m³) for the high intensity user identified in Section 2.4.2.4.8 does not exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994) described in Section 3.2.3.1.1 but is greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

Table 4-78. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Carburetor Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.10	158	30
		Medium Intensity User	1.6	10	
		High Intensity User	16	1.0	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.4 Coil Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the coil cleaner consumer use are presented in 4-78 and 4-79, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.9. Inhalation exposures were modeled for 18 different scenarios and dermal exposure evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-79. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Coil Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	5.5	60	30
		Medium Intensity User	0.57	5.9	
		High Intensity User	0.11	0.61	
8-hr	290	Low Intensity User	13	69	30
		Medium Intensity User	1.3	6.8	

		High Intensity User	0.14	0.57	
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¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low exposure bystanders at 8 hrs.

The peak exposure value (8080 mg/m³) and the 1-hr maximum TWA (7770 mg/m³) for the high intensity user identified in Section 2.4.2.4.9 exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994). The peak exposure value (4330 mg/m³) for the moderate intensity user (Section 2.4.2.4.9) does not exceed the NIOSH IDLH but is greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

Table 4-80. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Coil Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.72	22	30
		Medium Intensity User	9.0	1.8	
		High Intensity User	72	0.22	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for all the exposure scenarios.

4.3.2.3.5 Electronics Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the electronics cleaner consumer use are presented in Table 4-81 and Table 4-82, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.11. Inhalation exposures were modeled for nine different scenarios and dermal exposure evaluated for three scenarios (combinations of the duration of use and a single identified weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section

4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-81. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Electronics Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	1171	8027	30
		Medium Intensity User	91	633	
		High Intensity User	6.5	31	
8-hr	290	Low Intensity User	2492	10794	30
		Medium Intensity User	195	854	
		High Intensity User	13	46	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures for high intensity users and high intensity bystanders at 1 hr.

Table 4-82. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Electronics Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.013	1208	30
		Medium Intensity User	0.049	328	
		High Intensity User	0.25	64	

For acute dermal exposures, MOEs are greater than the benchmark MOE for consumer users for all the exposure scenarios.

4.3.2.3.6 Engine Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the engine cleaner consumer use are presented in Table 4-83 and Table 4-84, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.12. Inhalation

exposures were modeled for 27 different scenarios and dermal exposure evaluated for nine scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-83. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Engine Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	5.4	47	30
		Medium Intensity User	0.62	5.1	
		High Intensity User	0.16	0.88	
8-hr	290	Low Intensity User	12	50	30
		Medium Intensity User	1.3	5.4	
		High Intensity User	0.22	0.77	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low exposure bystanders.

Table 4-84. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Engine Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.51	32	30
		Medium Intensity User	3.4	4.7	
		High Intensity User	42	0.38	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

The peak exposure value (5480 mg/m³) and the 1-hr maximum TWA (5100 mg/m³) for the high intensity user identified in Section 2.4.2.4.12 do not exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994) described in Section 3.2.3.1.1 but are greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

4.3.2.3.7 Gasket Remover

Estimates of MOEs for acute inhalation and dermal exposures for the gasket remover consumer use are presented in Table 4-85 and Table 4-86, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.13. Inhalation exposures were modeled for 18 different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate, as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-85. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Gasket Remover Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	5.9	51	30
		Medium Intensity User	1.1	9.1	
		High Intensity User	0.22	1.4	
8-hr	290	Low Intensity User	13	55	30
		Medium Intensity User	2.3	9.7	
		High Intensity User	0.42	1.4	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low intensity bystanders.

Table 4-86. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Gasket Remover Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.56	29	30
		Medium Intensity User	5.6	2.9	

		High Intensity User	22	0.72	
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For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

The peak exposure value (5120 mg/m³) for the high intensity user identified in Section 2.4.2.4.13 does not exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994) described in Section 3.2.3.1.1 but is greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

4.3.2.3.8 Adhesives

Estimates of MOEs for acute inhalation and dermal exposures for the adhesive consumer use are presented in Table 4-87 and Table 4-88, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.1. Inhalation exposures were modeled for 27 different scenarios and dermal exposure was evaluated for nine scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-87. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesives Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	199	2188	30
		Medium Intensity User	12	130	
		High Intensity User	0.53	4.2	
8-hr	290	Low Intensity User	452	2535	30
		Medium Intensity User	27	150	
		High Intensity User	1.1	4.7	

¹ Data from Putz et al. (1979)

The MOEs are < benchmark MOE for the 1 hr and 8 hr values high end exposure scenarios.
The MOEs are > benchmark MOE for most medium and low exposure scenarios.

Table 4-88. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Adhesives Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.04	372	30
		Medium Intensity User	0.60	27	
		High Intensity User	2.55	6.3	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.9 Auto Leak Sealer

Estimates of MOEs for acute inhalation and dermal exposures for auto leak sealing consumer uses are presented in Table 4-89 and Table 4-90, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposure for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results for users as acute ADRs are described in Section 2.4.2.4.1. Inhalation and dermal exposures were modeled for three different scenarios respectively (combinations of the duration of use and a single value for weight fraction for receptors as adults and two youth age groups)

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint described in Section 3.2.5.3.

Table 4-89. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Auto Leak Sealer Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	120	1031	30
		Medium Intensity User	123	1015	

		High Intensity User	210	1117	
8-hr	290	Low Intensity User	255	1107	30
		Medium Intensity User	259	1077	
		High Intensity User	274	980	

¹ Data from Putz et al. (1979)

For acute inhalation exposures, MOEs are less than the benchmark MOE for consumer users and bystanders at 1-hr and 8-hr exposures for all the exposure scenarios.

Table 4-90. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Auto Leak Sealer Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	1.65	10	30
		Medium Intensity User	3.23	5.0	
		High Intensity User	4.1	3.9	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for all the exposure scenarios.

4.3.2.3.10 Brush Cleaner

Estimates of MOEs for acute inhalation and dermal exposures for the brush cleaner consumer use are presented in Table 4-91 and Table 4-92, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.6. Inhalation exposures were modeled for nine different scenarios and dermal exposure was evaluated for three scenarios (combinations of the duration of use and a weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-91. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Brush Cleaner Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	3956	44077	30
		Medium Intensity User	786	6209	
		High Intensity User	462	1293	
8-hr	290	Low Intensity User	8981	50216	30
		Medium Intensity User	1653	6916	
		High Intensity User	191	919	

¹ Data from Putz et al. (1979)

The MOEs > benchmark MOE for all the PODs.

Table 4-92. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Brush Cleaner Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.040	396	30
		Medium Intensity User	0.48	33	
		High Intensity User	3.39	4.7	

For acute dermal exposures, the MOE is less than the benchmark MOE for consumer users for the high intensity user scenarios.

4.3.2.3.11 Adhesive Remover

Estimates of MOEs for acute inhalation and dermal exposures for the adhesive remover consumer uses are presented in Table 4-93 and Table 4-94, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.2. Inhalation exposures were modeled for 18 different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-93. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesive Remover Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	255	2869	30
		Medium Intensity User	17	134	
		High Intensity User	11	14	
8-hr	290	Low Intensity User	581	3269	30
		Medium Intensity User	36	150	
		High Intensity User	4.3	16	

¹ Data from Putz et al. (1979)

The MOEs are > benchmark MOE.

Table 4-94. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Adhesive Remover Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.75	21	30
		Medium Intensity User	22.41	0.71	
		High Intensity User	179.26	0.090	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for all the exposure scenarios.

4.3.2.3.12 Auto AC Refrigerant

Estimates of MOEs for acute inhalation and dermal exposures for the auto AC refrigerant consumer uses are presented in Table 4-95 and Table 4-96, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal

exposure results are presented for users as acute ADRs in Section 2.4.2.4.4. Inhalation exposures were modeled for 18 different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-95. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Auto AC Refrigerant Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	102	875	30
		Medium Intensity User	8.8	72	
		High Intensity User	3.6	19	
8-hr	290	Low Intensity User	216	939	30
		Medium Intensity User	18	76	
		High Intensity User	4.7	17	

¹ Data from Putz et al. (1979)

The MOEs are < benchmark MOE for the 1-hr and 8-hr values for high end exposure scenarios (user and bystander) and medium exposure scenarios for users.

Table 4-96. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Auto AC Refrigerant Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.020	1482	30
		Medium Intensity User	0.12	164	
		High Intensity User	0.15	21	

For acute dermal exposures, the MOE is less than the benchmark MOE for consumer users for the high intensity user scenario.

4.3.2.3.13 Cold Pipe Insulation Spray

Estimates of MOEs for acute inhalation and dermal exposures for the cold pipe insulation spray consumer use are presented in Table 4-97 and Table 4-98, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used

respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.10. Inhalation exposures were modeled for 18 different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-97. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Cold Pipe Insulation Spray Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	16	167	30
		Medium Intensity User	1.6	17	
		High Intensity User	0.28	2.2	
8-hr	290	Low Intensity User	35	194	30
		Medium Intensity User	3.6	20	
		High Intensity User	0.59	2.4	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low exposure bystanders and low exposure user at 8 hrs.

Table 4-98. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Cold Pipe Insulation Spray Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE
			Acute ADD (mg/kg/day)	MOE	(= Total UF)
Impairment of the CNS	16	Low Intensity User	0.049	325	30
		Medium Intensity User	0.78	20	
		High Intensity User	1.95	8.2	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.14 Sealants

Estimates of MOEs for acute inhalation and dermal exposures for the sealant consumer use are presented in Table 4-99 and Table 4-100, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.14. Inhalation exposures were modeled for 18 different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-99. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Sealants Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	35	304	30
		Medium Intensity User	2.9	24	
		High Intensity User	0.59	3.8	
8-hr	290	Low Intensity User	75	327	30
		Medium Intensity User	6.1	26	
		High Intensity User	1.1	3.6	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low intensity users and bystanders.

Table 4-100. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Sealants Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.081	198	30
		Medium Intensity User	1.0	16	
		High Intensity User	1.30	12	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

4.3.2.3.15 Weld Spatter Protectant

Estimates of MOEs for acute inhalation and dermal exposures for the weld spatter protectant consumer use are presented in Table 4-101 and Table 4-102, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2. For inhalation, low, moderate and high intensity users are characterized by the 10th, 50th, and 95th percentile duration of use and mass of product used respectively and minimum, midpoint, and maximum reported weight fractions where possible respectively. Characterization of low intensity, moderate intensity and high intensity users for dermal followed the same protocol as those described for the inhalation results, but only encompassing the two varied duration of use and weight fraction parameters. Inhalation exposures are presented for users and bystanders for TWAs of 1 hr and 8 hrs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.15. Inhalation exposures were modeled for nine different scenarios and dermal exposure was evaluated for six scenarios (combinations of the duration of use and weight fraction for receptors as adults and two youth age groups).

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and low to medium for the dermal estimate as discussed in Section 2.4.2.6. The study that supports the CNS health concern is described above in Section 4.3.1. Overall, EPA has medium confidence in the acute endpoint, and Section 3.2.5.3 describes the justification for this human health rating.

Table 4-101. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Weld Spatter Protectant Use

HEC Time Period	Acute HEC (mg/m ³)	Exposure Scenario	User MOE	Bystander MOE	Benchmark MOE
Endpoint = CNS Effects ¹					(= Total UF)
1-hr	840	Low Intensity User	4.6	51	30
		Medium Intensity User	0.94	10	
		High Intensity User	0.16	1.3	
8-hr	290	Low Intensity User	11	59	30
		Medium Intensity User	2.1	12	
		High Intensity User	0.35	1.5	

¹ Data from Putz et al. (1979)

The MOEs < benchmark MOE for both 1-hr and 8-hr exposures, except for the low intensity bystanders.

Table 4-102. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Weld Spatter Protectant Use

Health Effect	Acute HED (mg/kg/day)	Exposure Scenario	Adult User		Benchmark MOE (= Total UF)
			Acute ADD (mg/kg/day)	MOE	
Impairment of the CNS	16	Low Intensity User	0.25	65	30
		Medium Intensity User	2.0	8.2	
		High Intensity User	4.9	3.3	

For acute dermal exposures, MOEs are less than the benchmark MOE for consumer users for the medium and high intensity user scenarios.

The peak exposure values (6150, 5050 and 4130 mg/m³) for the high, moderate and low intensity users as well as the 1-hr maximum TWA (5110 mg/m³) for the high intensity user identified in Section 2.4.2.4.15 do not exceed the NIOSH IDLH of 7981 mg/m³ (NIOSH, 1994) but are greater than one half of the IDLH. The NIOSH IDLH value was set to avoid situations that are immediately dangerous to life or health and is a value above which individuals should not be exposed for any length of time.

4.4 Assumptions and Key Sources of Uncertainty

4.4.1 Key Assumptions and Uncertainties in the Environmental Exposure Assessment

Modeled Surface Water Concentrations

Modeled releases using E-FAST 2014 used 2016 TRI and 2016 DMR data to estimate releases. However, both data sources are self-reported and have reporting requirements that limit the number of reporters. Due to these limitations, some sites that manufacture, process, or use methylene chloride may not report to these datasets, are not included in this analysis and therefore actual environmental exposures may be underestimated. Facilities are only required to report to TRI if the facility has 10 or more full-time employees, is included in an applicable NAICS code, and manufactures, processes, or uses the chemical in quantities greater than a certain threshold (25,000 pounds for manufacturers and processors and 10,000 pounds for users). DMR data are submitted by NPDES permit holders to states or directly to the EPA according to the monitoring requirements of the facility's permit. States are only required to load major discharger data into DMR and may or may not load minor discharger data. The definition of major vs. minor discharger is set by each state and could be based on discharge volume or facility size. Due to these limitations, some sites that discharge may not be included in the DMR dataset.

Use of facility data to estimate environmental exposures is constrained by a number of uncertainties including: the heterogeneity of processes and releases among facilities grouped within a given sector; assumptions made regarding sector definitions used to select facilities covered under the scope; and fluctuations in the level of production and associated environmental releases incurred as a result of changes in standard operating procedures.

Uncertainty may also arise from omissions in the reporting data, such as sectors that are not required to report, facilities that fall below the reporting threshold, or facilities for which forms simply are not filed. Additionally, some of the reported information reflects approximations rather than actual measured emissions or release data potentially leading to mischaracterization of actual releases. While these limitations are important, their impact on estimating exposure potential may be less than that associated with the assumptions made regarding environmental releases discussed below. Nevertheless, it is important to note that both TRI and DMR datasets are based on the most comprehensive, best available data at a nationwide scale. TRI data can include monitoring data, mass balances, emission factors, or engineering calculations. DMR is based on representative pollutant monitoring data at facility outfalls and corresponding wastewater discharge.

The days of release applied in modeling have a direct impact on predicting surface water concentrations. The greater the number of release days assumed, the more the per-day release is diluted (assuming the same overall annual loading estimate). For each condition of use, EPA estimated the average daily releases and number of release days per year since actual facility reporting of release days was not available as described in Section 2.2.1. EPA estimated a high and low days of release frequency for all direct releasers and a high days of release frequency for all indirect releasers. Actual release days may vary across and between industries and may not be accurately represented by these assumed default values. There is some uncertainty regarding which release frequency is more likely, but when both high and low days of release frequency are evaluated it is expected to cover the range of possible releases to surface water bodies.

Another key parameter in modeling is the applied stream flow distribution, which provides for the immediate dilution of the release estimate. The flow distributions are applied by selecting a facility-specific NPDES code in E-FAST 2014. When site-specific or surrogate site-specific stream flow data were not available, flow data based on a representative industry sector were used in the assessment. This includes cases where a receiving facility for an indirect release could not be determined. In such cases, it is likely that the stream concentration estimates are higher than they would be if a facility-specific NPDES code was able to be applied, except in certain cases (e.g., NPDES associated with low-flow or intermittent streams or bays). Additionally, the stream flow data currently available in E-FAST 2014 are 15 to 30 years old and may not represent current conditions at a particular location. Nevertheless, the used datasets represent the most comprehensive and accurate nationwide datasets available for modeling evaluation and analysis.

To better assess the effect that these properties may have on instream concentrations of methylene chloride, the volatilization half-life of methylene chloride from a hypothetical reservoir was estimated using the EPISuite model across a range of depths, water velocities, and wind speeds. The evaluated waterbody was informed by dimensions of the EPA Standard Reservoir that has a depth of 2.74 m, width of 82.2 m and flow of 25.01 m³/hr ([Jones et al., 1998](#)). Depth was subsequently varied from 1-10 m, water velocities between 3.09E-05 – 0.5 m/s, and wind speeds between 0.5 – 5.5 m/s. Results showed wide variability in estimated volatilization half-lives ranging from a matter of less than 2 hours (lowest water depth and greatest wind and water velocities) to more than 600 years (greatest water depth and lowest wind and water velocities). Some trends emerged as with increasing depth; volatilization half-lives

increased. For example, a factor of 10 increase in depth led to an approximately 40-50 times decrease in volatilization across the changes in wind and water velocities. In contrast, increasing wind and stream velocities resulted in decreasing half-lives as an 11-times increase in wind speed led to a 6-7 times decrease in half lives across changes in depth and water velocity. While the inability to consider fate or hydrologic transport characteristics is a limitation of the EFAST model, given the wide degree of variation observed in just one such property for methylene chloride, the effect of these properties on estimating instream concentrations is expected to be highly variable and site-specific depending on stream geometries, as well as flow and environmental conditions. Therefore, the estimated concentrations provided for this model are within the bounds of variability and a reasonable estimation of actual instream concentrations. Given this variation, E-FAST surface water concentrations may best represent concentrations found at the point of discharge. The farther from the facility, the more uncertainty, and the lower the confidence EPA has in the concentration.

Finally, EPA did not consider releases' combined impact on concentrations in the same waterbody. This may lead to an underestimation of surface water concentrations in waterbodies with multiple releases coming from one facility or waterbodies with multiple facilities contributing releases.

Measured Surface Water Data and Watershed Analysis

The WQP Tools contains data from USGS-NWIS and STORET databases, and is one of the largest environmental monitoring databases in the U.S.; however, comprehensive information needed for data interpretation is not always reasonably available. In some instances, proprietary information may be withheld, or specific details regarding analytical techniques may be unclear, or not reported at all. As a result, there are uncertainties in the reported data that are difficult to quantify with regard to impacts on exposure estimates.

The quality of the data provided in the USGS-NWIS and STORET datasets varies, and some of the information provided is non-quantitative. While a large number of individual sampling results were obtained from these datasets, the monitoring studies used to collect the data were not necessarily specifically designed to evaluate methylene chloride distribution across the U.S. The available data represent a variety of discrete locations and time periods; therefore, it is uncertain whether the reported data are representative of all possible nationwide conditions. Nevertheless, these limitations do not diminish the overall findings reported in this assessment that exposure data showed no instances where measured methylene chloride levels in the ambient environment exceeded the identified hazard benchmarks for water or organisms. (Section 4.2.2)

It is also important to note that only a few USGS-NWIS and STORET monitoring stations aligned with the watersheds of the methylene chloride-releasing facilities identified under the scope of this assessment, and the co-located monitoring stations had samples with concentrations below the detection limit; therefore, no direct correlation can be made between them. Additionally, the evaluated databases represent the best-known available records of actual methylene chloride concentrations in the environment.

With respect to the geospatial comparison of modeled estimates with ambient data obtained from WQX, one limitation is the accuracy of the latitudes and longitudes. The geographic coordinates

for facilities were obtained from the FRS Interests geodatabase, which are assigned through various methods including photo-interpretation, address matching, and GPS. These are considered “Best Pick” coordinates. While EPA does assign accuracy values for each record based on the method used, the true accuracy of any individual point is unknown. Also, in some cases the receiving facilities for indirect releases could not be determined. In these cases, the location of the active releaser was mapped. As such, the co-location of facilities and monitoring sites may have been missed. As the number of unknown receiving facilities was small and most monitoring sites had samples with concentrations below the detection limit, this would have minimal impact on the watershed analysis.

4.4.2 Key Assumptions and Uncertainties in the Occupational Exposure Assessment

Key uncertainties in the occupational exposure assessment are discussed in the following sections. One overarching uncertainty is that exposures to methylene chloride from outside the workplaces are not included in the occupational assessment, which may lead to an underestimate of occupational exposure. Another overarching uncertainty is that inhalation and dermal exposures were assessed separately, which may also lead to an underestimation of occupational exposure.

4.4.2.1 Occupational Inhalation Exposure Concentration Estimates

Air concentrations. In most scenarios where data were available, EPA did not find enough reasonably available data to determine complete statistical distributions of actual air concentrations for the workers exposed to methylene chloride. Ideally, EPA would like to know 50th and 95th percentiles for each exposed population. In the absence of percentile data for monitoring, the air concentration means and medians (means are preferred over medians) of the data sets served as substitutes for 50th percentiles (central tendencies) of the actual distributions, whereas high ends of ranges served as substitutes for 95th percentiles of the actual distributions. However, these substitutes are uncertain and are weak substitutes for the ideal percentiles. For instance, in the few cases where enough data were found to determine statistical means and 95th percentiles, the associated substitutes (i.e., medians and high ends of ranges) were shown to overestimate exposures, sometimes significantly. While it is clear that most air concentration data represent real exposure levels, EPA cannot determine whether these concentrations are representative of the statistical distributions of actual air concentrations to which workers are exposed. It is unknown whether these uncertainties overestimate or underestimate exposures. Additionally, there are various potential worker activities and/or sites within each OES that may have varying levels of exposures. If the exposure estimate is based on one or very few worker activities or sites within the OES, it could potentially underestimate or overestimate exposures for other workers included in the same OES.

Exposures for occupational non-users can vary substantially. Most data sources do not sufficiently describe the proximity of these employees to the exposure source. As such, exposure levels for the “occupational non-user” category will have high variability depending on the specific work activity performed. It is possible that some employees categorized as “occupational non-user” have exposures similar to those in the “worker” category depending on

their specific work activity pattern. ONUs are likely a heterogeneous population of workers, and some could be exposed more than just occasionally to high concentrations. It is unknown whether these uncertainties overestimate or underestimate exposures. The available data and modeling approaches for assessing inhalation exposures are shown in Table 4-103 for both workers and ONUs.

Table 4-103 Table of Occupational Exposure Assessment Approach for Inhalation

Exposure Scenario	Worker PBZ Monitoring Data (8-hr TWA)	Modeling: Deterministic Worker *	Modeling: Probabilistic Worker NF / ONU FF	ONUs Monitoring data
1 Manufacturing	X			
2 Import/ Repackaging/ Distribution	X	X		
3 Processing as a reactant	X	X		Area monitoring ^
4 Processing into a formulation	X	X		
5 Batch vapor degreasing			X	
6 ConveyORIZED vapor degreasing			X	
7 Cold Cleaning	X		X	
8 Commercial Aerosol Products	X		X	
9 Adhesives and Sealants – spray and non-spray	X			Area monitoring ^
10 Paints and coatings - paint application – spray including: Paints and coatings - paint removers 2014 EPA Risk Assessment	X			
11 Adhesive and Caulk Removers	X			
12 Fabric Finishing	X			ONU specific PBZ monitoring
13 Spot Cleaning	X		‡	
14 Cellulose Triacetate Film Production	X			
15 Flexible Polyurethane Foam Manufacturing	X			
16 Laboratory chemicals	X			
17 Plastic and rubber products	X*			ONU specific PBZ monitoring
18 Lithographic Printing	X			
19 Miscellaneous Non-Aerosol Uses	X			
20 Waste Handling	X	X		

^ While area monitoring data were identified, there is some uncertainty about the representativeness of these data for ONU exposures for these specific exposure scenarios because of the intended sample population and the selection of the specific monitoring location.

* The deterministic modeling approach only addresses unloading of methylene chloride from transport containers, which is not presented because it is only appropriate for filling data gaps as it provides estimates for only one potential activity. This approach does not estimate exposures for ONUs.

‡ EPA has developed a model to evaluate potential worker and ONU exposures during spot cleaning for various solvents; however, the specific methylene chloride use rate during spot cleaning was not reasonably available. This is a critical data gap and other solvent use rates may not be applicable.

Additionally, some data sources may be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. These sources may cause exposures to be underestimated or overestimated.

Due to data limitations in most OESs, EPA combined inhalation data from two or more data sets when metadata were not available to distinguish between OES subcategories. These combinations introduce uncertainties as to whether data from disparate worker populations had been combined into one OES or OES subcategory. This same uncertainty applies to mixing data collected pre-PEL change with data collected post-PEL change.

Where data were not reasonably available, the modeling approaches used to estimate air concentrations also have uncertainties. Parameter values used in models did not all have distributions known to represent the modeled scenario. It is also uncertain whether the model equations generate results that represent actual workplace air concentrations. It is unknown whether these uncertainties overestimate or underestimate exposures. Additional model-specific uncertainties are included below.

Averaging Times. EPA cannot determine how accurately the assumptions of exposure frequencies (days/yr exposed) and exposed working years may represent actual exposure frequencies and exposed working years. For example, tenure is used to represent exposed working years, but many workers may not be exposed during their entire tenure. It is unknown whether these uncertainties overestimate or underestimate exposures, although the high-end values may result in overestimates when used in combination with high-end values of other parameters.

4.4.2.2 OSHA Data Analysis

The data for the OSHA analysis originated from a docket comment from Dr. Finkel, who obtained dataset via a Freedom of Information Act (FOIA) request from OSHA ([Finkel, 2017](#)). The Finkel data only provide SIC codes, which are only sufficient to relate exposures to broad industry sectors. Within each industry, there may be worker activities that span several OES. For example, an automotive repair shop may use MC-containing paint strippers, paints and coatings, adhesives, and non-aerosol cleaning solvents. Without worker activity descriptions for each measured exposure, it was not possible to distinguish between workers and ONUs. For the purpose of this analysis, EPA crosswalked reported SIC codes to 2017 NAICS codes and grouped NAICS codes that may be relevant to each condition of use to assign data to OESs. Sample times also varied; EPA assumed that any measurement longer than 15 minutes was done to assess compliance with the 8-hr TWA PEL, as opposed to the 15-minute STEL, and averaged all applicable data points over 8 hours. Therefore, there may be shorter-term data that do not

fully represent the exposures over the full work shift, which would result in underestimated exposures when averaged over an 8-hr time period.

Note that the Finkel (2017) data were not verified for quality by OSHA and did not fully describe the metadata. EPA separately consulted with OSHA and discussed data needs for the risk evaluations. OSHA subsequently provided a subset of data that also included worker activity descriptions and were verified for quality and were subsequently used in the risk evaluation (OSHA, 2019).

For the analysis, EPA defined the pre-rule period as prior to April 10, 1997 and the post-rule period as after April 10, 2000. Some companies may have begun implementing controls to reduce exposure prior to the official rule date, which would result in smaller pre- to post-PEL reductions. However, it is not possible to tell when each company undertook measures to comply to the PEL.

EPA's judgments about which industries (represented by NAICS codes) are associated with the uses assessed in this report are based on EPA's understanding of how methylene chloride is used in each industry. Designations of which industries have potential exposures is nevertheless subjective, and some industries with few exposures might erroneously be included, or some industries/occupations with exposures might erroneously be excluded. This would result in inaccuracy but would be unlikely to systematically either overestimate or underestimate the exposures.

OSHA data are typically obtained from inspections, which may be the result of worker complaints and may provide exposure results that are generally more conservative than the industry average. Additionally, the comparison likely does not compare pre- and post-PEL worker exposures at the same sites involved in the processes, so a direct assessment of the PEL impact is not possible.

4.4.2.3 Near-Field/Far-Field Model Framework

The near-field/far-field approach is used as a framework to model inhalation exposure for many conditions of use. The following describe uncertainties and simplifying assumptions generally associated with this modeling approach:

- There is some degree of uncertainty associated with each model input parameter. In general, the model inputs were determined based on review of available literature. Where the distribution of the input parameter is known, a distribution is assigned to capture uncertainty in the Monte Carlo analysis. Where the distribution is unknown, a uniform distribution is often used. The use of a uniform distribution will capture the low-end and high-end values but may not accurately reflect actual distribution of the input parameters.
- The model assumes the near-field and far-field are well mixed, such that each zone can be approximated by a single, average concentration.
- All emissions from the facility are assumed to enter the near-field. This assumption will overestimate exposures and risks in facilities where some emissions do not enter the airspaces relevant to worker exposure modeling.

- The exposure models estimate airborne concentrations. Exposures are calculated by assuming workers spend the entire activity duration in their respective exposure zones (i.e., the worker in the near-field and the occupational non-user in the far-field). Since vapor degreasing and cold cleaning involve automated processes, a worker may actually walk away from the near-field during part of the process and return when it is time to unload the degreaser. As such, assuming the worker is exposed at the near-field concentration for the entire activity duration may overestimate exposure. The assumption that ONUs are present only in the far-field could result in underestimates for ONUs present in the near-field.
- For certain applications (e.g., vapor degreasing), methylene chloride vapor is assumed to emit continuously while the equipment operates (i.e., constant vapor generation rate). Actual vapor generation rate may vary with time. However, small time variability in vapor generation is unlikely to have a large impact in the exposure estimates as exposures are calculated as a time-weighted average.
- The exposure models represent model workplace settings for each methylene chloride condition of use. The models have not been regressed or fitted with monitoring data.
- Beyond the exceptions noted, it is unknown whether these uncertainties overestimate or underestimate exposures.

Each subsequent section below discusses uncertainties associated with the individual model.

4.4.2.3.1 Vapor Degreasing Models

The OTVD and conveyORIZED vapor degreasing assessments use a near-field/far-field approach to model worker exposure. In addition to the uncertainties described above, the vapor degreasing models have the following uncertainties:

- To estimate vapor generation rate for each equipment type, EPA used a distribution of the emission rates reported in the 2014 NEI for each degreasing equipment type. NEI only contains information on major sources not area sources. Therefore, the emission rate distribution used in modeling may not be representative of degreasing equipment emission rates at area sources.
- The emission rate for conveyORIZED vapor degreasing is based on equipment at a single site and the emission rates for web degreasing are based on equipment from two sites. It is uncertain how representative these data are of a “typical” site.
- EPA assumes workers and occupational non-users remove themselves from the contaminated near- and far-field zones at the conclusion of the task, such that they are no longer exposed to any residual methylene chloride in air, which may underestimate exposures.
- Beyond the exceptions noted, it is unknown whether these uncertainties overestimate or underestimate exposures.

4.4.2.3.2 Brake Servicing Model

The aerosol degreasing assessment also uses a near-field/far-field approach to model worker exposure. Specific uncertainties associated with the aerosol degreasing scenario are presented below:

- The model references a CARB study ([CARB, 2000](#)) on brake servicing to estimate use rate and application frequency of the degreasing product. The brake servicing scenario may not be representative of the use rates for other aerosol degreasing applications involving methylene chloride.
- Because market penetration data were not available for methylene chloride-containing products, EPA assumed the market penetration for perchloroethylene as an upper bound because perchloroethylene comprises the majority of the chlorinated solvent-based degreaser volume ([CARB, 2000](#)).
- EPA found 10 different aerosol degreasing formulations containing methylene chloride. For each Monte Carlo iteration, the model determines the methylene chloride concentration in product by selecting one of 10 possible formulations, assuming the distribution for each formulation is equal. It is uncertain if this distribution is representative of all sites in the U.S.
- Aerosol formulations were taken from available safety data sheets, and most were provided as ranges. For each Monte Carlo iteration, the model selects a methylene chloride concentration within the range of concentrations using a uniform distribution. In reality, the methylene chloride concentration in the formulation may be more consistent than the range provided.
- It is unknown whether these uncertainties overestimate or underestimate exposures.

4.4.2.4 Occupational Dermal Exposure Dose Estimates

The *Dermal Exposure to Volatile Liquids Model* used for modeling occupational dermal exposures accounts for the effect of evaporation on dermal absorption for volatile chemicals and the potential exposure reduction due to glove use. The model does not account for the transient exposure and exposure duration effect, which likely overestimates exposures. The model assumes one exposure event per day, which likely underestimates exposure as workers often come into repeat contact with the chemical throughout their workday. Surface areas of skin exposure are based on skin surface area of hands from EPA's Exposure Factors Handbook, but actual surface areas with liquid contact are unknown and uncertain for all OESs. For many OESs, the high end assumption of contact over the full area of two hands likely overestimates exposures. Weight fractions are usually reported to CDR and shown in other literature sources as ranges, and EPA assessed only upper ends of ranges. The glove protection factors, based on the ECETOC TRA model as described in Section 2.4.1.1, are "what-if" assumptions and are uncertain. EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces of the OESs. Except where specified above, it is unknown whether most of these uncertainties overestimate or underestimate exposures. The representativeness of the modeling results toward the true distribution of dermal doses for the OESs is uncertain.

4.4.3 Key Assumptions and Uncertainties in the Consumer Exposure Assessment

Systematic review was conducted to identify chemical- and product-specific monitoring and use data for assessing consumer exposures. As no product-specific monitoring data were identified, exposure scenarios were assessed using a modeling approach that requires the input of various

chemical parameters and exposure factors. When possible, default model input parameters were modified based on chemical and product specific inputs available in literature and product databases. Uncertainties and assumptions related to these inputs are discussed below.

Background Exposure

One overarching uncertainty is that the risk estimations for consumers may be underestimations, because background exposures are not incorporated to the risk estimations for each COU. While there are documented background exposures of methylene chloride in residential or consumer environments (Section 2.4.2.5), those concentrations were not attributable to a specific condition of use and therefore not included in our evaluation. Ambient air samples worldwide have shown measured levels of methylene chloride, with background levels usually around 50 parts per trillion ([ATSDR, 2000](#)). National Oceanic and Atmospheric Administration (NOAA) monitoring data between 1994 and 2016 show mid-latitude northern hemisphere atmospheric concentrations to decrease slightly from 1994 to the early 2000s, and then increase thereafter to present day, with monthly mean concentrations ranging from approximately 30-80 parts per trillion ([Hossaini et al., 2015](#)). Similarly, air concentrations in the continental U.S. between 2003 and 2014 showed either no trend or increasing levels of methylene chloride ([U.S. EPA, 2016](#)). The 2011 National Air Toxics Assessment (NATA) modeled concentrations for various air toxics nationwide at a census tract level. This screening level tool modeled a maximum total methylene chloride concentration of 5,000 parts per trillion ($18 \mu\text{g}/\text{m}^3$). Greater than 94% of all modeled tracts were less than 100 parts per trillion. While available indoor air measurements for methylene chloride are less prevalent, it may be present in this environment due to its variety of uses including consumer uses.

Inhalation and Dermal Aggregate Exposure

Another overarching uncertainty is that inhalation and dermal exposures were assessed separately, which may also lead to an underestimation of consumer exposure. There is low confidence in the result of aggregating the dermal and inhalation risks for this chemical if EPA uses an additive approach, due to the uncertainty in the data. EPA does not have data that could be reliably modeled into the aggregate, which would be a more accurate approach than adding, such as through a PBPK model. Using an additive approach to aggregate risk in this case would result in an overestimate of risk. Given all the limitations that exist with the data, EPA's approach is the best available approach.

Dermal Approach

For the presented dermal exposure evaluation, EPA used product specific information for individual COUs, likely use patterns, and professional judgement to consider whether a product was expected to have dermal contact with impeded or unimpeded evaporation. As explained in Section 2.4.2.3.1.2, scenarios expecting unimpeded evaporation were considered using the P_DER2a (Fraction Absorbed) submodel and scenarios expecting impeded evaporation used the P_DER2b (Permeability) submodel. Each submodel within CEM has given limitations and uncertainties associated with the use of that model which are described below and comparable results for each model are available in CONSUMER EXPOSURES Appendix G.

A key assumption of the permeability submodel is that the model assumes a constant supply of chemical directly in contact with the dermal surface. However, it is unlikely that dermal contact

would remain unimpeded during the entire use duration, particularly for central-tendency and high-end use durations (See “Duration of Use” section below). It is more likely that such contact would be intermittent and may lead to overestimates in overall exposure. Alternatively, the fraction absorbed submodel assumes the amount retained on skin was equal to the amount absorbed into the stratum corneum (see below in “Amount Retained on Skin”). It is likely this represents an overestimate as a portion of chemical applied to the top of the stratum corneum is subject to evaporation. However, this submodel also assumes that the given mass in the amount retained is only applied once. For uses with extended product use times and chemical properties, there is the possibility for the mass in the amount retained to be “filled” multiple times leading to possible underestimates in exposure.

There is related uncertainty surrounding the application of exposure durations for such scenarios. The exposure durations modeled are based on reported durations of product use and may not reflect reasonable durations of such dermal contact with impeded or unimpeded evaporation. In many cases, the exposure duration modeled could exceed a reasonable duration of such dermal contact. Therefore, dermal exposure results based on the higher-end durations (i.e., those associated with the moderate- and high-intensity user scenarios) may overestimate or underestimate dermal exposure.

For both submodels, a potential source of overestimation is the application of a single formulation density to scenarios covering a range of specific methylene chloride-containing products with a range of formulation densities. For such scenarios, a single (highest) density was chosen to convert the mass used input obtained from the Westat (1987) survey from ounces of product to grams of product. For some scenarios, this may have driven up the mass used, though the degree of this impact is dependent on the broadness of the density range for that condition of use.

Product & Market Profile

The products and articles assessed in this risk evaluation are largely based on EPA’s 2016-2017 Use and Market Profile for Methylene Chloride, as well as EPA’s Use Report and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: Methylene Chloride, which provide information on commercial and consumer products available in the U.S. marketplace at that time. While it is possible that some products may have changed since 2017, EPA believes that the timeframe is recent enough to still represent the current market. Information on products from the Use and Market Profile was augmented with other sources such as the NIH Household Product Survey and EPA’s CPDat, as well as available product labels and SDSs. However, it is still possible that the entire universe of products may not have been identified, due to market changes or research limitations.

U.S. EPA (1987) Consumer Use Survey

A number of product labels and/or technical fact sheets were identified for use in assessing consumer exposure. The identified information often did not contain product-specific use data, and/or represented only a small fraction of the product brands containing the chemical of interest. A comprehensive survey of consumer use patterns in the U.S., the *Household Solvent Product: A National Usage Survey* (U.S. EPA, 1987), was used to parameterize critical consumer modeling inputs, based on applicable product and use categories. This large survey of

over 4,920 completed questionnaires, obtained through a randomized sampling technique, is highly relevant because the primary purpose was to provide statistics on the use of solvent-containing consumer products for the calculation of exposure estimates. The survey focused on 32 different common household product categories, generally associated with cleaning, painting, lubricating, and automotive care. There is some uncertainty due to the age of the use pattern data, as specific products in the household product categories have likely changed over time. For instance, a consumer movement towards more do-it-yourself projects with products containing the chemical may lead to an underestimate of consumer use patterns described within the survey in some instances. Nevertheless EPA assumes that the use pattern data presented in U.S. EPA (1987) reflects reasonable estimates for current use patterns of similar product type. These estimates were deemed to be reasonable due to the range of use patterns evaluated (e.g., ranging from 10th to 95th percentile) and that this dataset represents the most recent, relevant and nationally-representative data available for use pattern data in most cases. U.S. EPA (1987) aimed to answer the following key questions for each product category, some of which were used as key model inputs in this consumer assessment:

- room of product use (key input: environment of use),
- how much time was spent using the product (key input: duration of product use per event),
- how much of the product was used (key input: mass of product used per event),
- how often the products were used,
- when the product was last used,
- product formulation,
- brand names used, and
- degree of ventilation or other protective measures undertaken during product use.

The strengths and weakness of the Westat survey are discussed in more detail below with an emphasis on the key modeling inputs.

Product Use Category

A crosswalk was completed to assign consumer products in the current risk evaluation to one of the product or article scenarios in the CEM model, and then to an appropriate survey category. Although detailed product descriptions were not provided in U.S. EPA (1987), a list of product brands and formulation type in each category was useful in pairing the survey product categories to the scenarios being assessed. In most cases, the product categories in U.S. EPA (1987) aligned reasonably well with the products being assessed. For product scenarios without an obvious survey scenario match, professional judgment was used to make an assignment. For a limited number of scenarios, technical fact sheets or labels with information on product use amounts were available, and this information was used in the assessment as needed.

Another limitation of the U.S. EPA (1987) data is that while the overall respondent size of the survey was large, the number of users in each product category was varied, with some product categories having a much smaller pool of respondents than others. Product categories such as spot removers, cleaning fluids, glues and adhesives, lubricants, paints, paint strippers, fabric water repellents, wood stains, tire cleaners, engine degreasers, carburetor cleaners, and specialized electronic cleaners had sample sizes ranging from roughly 500 to 2,000 users;

whereas, categories such as shoe polish, adhesive removers, rust removers, primers, outdoor water repellents, gasket removers and brake cleaners had sample sizes of less than 500 users.

The survey was conducted for adults ages 18 and older. Most consumer products are targeted to this age category, and thus the respondent answers reflect the most representative age group. However, youth may also be direct users of some consumer products. It is unknown how the usage patterns compare between adult and youth users, but it is assumed that the product use patterns for adults will be very similar to, or more conservative (i.e., longer use duration, higher frequency of use) than use patterns for youth.

Room of Use

The CEM model requires specification of a room of use, which results in the following default model assumptions (relevant for inhalation exposure only): ventilation rates, room volume, and the amount of time per day that a person resides in the room of use. The U.S. EPA (1987) survey provided the location of last product use for the following room categories: basement, living room, other inside room, garage, and outside. The room with the highest percentage was selected as the room to model in CEM. For some specific product scenarios, however, professional judgement was used to assign the room of use; these selections are documented in the input section. For many scenarios in which “other inside room” was the highest percentage, the utility room was selected as the default room of use. The utility room is a smaller room, and therefore may provide a more conservative assumption for peak concentrations. In cases where outside was identified as the “room of use,” but it was deemed reasonable to assume the product could be used inside (such as for auto care products), the garage was typically selected as the room of use.

Amount of Product Used and Duration of Product Use

The U.S. EPA (1987) survey reported ounces per use, derived from the ounces of product used per year (based on can size and number of cans used), divided by the number of reported uses per year. The duration of use (in minutes) reported in U.S. EPA (1987) was a direct survey question. An advantage to these parameters is that the results are reported in percentile rankings and were used to develop profiles of high intensity, moderate intensity, and low intensity users of the products (95th, 50th, and 10th percentile values, respectively). In cases where a product was not crosswalked to a CEM scenario, the amount of product used was tailored to those specific products instead of depending on U.S. EPA (1987) data.

Ventilation and Protection

For most scenarios, the CEM model was run using median air exchange rates from EPA’s Exposure Factors Handbook (2011a), and interzone ventilation rates derived from the air exchange rates and the default median building volume from EPA’s Exposure Factors Handbook (2011a). These inputs do not incorporate any measures that would serve to increase air exchange. The U.S. EPA (1987) survey questions indicated that most respondents did not have an exhaust fan on when using these products, most respondents kept the door to the room open when using these products, and most people reported reading the directions on the label. The modeling conducted by EPA did not account for specific product instructions or warning labels. For example, some product labels might indicate that protective equipment (chemical resistant gloves or respirator) should be worn, which would lower estimated exposures.

Other Parameters and Data Sources

Activity Patterns: EPA assumed that a consumer product would be used only once per day. This is a realistic assumption for most scenarios, but a high-intensity user could use the same product multiple times in one day. Additionally, CEM allows for selection of activity patterns based on a “stay-at-home” resident or a part-time or full-time “out-of-the home” resident. The activity patterns were developed based on CHAD data of activity patterns, which is an EPA database that includes more than 54,000 individual study days of detailed human behavior ([Isaacs, 2014](#)). It was assumed that the user followed a “stay-at-home” activity pattern that would place them in the home and room of use for more time than a part-time or full-time “out-of-the home” resident. Applying an “out-of-the home” resident activity pattern would reduce estimated exposures. EPA also assumed that bystanders did not enter the room of use during the product use period as entering the room of use during this period would be expected to be similar to the evaluated user scenario. Therefore, reported bystander exposures may be underestimated, but reported user exposures would be expected to be inclusive of this situation.

Product Density: If available, product-specific densities were obtained from SDS information, and used to convert the ounces of the product used from U.S. EPA ([1987](#)), to grams of product used. If product-specific densities were not available, default product densities from the CEM User Guide ([EPA, 2017](#)) were used.

Amount Retained on Skin: For estimation of dermal exposure using the Fraction Absorbed Method within CEM as outlined in Section 2.4.2.3.1.2 (P_DER2a), the amount retained on skin parameter (AR) was assumed to equal the amount absorbed in the top of the stratum corneum (SC). In practice, a portion of the amount of chemical applied on top of the SC at the beginning of exposure (AR term) will evaporate and another portion will enter into the top layer of the SC. That portion entering the SC is then subject to potential further-evaporation from the SC or further penetration into the dermis layer.

4.4.4 Key Assumptions and Uncertainties in Environmental Hazards

While EPA determined that there was sufficient environmental hazard data to characterize environmental hazards of methylene chloride, uncertainties exist.

EPA used sub-chronic data, measuring a developmental effect in embryo and larvae, to calculate the amphibian chronic COC, which introduces some uncertainty about whether we are overestimating or underestimating risk from chronic exposure. Assessment factors (AFs) were used to calculate the acute and chronic COCs for methylene chloride. AFs account for the uncertainty in the differences in inter- and intra-species variability, as well as laboratory-to-field variability and are routinely used within TSCA for assessing the hazard of new industrial chemicals (with very limited environmental test data). However, there is no way of knowing exactly how much uncertainty to account for in the AFs. Therefore, there is uncertainty associated with the use of the specific AFs used in the hazard assessment. For example, a standard UF has not been established for amphibians by the EPA under TSCA, because there are few amphibian studies for industrial chemicals. It is unclear whether using an assessment factor of 10 to calculate the acute COC value for amphibians using the sub-chronic embryo-larvae test

data is sufficiently protective or is overly protective of amphibian exposures to methylene chloride.

EPA has uncertainty in its quantitative analysis of sediment-dwelling species, because several assumptions were made. While no ecotoxicity studies were available for sediment-dwelling species (e.g., *Lumbriculus variegatus*, *Hyalella azteca*, *Chironomus riparius*), aquatic invertebrates were used as a surrogate species. EPA is uncertain whether methylene chloride is more or less toxic to daphnia than sediment-dwelling species. However, because methylene chloride is not expected to sorb to sediment and will instead remain in pore water, daphnia which feed through the entire water column were deemed to be an acceptable surrogate species for sediment invertebrates. Additionally, methylene chloride is expected to be in sediment and pore water with concentrations similar to or less than the overlying water due to its water solubility (13 g/L), low partitioning to organic matter ($\log K_{OC} = 1.4$), and biodegradability in anaerobic environments. Thus, methylene chloride concentrations in sediment and pore water are expected to be similar to or less than the concentrations in the overlying water, and concentrations of methylene chloride in the deeper part of sediment, where anaerobic conditions prevail, are expected to be lower.

There are additional factors that affect the potential for adverse effects in aquatic organisms. Life-history factors and the habitat of aquatic organisms influences the likelihood of exposure above the hazard benchmark in an aquatic environment.

4.4.5 Key Assumptions and Uncertainties in the Human Health Hazards

Effects from Acute and Short-term Exposure - CNS Depression

There is some uncertainty in choosing Putz et al. (1979) for the POD. At higher concentrations, some human experimental studies did not identify significant CNS-related effects (Kozena et al., 1990; Gamberale et al., 1975; Divincenzo et al., 1972). Yet, all three studies received low data quality ratings due to non-standard methods of exposure generation (e.g., (Kozena et al., 1990; Gamberale et al., 1975)) or lack of information on results (Divincenzo et al., 1972). Furthermore, Putz et al. (1979) uses changes in a complex task, which would not be identified in studies of simple reaction time (e.g., (Gamberale et al., 1975)).

EPA considers that there is some uncertainty using an effect of limited severity (7% decreased visual performance). However, to account for the limited severity, EPA applied a smaller UF for LOAEL to NOAEL (3 vs.10) when setting the benchmark MOE. Furthermore, it is important to consider less severe effects rather than quantifying only more severe effects, in part, due to the possibility of serious harm and death as concentrations and exposure durations increase.

There is also uncertainty in using the Ten Berge et al. (1986) approach to convert the POD value from 1.5 hours to PODs appropriate for the 15-minute, 1-hour and 8-hour exposure durations. Weaknesses in the ten Berge approach include reliance on an “n” estimated using lethality data, which may not apply to CNS effects. In addition, using the ten Berge equation may result in inaccuracies when extrapolating to exposure durations that are very different from the exposure duration used in Putz et al. (1979), especially longer durations. Also, the ten Berge equation does

not account for full toxicokinetic variability among humans. The AEGL program used a PBPK model described by [Bos et al. \(2006\)](#) instead. The model accounts for the distribution of GSTT1 isoenzyme among humans, predicted methylene chloride concentrations in the brain and COHb levels in blood. However, [Bos et al. \(2006\)](#) acknowledge that there are no adequate data on MC in rat or human brains and assumes that at longer exposures, the more relevant endpoint is COHb only, which doesn't account for the direct effect of methylene chloride. Also, the model overpredicts MC and COHb concentration by up to 50%; thus, the lower POD predicted by the model for longer exposure durations may be partially due to this overprediction.

For shorter durations, the results using [Ten Berge et al. \(1986\)](#) are similar to the results of the PBPK model.²³ Due to the uncertainties related to the [Bos et al. \(2006\)](#) PBPK model, EPA believes that the ten Berge equation is appropriate to use in the current risk evaluation.

EPA recognizes that at higher methylene chloride exposure concentrations and durations, COHb concentrations in blood may stay in the body for a longer time and lead to effects such as decreased time to angina in individuals with cardiac disease. However, the concentrations used for the PODs are lower and thus, COHb retention is shorter.

OSHA has established a 15-minute STEL ([OSHA, 1997a](#)) of 433 mg/m³, which differs from using the current POD and benchmark MOE. However, OSHA acknowledges that it was chosen as a feasible value for the workplace and acknowledge uncertainty as to whether the value would adequately protect physically active workers ([OSHA, 1997a](#)). Therefore, the value is not appropriate because TSCA does not allow consideration of non-risk factors when evaluating risks.

Immune System Effects

Although there is some evidence for immunosuppression as identified by Aranyi et al. ([1986](#)), EPA cannot easily conclude from animal studies that methylene chloride results in immunotoxicity-related effects due to a limited database and lack of association among other studies. However, Aranyi et al. ([1986](#)) identified an effect at a concentration lower than the chosen POD, and if this effect is real, there is some uncertainty in the risk evaluation conclusions and risks could be underestimated.

Nervous System Effects

EPA has not advanced the ASD hazard to dose-response due to numerous uncertainties identified in Section 3.2.4.1.4 (Weight of the Scientific Evidence, Nervous System Effects) related to confounding from co-exposures and lack of temporal specificity in the studies evaluating this effect. Furthermore, the results were most often not statistically significant. However, the human studies, while not establishing causality with developmental exposures consistently, identified odds ratios greater than one indicating an association between methylene chloride and ASD.

²³ PBPK vs. Default: 290 vs. 310 ppm (10 min); 230 vs. 210 ppm (30 min); 200 vs. 170 ppm (1 hr)

There is also uncertainty regarding nervous system effects from chronic exposure. Available studies of developmental neurotoxicity in humans and animals did not allow for quantitative risk evaluation.

Liver Effects

In the evaluation of liver effects from chronic methylene chloride exposure, EPA considered the 1st percentile in the PBPK model to account for sensitive individuals in the population as the most appropriate percentile for this modeling. However, alternate percentile values are similar to the 1st percentile of 17.2 mg/m³; the 5th percentile is 21.3 mg/m³ and the mean is 48.5 mg/m³ (a difference of less than 3-fold).

Reproductive/Developmental Effects

EPA did not carry reproductive/developmental effects forward for dose-response modeling because data are inconclusive. However, there is uncertainty about such effects given endpoints identified within epidemiological studies and effects observed in animal studies.

Cancer

There is uncertainty regarding modeling liver and lung tumors for humans. First, the majority of epidemiology studies did not identify an association between methylene chloride and liver or lung cancer, although there are issues with unequal comparison groups that include workers vs. the general population or differences in smoking status that may lead to attenuated effects, as noted in Section 3.2.4.2. Second, increases in genotoxicity are correlated with increases in GST/GSTT1 activity in many test systems and mice lung and liver tissues have higher levels of GSTT1 compared with these tissues in humans. EPA did, however, address this uncertainty by using a PBPK model to account for differences in GST activity between mice and humans and among humans.

There is also uncertainty regarding the association between methylene chloride and risk of developing tumors in other tissues. Human GSTT1 activity is higher in other tissues compared with the liver. For example, the GSTT1 activity in erythrocytes for human high conjugators is the same as male mice, and workers exposed to methylene chloride had increased frequencies of micronuclei and DNA damage in peripheral blood lymphocytes. Furthermore, hematopoietic tumors have been observed in some epidemiology studies and are more consistently associated with methylene chloride than other tumor types. Thus, hematopoietic tumors may be of concern for humans.

Animal studies consistently identify methylene chloride exposure as associated with mammary tumors, and the IURs for mammary tumors are of greater magnitude than the combined liver and lung tumor IURs. Furthermore, breast cancer has been identified in one human epidemiology study (see Section 3.2.3.2.1). However, very few tumors from the animal studies are malignant, the dose metric for breast cancer is not certain and data on mutagenicity in these tissues is lacking. In addition, a small fraction 0.1% of fibroadenomas lead to carcinomas ([Russo, 2015](#)). Thus, EPA chose not to use the animal mammary tumor data in this risk evaluation.

Another uncertainty is the lack of positive genotoxicity results in livers of mice exposed via inhalation of 800 ppm methylene chloride for four weeks ([Suzuki et al., 2014](#)). Therefore, it is uncertain whether lower methylene chloride concentrations would result in cancer. However, MOAs that suggest possible non-linear relationships have not been adequately developed for methylene chloride. Andersen et al. ([2017](#)) suggested a MOA related to hypoxia and changes in the circadian clock. Although this is an interesting hypothesis and may have merit, 1) the study measured only gene expression changes, 2) EPA found no well-established MOA and 2) related methylene chloride mechanistic data supporting the MOA were lacking. Finally, Andersen et al. ([2017](#)) identified their conclusions regarding the possible MOA as tentative. EPA found no other data supporting alternate MOAs.

Route to Route Extrapolation

There is uncertainty in extrapolating the hazard endpoints across routes. For example, although EPA does expect that some neurotoxicity may result from dermal exposure, there may be additional absorption through nasal passages to the brain. Furthermore, there is uncertainty regarding the likelihood that dermal exposure will result in lung cancer, but because humans may experience different cancers than rodents, EPA has assumed that the slope factor of the combined tumor types can be considered generally representative of the potential for cancers of other types.

4.4.6 Key Assumptions and Uncertainties in the Environmental Risk Estimation

There was uncertainty related to environmental risk for methylene chloride. EPA used both E-FAST and monitored data to characterize acute and chronic exposures of methylene chloride to aquatic organisms.

E-FAST: In some ways the E-FAST underestimates exposure, because data used in E-FAST include TRI and DMR data. TRI does not include facilities with fewer than 10 full time employees, nor does it cover certain sectors, which may lead to underestimates in total methylene chloride releases to the environment. In other ways the E-FAST overestimate exposure, because methylene chloride is a volatile chemical, and E-FAST doesn't take volatilization into consideration; and, for static water bodies, E-FAST doesn't take dilution into consideration.

E-FAST 2014 does not take volatilization or other fate and hydrologic transport characteristics into consideration when estimating surface water concentrations. Additionally, for static water bodies, E-FAST 2014 may not take dilution into consideration. As such, for a volatile chemical such as methylene chloride, this may lead to overestimates in actual exposure concentrations.

To better assess the effect that these properties may have on instream concentrations of methylene chloride, the volatilization half-life of methylene chloride from a hypothetical reservoir was estimated using the EPISuite model across a range of depths, water velocities, and wind speeds. The evaluated waterbody was informed by dimensions of the EPA Standard Reservoir that has a depth of 2.74 m, width of 82.2 m and flow of 25.01 m³/hr ([Jones et al.](#),

1998). Depth was subsequently varied from 1-10 m, water velocities between $3.09E-05$ – 0.5 m/s, and wind speeds between 0.5 – 5.5 m/s. Results showed wide variability in estimated volatilization half-lives ranging from a matter of less than 2 hours (lowest water depth and greatest wind and water velocities) to more than 600 years (greatest water depth and lowest wind and water velocities). Some trends emerged as with increasing depth; volatilization half-lives increased. For example, a factor of 10 increase in depth led to an approximately 40-50 times decrease in volatilization across the changes in wind and water velocities. In contrast, increasing wind and stream velocities resulted in decreasing half-lives as an 11-times increase in wind speed led to a 6-7 times decrease in half lives across changes in depth and water velocity.

While the inability to consider fate or hydrologic transport characteristics is a limitation of the EFAST model, given the wide degree of variation observed in just one such property for methylene chloride, the effect of these properties on estimating instream concentrations is expected to be highly variable and site-specific depending on stream geometries, as well as flow and environmental conditions. Therefore, the estimated concentrations provided for this model are within the bounds of variability and a reasonable estimation of actual instream concentrations. Given this variation, E-FAST surface water concentrations may best represent concentrations found at the point of discharge. The farther from the facility, the more uncertainty, and the lower the confidence EPA has in the concentration.

Additionally, there is some uncertainty around modeled releases that have surface water concentrations greater than the highest COC for fish (7,581 ppb). As stated in Section 4.2.2, both of the releases originated from the same indirect discharging facility, VEOLIA ES TECHNICAL SOLUTIONS LLC (MIDDLESEX, NJ), which is categorized in the recycling and disposal OES. The releases were transferred to separate receiving facilities for treatment: Clean Harbors of Baltimore with a modeled concentration of 17,000 ppb. These concentrations are 5 to 11 times higher than the next highest surface water concentration modeled. A NPDES or surrogate NPDES code of the receiving facilities could not be identified in E-FAST 2014; therefore, the model runs were made using the POTW industry sector as a surrogate, as described in Section 4.2.2. Site-specific flows would improve the accuracy of the estimates, but due to the large release amounts it is likely that even site-specific flows would result in concentrations that would exceed one or more COC. Better understanding of how the methylene chloride transferred to these facilities was handled or treated is likely to lead to better estimated releases and exposure concentrations from these facilities. The remaining facilities with 7Q10 SWCs that exceeded a COC also generally had high annual release amounts. Some facilities with lower release amounts, such as LONG BEACH (C) WPCP LONG BEACH discharged to a still waterbody which utilized a dilution factor of 1.

Monitored data: The available monitored data was limited temporally and geographically. Aquatic environmental conditions such as temperature and composition (i.e., total organic carbon, water hardness, dissolve oxygen, and pH) can fluctuate with the seasons, which could affect methylene chloride concentrations in water and sediment pore water. In addition, methylene chloride monitoring data was collected only in certain areas, and within a limited number of states in the U.S. There were no measurements available immediately downstream from facilities releasing methylene chloride to surface water; these data are only a limited representation of ambient water. limitation

Additionally, as mentioned previously, EPA did not consider releases' combined impact on concentrations in the same waterbody. This may lead to an underestimation of surface water concentrations in waterbodies with multiple releases coming from one facility or waterbodies with multiple facilities contributing releases. For example, Clean Harbors Baltimore received multiple waste streams and had several releases to the same waterbody.

4.4.7 Key Assumptions and Uncertainties in the Human Health Risk Estimation

Occupational Exposure

Air concentrations. In most scenarios where data were available, EPA did not find enough data to determine complete statistical distributions of actual air concentrations for the workers exposed to methylene chloride. Ideally, EPA would like to know 50th and 95th percentiles for each exposed population. In the absence of percentile data for monitoring, the air concentration means and medians (means are preferred over medians) of the data sets served as substitutes for 50th percentiles (central tendencies) of the actual distributions, whereas high ends of ranges served as substitutes for 95th percentiles of the actual distributions. However, these substitutes are uncertain and are weak substitutes for the ideal percentiles. For instance, in the few cases where enough data were found to determine statistical means and 95th percentiles, the associated substitutes (i.e., medians and high ends of ranges) were shown to overestimate exposures, sometimes significantly. While it is clear that most air concentration data represent real exposure levels, EPA cannot determine whether these concentrations are representative of the statistical distributions of actual air concentrations to which workers are exposed. It is unknown whether these uncertainties overestimate or underestimate exposures. The range of air concentration estimates from central tendency to high-end was generally not large (e.g., less than 20-fold for most OESs). Because of this the results of risk characterization were generally not sensitive to the individual estimates of the central tendency and high-end separately but rather were based on considering both central tendency and high-end exposure estimates, which increase the overall confidence in the risk characterization. For example, where both the central tendency and high-end showed risk, EPA had higher confidence in the risk characterization.

Exposures for ONUs can vary substantially. Most data sources do not sufficiently describe the proximity of these employees to the exposure source. As such, exposure levels for the "occupational non-user" category will have high variability depending on the specific work activity performed. It is possible that some employees categorized as "occupational non-user" have exposures similar to those in the "worker" category depending on their specific work activity pattern. It is unknown whether these uncertainties overestimate or underestimate exposures.

Additionally, some data sources may be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. These sources may cause exposures to be overestimated.

Where data were not available, the modeling approaches used to estimate air concentrations also have uncertainties. Parameter values used in models did not all have distributions known to represent the modeled scenario. It is also uncertain whether the model equations generate results that represent actual workplace air concentrations. It is unknown whether these uncertainties overestimate or underestimate exposures. Additional model-specific uncertainties are included below.

Averaging Times. EPA cannot determine how accurately the assumptions of exposure frequencies (days/yr exposed) and exposed working years may represent actual exposure frequencies and exposed working years. For example, tenure is used to represent exposed working years, but many workers may not be exposed during their entire tenure. It is unknown whether these uncertainties overestimate or underestimate exposures, although the high-end values may result in overestimates when used in combination with high-end values of other parameters.

Dermal Exposure. As stated in Section 4.4.2.4, the *Dermal Exposure to Volatile Liquids Model* used for modeling occupational dermal exposure does not account for the transient exposure and exposure duration effect, which likely overestimate exposure. The model assumes one exposure event per day, which likely underestimates exposure. Surface areas of skin exposure are based on skin surface area of hands from EPA's Exposure Factors Handbook, but actual surface areas with liquid contact are unknown and uncertain for all OESs. For many OESs, the high-end assumption of contact over the full area of two hands likely overestimates exposures. Weight fractions are usually reported to CDR and shown in other literature sources as ranges, and EPA assessed only upper ends of ranges. The glove protection factors, based on the ECETOC TRA model as described in Section 2.4.1.1, are "what-if" assumptions and are uncertain. EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces of the OESs. Except where specified above, it is unknown whether most of these uncertainties overestimate or underestimate exposures. The representativeness of the modeling results toward the true distribution of dermal doses for the OESs is uncertain.

Consumer Exposure

EPA's approach recognizes the need to include uncertainty analysis. An important distinction for such an analysis concerns variability versus sensitivity – both aspects need to be addressed. Variability refers to the inherent heterogeneity or diversity of data in an assessment²⁴. It is "a quantitative description of the range or spread of a set of values"²⁵ and is often expressed through statistical metrics, such as variance or standard deviation, that reflect the underlying variability of the data. Sensitivity refers to an analysis of the predictability of a response variable, whereby a change in a given parameter or assumption affects a response variable. For a full discussion of the sensitivity analysis please refer to the Supplemental Information on Consumer Exposure Assessment, Section 2.1. Uncertainty refers to a lack of data or an incomplete understanding of the context of the risk assessment decision.

²⁴ <https://www.epa.gov/expobox/uncertainty-and-variability>

²⁵ <https://www.epa.gov/expobox/exposure-factors-handbook-chapter-2>

Variability cannot be reduced, but it can be better characterized. Uncertainty can be reduced by collecting more or better data. Quantitative methods to address uncertainty include non-probabilistic approaches such as sensitivity analysis and probabilistic methods such as Monte Carlo analysis. Uncertainty can also be addressed qualitatively, by including a discussion of factors such as data gaps and subjective decisions or instances where professional judgment was used.

With these approaches, the output of the model, CEM, is fully determined by the choices of parameter values and initial conditions. Stochastic approaches feature inherent randomness, such that a given set of parameter values and initial conditions can lead to an ensemble of different model outputs. Because EPA's largely deterministic approach involves choices regarding low, medium, and high values for highly influential factors such as chemical mass and frequency/duration of product use, it likely captures the range of potential exposure levels although it does not necessarily enable characterization of the full probabilistic distribution of all possible outcomes.

Certain inputs to which model outputs are sensitive, such as zone volumes and airflow rates, were not varied across product-use scenarios. As a result, model outcomes for extreme circumstances such as a relatively large chemical mass in a relatively low-volume environment likely are not represented among the model outcomes. Such extreme outcomes are believed to lie near the upper end (e.g., at or above the 90th percentile) of the exposure distribution.

Human Health Hazards

Effects resulting from acute exposure. There is uncertainty in converting the POD value from 1.5 hrs to PODs appropriate for the 15-min, 1-hr and 8-hr exposure durations used in the risk evaluation. EPA used a default approach ([Ten Berge et al., 1986](#)), which is a modification of Haber's rule, to convert the POD to other exposure durations. Although there are acute PBPK models, there are uncertainties associated with the PBPK model used for AEGLs, and there are few differences between the ten Berge and acute PBPK approaches for shorter exposure durations.

The adverse effect used in this risk evaluation was related to changes in a complex task as measured by Putz et al. ([1979](#)), which might not be identified in a study that measured simple reaction tasks. However, EPA applied a smaller UF for LOAEL to NOAEL (3 vs.10) when setting the benchmark MOE based on the severity of changes identified by Putz et al. ([1979](#)).

EPA determined that it is important to consider less severe effects rather than quantifying only more severe effects, in part, due to the possibility of serious harm and death as concentrations and exposure durations increase.

Cancer. Epidemiology studies are inconclusive for the lung and liver tumors modeled in the current assessment. Also, there are some mixed results in genotoxicity studies including negative results at certain concentrations. EPA did, however, address uncertainties in the enzyme considered to be associated with genotoxicity by using a PBPK model to account for differences between species and among humans.

There is uncertainty in the type of tumors modeled. Epidemiological studies are more consistent for the association between methylene chloride and hematopoietic-related cancers and humans do have increased frequencies of micronuclei and DNA damage in peripheral blood lymphocytes in workplaces using methylene chloride. Also, animal studies consistently identify methylene chloride exposure as associated with mammary tumors with a higher IUR than for the combined liver and lung tumor IUR. However, very few tumors from the animal studies are malignant. In addition, a small fraction 0.1% of fibroadenomas lead to carcinomas ([Russo, 2015](#)).

Exposures to methylene chloride were evaluated by inhalation and dermal routes separately. Inhalation and dermal exposures are assumed to occur simultaneously for workers and consumers. EPA chose not to employ simple additivity of exposure pathways within a condition of use because of the uncertainties present in the current exposure estimation procedures and this may lead to an underestimate of exposure. EPA does not have data that could be reliably modeled into the aggregate, which would be a more accurate approach than adding, such as through a PBPK model. Using an additive approach to aggregate risk in this case would result in an overestimate of risk. Given all the limitations that exist with the data, EPA's approach is the best available approach. This lack of aggregation may lead to an underestimate of exposure but based on physical chemical properties inhalation exposure represents the predominant exposure pathway.

4.5 Potentially Exposed or Susceptible Subpopulations

TSCA requires that the determination of whether a chemical substance presents an unreasonable risk include consideration of unreasonable risk to "a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation" by EPA. TSCA § 3(12) states that "the term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly." PESS are incorporated within the risk characterization (Section 4.3) and are described below.

EPA identified groups of individuals with greater exposure as 1) workers in occupational scenarios and 2) individuals in multiple age groups for the consumer exposure scenarios. EPA examined worker exposures in this risk evaluation for several occupational scenarios (see Section 2.4.1 for these exposure scenarios). For the evaluation of consumer exposures and as described in Section 2.4.2.3.2, dermal exposure results are presented for users of three possible age groups: adults and two youth age groups (16-20 years and 11-15 years). Inhalation exposures are presented as concentrations encountered for users and non-user bystander populations and are independent of age group.

In developing the hazard assessment, EPA evaluated available data to ascertain whether some human subpopulations may have greater susceptibility than the general population to the chemical's hazard(s). EPA identified several human subpopulations that are potentially more susceptible to the adverse health effects from methylene chloride compared with the general population. A genetic polymorphism in the GSTT1 enzyme results in a distribution of 32% GSTT1 +/+, 48% GSTT1 +/-, and 20% GSTT1 -/- individuals in the U.S. population ([Haber et](#)

[al., 2002](#)). GSTT1 +/+ individuals are more susceptible to getting cancer from methylene chloride (Section 3).

Individuals with cardiac disease are a potentially susceptible subpopulation. During exercise, cardiac patients have experienced angina more quickly after CO exposure, which is associated with increased COHb levels ([Nac/Aegl, 2008b](#)). EPA considers that increased COHb levels resulting from methylene chloride exposure may also result in similar adverse effects in individuals with cardiac disease.

The COHb generated from methylene chloride is additive to COHb in certain populations, exacerbating the increased susceptibility to angina among individuals with cardiac disease. For example, smokers have higher COHb levels than the general population ([ATSDR, 2000](#)). Also, individuals who are GSTT1 -/- may have higher COHb concentrations based on greater metabolism of methylene chloride via CYP450 2E1 than via the GSTT1 metabolic pathway ([Nac/Aegl, 2008b](#)). Furthermore, the hemoglobin of fetuses, infants and toddlers has greater affinity for CO compared with hemoglobin of adults, possibly resulting in increased COHb levels ([OEHHA, 2008b](#)). Finally, consuming alcohol can induce the CYP2E1 enzyme and increased COHb ([Nac/Aegl, 2008b](#)).

Although EPA has identified these potentially susceptible populations due to increased COHb levels, simultaneous exposure to methylene chloride and alcohol or other substances can also decrease the metabolic rate, attenuating the increased susceptibility among these individuals ([Nac/Aegl, 2008b](#)).

In addition to having greater exposure to methylene chloride in breastmilk ([Jensen, 1983](#); [Pellizzari et al., 1982](#); [Erickson et al., 1980](#)) and greater susceptibility from COHb, the newborn and infant are susceptible lifestages associated with rapid growth that includes the heart and brain. Also, Alexeeff and Kilgore ([1983](#)) identified a statistically significant difference in a passive avoidance learning task among three-day old mice exposed to methylene chloride compared with controls but no differences for 5- and 8-week old mice.

To account for variation in sensitivity within human populations, intraspecies UFs were applied for non-cancer effects. The UF values selected are described in section 3.2.5.2.

All potentially exposed and susceptible subpopulations are included in the quantitative and qualitative analyses described in this risk characterization (Section 4.3).

4.6 Aggregate and Sentinel Exposures

Section 2605(b)(4)(F)(ii) of TSCA requires the EPA, as a part of the risk evaluation, to describe whether aggregate or sentinel exposures under the conditions of use were considered and the basis for their consideration. The EPA has defined aggregate exposure as “*the combined exposures to an individual from a single chemical substance across multiple routes and across multiple pathways* (40 CFR § 702.33).” In this risk evaluation aggregate exposure was evaluated first by determining both the exposure to methylene inhalation and dermal contact separately. Time profiles of each type of exposure were estimated for a variety of occupational categories and household consumer uses, behaviors, and activity profiles. Inhalation exposure is specified by the air concentration encountered as a function of time during the workday or for 24 hr from the start of a household application. Dermal contact is characterized by the weight fraction of methylene chloride in the product being used, the surface area of skin (hands) exposed, and the duration of the dermal exposure. For workplace exposures inhalation and dermal exposures are assumed to occur simultaneous, i.e., both occur at the start of the task and continue through the end of the task, shift, or workday. For household exposures inhalation and dermal exposures occur at the start of the task and continue through the end of the task. EPA Consumer inhalation exposures typically continue for some time after the task is complete, although at a lower concentration, while the individual remains in the rest of house. The available PBPK models lack a dermal compartment and therefore a PBPK model for aggregating inhalation and dermal exposures is not reasonably available. Aggregating inhalation and dermal exposures without the use of a PBPK model would introduce additional uncertainties and was not included here. EPA chose not to employ simple additivity of exposure pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures. This lack of aggregation may lead to an underestimate of exposure but based on physical chemical properties inhalation exposure represents the predominant exposure pathway.

The EPA defines sentinel exposure as “*the exposure to a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures* (40 CFR § 702.33).” In terms of this risk evaluation, the EPA considered sentinel exposure by estimating the plausible upper bound relative to the highest exposure given the details of the conditions of use and the potential exposure scenarios. Sentinel exposures for workers are the high-end no PPE scenario within each OES. For consumer exposures, a range of consumer inhalation and dermal estimates for each consumer condition of use were provided by varying duration of use per event, amount of chemical in the product and mass of product used per event, while retaining central-tendency inputs for exposure factors and exposure setting characteristics. In presenting the inhalation results, high intensity use was characterized by the model iteration that utilized the 95th percentile duration of use and mass of product used [as presented in U.S. EPA (1987)] and the maximum weight fraction derived from product specific SDS, when available. Dermal exposures for high intensity use were characterized by the model iteration that utilized the 95th percentile duration of use and maximum weight fraction.

5 UNREASONABLE RISK DETERMINATION

5.1 Overview

In each risk evaluation under TSCA section 6(b), EPA determines whether a chemical substance presents an unreasonable risk of injury to health or the environment, under the conditions of use. These determinations do not consider costs or other non-risk factors. In making these determinations, EPA considers relevant risk-related factors, including, but not limited to: the effects of the chemical substance on health and human exposure to such substance under the conditions of use (including cancer and non-cancer risks); the effects of the chemical substance on the environment and environmental exposure under the conditions of use; the population exposed (including any potentially exposed or susceptible subpopulations (PESS)); the severity of hazard (including the nature of the hazard, the irreversibility of the hazard); and uncertainties. EPA also takes into consideration the Agency's confidence in the data used in the risk estimate. This includes an evaluation of the strengths, limitations, and uncertainties associated with the information used to inform the risk estimates and the risk characterization. This approach is in keeping with the Agency's final rule, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726).²⁶

This section describes the final unreasonable risk determinations for the conditions of use in the scope of the risk evaluation. The final unreasonable risk determinations are based on the risk estimates in the final risk evaluation, which may differ from the risk estimates in the draft risk evaluation due to peer review and public comments. Therefore, the final unreasonable risk determinations of some conditions of use may differ from those in the draft risk evaluation.

5.1.1 Human Health

EPA's risk evaluation identified non-cancer adverse effects from acute and chronic inhalation and dermal exposures to methylene chloride, and cancer from chronic inhalation and dermal exposures to methylene chloride. The health risk estimates for all conditions of use are in Section 4.1 (Table 4-2 and Table 4-3).

For the methylene chloride risk evaluation, EPA identified as Potentially Exposed or Susceptible Subpopulations: workers and ONUs, including males, females of reproductive age, and adolescents; and consumer users and bystanders (of any age group, including infants, toddlers, children, and elderly).

EPA evaluated exposures to workers, ONUs, consumer users, and bystanders, using reasonably available monitoring and modeling data for inhalation and dermal exposures, as applicable. For example, EPA assumed that ONUs and bystanders do not have direct contact with methylene chloride; therefore, non-cancer effects and cancer from dermal exposures to methylene chloride were not evaluated. The description of the data used for human health exposure is in Section 2.4.

²⁶ This risk determination is being issued under TSCA section 6(b) and the terms used, such as unreasonable risk, and the considerations discussed are specific to TSCA. Other statutes have different authorities and mandates and may involve risk considerations other than those discussed here.

Uncertainties in the analysis are discussed in Section 4.4 and considered in the unreasonable risk determination for each condition of use presented below, including the fact that the dermal model used for occupational exposures does not address variability in exposure duration and frequency. An additional uncertainty includes the use of data generated before the OSHA Methylene Chloride standard was updated in 1997.

EPA did not evaluate hazards or exposures to the general population, and as such the unreasonable risk determinations for relevant conditions of use do not account for exposures to the general population. Additional details regarding the general population are in Section 1.4.2.

5.1.1.1 Non-Cancer Risk Estimates

The risk estimates of non-cancer effects (MOEs) refers to adverse health effects associated with health endpoints other than cancer, including to the body's organ systems, such as reproductive/developmental effects, cardiac and lung effects, and kidney and liver effects. The MOE is the point of departure (POD) (an approximation of the no-observed adverse effect level (NOAEL) or benchmark dose level (BMDL)) for a specific health endpoint divided by the exposure concentration for the specific scenario of concern. Section 3.2.5 presents the PODs for acute and chronic non-cancer effects for methylene chloride and Section 4.3 presents the MOEs for acute and chronic non-cancer effects.

The MOEs are compared to a benchmark MOE. The benchmark MOE accounts for the total uncertainty in a POD, including, as appropriate: (1) the variation in sensitivity among the members of the human population (i.e., intrahuman/intraspecies variability); (2) the uncertainty in extrapolating animal data to humans (i.e., interspecies variability); (3) the uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure to lifetime exposure (i.e., extrapolating from subchronic to chronic exposure); and (4) the uncertainty in extrapolating from a lowest observed adverse effect level (LOAEL) rather than from a NOAEL. A lower benchmark MOE (e.g., 30) indicates greater certainty in the data (because fewer of the default UFs relevant to a given POD as described above were applied). A higher benchmark MOE (e.g., 1000) would indicate more uncertainty for specific endpoints and scenarios. However, these are often not the only uncertainties in a risk evaluation. The benchmark MOE for acute non-cancer risks for methylene chloride is 30 (accounting for intraspecies and LOAEL to NOAEL variability). The benchmark MOE for chronic non-cancer risks for methylene chloride is 10 (accounting for interspecies and intraspecies variability). Additional information regarding the benchmark MOE is in Section 4.3.

5.1.1.2 Cancer Risk Estimates

Cancer risk estimates represent the incremental increase in probability of an individual in an exposed population developing cancer over a lifetime (excess lifetime cancer risk (ELCR)) following exposure to the chemical. Standard cancer benchmarks used by EPA and other regulatory agencies are an increased cancer risk above benchmarks ranging from 1 in 1,000,000 to 1 in 10,000 (i.e., 1×10^{-6} to 1×10^{-4}) depending on the subpopulation exposed. Generally, EPA

considers 1×10^{-6} to 1×10^{-4} as the appropriate benchmark for the general population, consumer users, and non-occupational PESS.²⁷

EPA, consistent with 2017 NIOSH guidance,²⁸ used 1×10^{-4} as the benchmark for the purposes of this unreasonable risk determination for individuals in industrial and commercial work environments. It is important to note that 1×10^{-4} is not a bright line and EPA has discretion to make unreasonable risk determinations based on other cancer risk benchmarks as appropriate.

5.1.1.3 Determining Unreasonable Risk of Injury to Health

Calculated risk estimates (MOEs or cancer risk estimates) can provide a risk profile by presenting a range of estimates for different health effects for different conditions of use. A calculated MOE that is less than the benchmark MOE supports a determination of unreasonable risk of injury to health, based on non-cancer effects. Similarly, a calculated cancer risk estimate that is greater than the cancer benchmark supports a determination of unreasonable risk of injury to health from cancer. Whether EPA makes a determination of unreasonable risk depends upon other risk-related factors, such as the endpoint under consideration, the reversibility of effect, exposure-related considerations (e.g., duration, magnitude, or frequency of exposure, or population exposed), and the confidence in the information used to inform the hazard and exposure values. A calculated MOE greater than the benchmark MOE or a calculated cancer risk estimate less than the benchmark, alone do not support a determination of unreasonable risk, since EPA may consider other risk based factors when making an unreasonable risk determination.

When making an unreasonable risk determination based on injury to health of workers (who are one example of PESS), EPA also makes assumptions regarding workplace practices and exposure controls, including engineering controls or use of personal protective equipment (PPE). EPA's decisions for unreasonable risk to workers are based on high-end exposure estimates, in order to capture not only exposures for PESS but also to account for the uncertainties related to whether or not workers are using PPE. However, EPA does not assume that ONUs use PPE. This is particularly relevant to methylene chloride, for which under the OSHA standard the only respirators that can be used are supplied-air respirators (i.e., APF of 25 would be the lowest APF that could be considered), further discussed in Section 2.4.1.1. Therefore, for each condition of use of methylene chloride with an identified risk for workers, EPA assumes, as a baseline, the use of a respirator with an APF of 25 or 50. Similarly, EPA assumes the use of gloves with PF of 5 and 10 in commercial settings and gloves with PF of 5 and 20 in industrial settings. However, EPA assumes that for some conditions of use, the use of appropriate respirators is not a standard

²⁷ As an example, when EPA's Office of Water in 2017 updated the Human Health Benchmarks for Pesticides, the benchmark for a "theoretical upper-bound excess lifetime cancer risk" from pesticides in drinking water was identified as 1 in 1,000,000 to 1 in 10,000 over a lifetime of exposure (EPA. Human Health Benchmarks for Pesticides: Updated 2017 Technical Document (pp.5). (EPA 822-R -17 -001). Washington, DC: U.S. Environmental Protection Agency, Office of Water January 2017. <https://www.epa.gov/sites/production/files/2015-10/documents/hh-benchmarks-techdoc.pdf>). Similarly, EPA's approach under the Clean Air Act to evaluate residual risk and to develop standards is a two-step approach that "includes a presumptive limit on maximum individual lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand" and consideration of whether emissions standards provide an ample margin of safety to protect public health "in consideration of all health information, including the number of persons at risk levels higher than approximately 1 in 1 million, as well as other relevant factors" (54 FR 38044, 38045, September 14, 1989).

²⁸ NIOSH Current intelligence bulletin 68: NIOSH chemical carcinogen policy (Whittaker et al. 2016).

industry practice, based on professional judgement given the burden associated with the use of supplied-air respirators, including the expense of the equipment and the necessity of fit-testing and training for proper use. Similarly, EPA does not assume that as a standard industry practice that workers in dry cleaning facilities use gloves for spot cleaning. Once EPA has applied the appropriate PPE assumption for a particular condition of use in each unreasonable risk determination, in those instances when EPA assumes PPE is used, EPA also assumes that the PPE is used in a manner that achieves the stated APF or PF.

In the methylene chloride risk characterization, neurotoxicity effects (CNS depression) were identified as the most sensitive endpoint for non-cancer adverse effect from acute inhalation and dermal exposures and liver effects were identified as the most sensitive endpoint for non-cancer adverse effects from chronic inhalation and dermal exposures for all conditions of use. However, additional risks associated with other adverse effects (e.g. other nervous system effects, immune system effects; reproductive and developmental effects; and irritation/burns) were identified for acute and chronic exposures. Determining unreasonable risk by using CNS and liver effects will also include the unreasonable risk from other endpoints resulting from acute or chronic inhalation and dermal exposures.

In accordance with EPA's Guidelines for Carcinogen Risk Assessment, methylene chloride is considered "likely to be carcinogenic to humans" and EPA calculated cancer risk estimates with a linear model. The cancer analysis is described in Section 3.2. EPA considered cancer risks estimates from chronic dermal or inhalation exposures in the unreasonable risk determination.

When making a determination of unreasonable risk, the Agency has a higher degree of confidence where uncertainty is low. Similarly, EPA has high confidence in the hazard and exposure characterizations when, for example, the basis for the characterizations is measured or monitoring data or a robust model and the hazards identified for risk estimation are relevant for conditions of use. Where EPA has made assumptions in the scientific evaluation, whether or not those assumptions are protective is also a consideration. Additionally, EPA considers the central tendency and high-end exposure levels when determining the unreasonable risk. High-end risk estimates (e.g., 95th percentile) are generally intended to cover individuals or sub-populations with greater exposure (PESS) and central tendency risk estimates are generally estimates of average or typical exposure. The high volatility of methylene chloride and potentially severe effects from short term (1-hr) exposure are factors when weighing uncertainties.

EPA may make a determination of no unreasonable risk for conditions of use where the substance's hazard and exposure potential, or where the risk-related factors described previously, lead the Agency to determine that the risks are not unreasonable.

5.1.2 Environment

EPA calculated a risk quotient (RQ) to compare environmental concentrations against an effect level.

The environmental concentration is determined based on the levels of the chemical released to the environment (e.g., surface water, sediment, soil, biota) under the conditions of use, based on the fate properties, release potential, and reasonably available environmental monitoring data. The effect level is calculated using concentrations of concern that represent hazard data for

aquatic, sediment-dwelling, and terrestrial organisms. Section 4.2. provides more detail regarding the risk quotient for methylene chloride.

5.1.2.1 Determining Unreasonable Risk of Injury to the Environment

An RQ equal to 1 indicates that the exposures are the same as the concentration that causes effects. An RQ less than 1, when the exposure is less than the effect concentration, supports a determination that there is no unreasonable risk of injury to the environment. An RQ greater than 1, when the exposure is greater than the effect concentration, supports a determination that there is unreasonable risk of injury to the environment. Consistent with EPA's human health evaluations, other risk-based factors may be considered (e.g., confidence in the hazard and exposure characterization, duration, magnitude, uncertainty) for purposes of making an unreasonable risk determination.

EPA considered the effects on the aquatic, sediment dwelling and terrestrial organisms. EPA provides estimates for environmental risk in Section 4.1. and Table 4-1.

5.2 Detailed Unreasonable Risk Determinations by Condition of Use

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
Manufacturing	Domestic manufacturing	Manufacturing	No	Section 5.2.1.1 and Section 5.2.2.
	Import	Import	Yes	Section 5.2.1.2 and Section 5.2.2.
Processing	Processing as a reactant	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants)	No	Section 5.2.1.3 and Section 5.2.2.
		Intermediate for pesticide, fertilizer, and other agricultural chemical manufacturing		
		Petrochemical manufacturing*		
		Intermediate for other chemicals		
Processing	Processing - incorporation into formulation, mixture or reaction products	Solvents (for cleaning or degreasing), including manufacturing of: <ul style="list-style-type: none"> All other basic organic chemical Soap, cleaning compound and toilet preparation 	Yes	Section 5.2.1.4 and Section 5.2.2.

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
		Solvents (which become part of product formulation or mixture), including manufacturing of: <ul style="list-style-type: none"> • All other chemical product and preparation • Paints and coatings 		
		Propellants and blowing agents for all other chemical product and preparation manufacturing		
		Propellants and blowing agents for plastics product manufacturing		
		Paint additives and coating additives not described by other codes*		
		Laboratory chemicals for all other chemical product and preparation manufacturing		
		Laboratory chemicals for other industrial sectors*		
		Processing aid, not otherwise listed for petrochemical manufacturing		
		Adhesive and sealant chemicals in adhesive manufacturing		
		Oil and gas drilling, extraction, and support activities*		
Processing	Repackaging	Solvents (which become part of product formulation or mixture) for all other chemical product and preparation manufacturing	Yes	Section 5.2.1.5 and Section 5.2.2.
		All other chemical product and preparation manufacturing*		
Processing	Recycling	Recycling	No	Section 5.2.1.6 and Section 5.2.2.
Distribution in commerce	Distribution	Distribution	No	Section 5.2.1.7 and Section 5.2.2.

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
Industrial/ commercial use	Solvent (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)	Yes	Section 5.2.1.8 and Section 5.2.2.
		In-line vapor degreaser (e.g., conveyORIZED, web cleaner)	Yes	Section 5.2.1.9 and Section 5.2.2.
		Cold cleaner	Yes	Section 5.2.1.10 and Section 5.2.2.
		Aerosol spray degreaser/cleaner	Yes	Section 5.2.1.11 and Section 5.2.2.
	Adhesives and sealants	Single component glues and adhesives and sealants and caulks	Yes	Section 5.2.1.12 and Section 5.2.2.
	Paints and coatings including commercial paint and coating removers	Paints and coatings use	Yes	Section 5.2.1.13. and Section 5.2.2.
		Commercial paint and coating removers, including furniture refinisher	Yes	Section 5.2.1.14. and Section 5.2.2.
		Adhesive/caulk removers	Yes	Section 5.2.1.15. and Section 5.2.2.
	Metal products not covered elsewhere	Degreasers – aerosol degreasers and cleaners	Yes	Section 5.2.1.16. and Section 5.2.2.
		Degreasers – non-aerosol degreasers and cleaners	Yes	Section 5.2.1.17. and Section 5.2.2.
	Fabric, textile and leather products not covered elsewhere	Textile finishing and impregnating/surface treatment products	Yes	Section 5.2.1.18. and Section 5.2.2.
	Automotive care products	Functional fluids for air conditioners: refrigerant, treatment, leak sealer	Yes	Section 5.2.1.19. and Section 5.2.2.
	Automotive care products	Interior car care – spot remover	Yes	Section 5.2.1.20. and Section 5.2.2.
		Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Yes	Section 5.2.1.21. and Section 5.2.2.
Apparel and footwear care products	Post-market waxes and polishes applied to footwear (e.g., shoe polish)	Yes	Section 5.2.1.22. and Section 5.2.2.	
Laundry and dishwashing products	Spot remover for apparel and textiles	Yes	Section 5.2.1.23. and Section 5.2.2.	

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
	Lubricants and greases	Liquid lubricants and greases	Yes	Section 5.2.1.24. and Section 5.2.2.
		Spray lubricants and greases	Yes	Section 5.2.1.25. and Section 5.2.2.
		Degreasers – aerosol degreasers and cleaners	Yes	Section 5.2.1.26. and Section 5.2.2.
		Degreasers –non-aerosol degreasers and cleaners	Yes	Section 5.2.1.27. and Section 5.2.2.
	Building/ construction materials not covered elsewhere	Cold pipe insulation	Yes	Section 5.2.1.28. and Section 5.2.2.
	Solvents (which become part of product formulation or mixture)	All other chemical product and preparation manufacturing	Yes	Section 5.2.1.29. and Section 5.2.2.
	Processing aid not otherwise listed	In multiple manufacturing sectors	Yes	Section 5.2.1.30. and Section 5.2.2.
	Propellants and blowing agents	Flexible polyurethane foam manufacturing	Yes	Section 5.2.1.31. and Section 5.2.2.
	Other uses	Laboratory chemicals - all other chemical product and preparation manufacturing	No	Section 5.2.1.32. and Section 5.2.2.
		Electrical equipment, appliance, and component manufacturing	Yes	Section 5.2.1.33. and Section 5.2.2.
		Plastic and rubber products (Plastic Product Manufacturing)	Yes	Section 5.2.1.34. and Section 5.2.2.
		Plastic and rubber products (Cellulose Triacetate Film Production)	Yes	Section 5.2.1.35. and Section 5.2.2.
		Anti-adhesive agent – anti-spatter welding aerosol	Yes	Section 5.2.1.36. and Section 5.2.2.
		Oil and gas drilling, extraction, and support activities	Yes	Section 5.2.1.37. and Section 5.2.2.
Toys, playground, and sporting equipment - including novelty articles (toys, gifts, etc.)		Yes	Section 5.2.1.38. and Section 5.2.2.	
Lithographic printing cleaner		Yes	Section 5.2.1.39. and Section 5.2.2.	
Carbon remover, wood floor cleaner, brush cleaner		Yes	Section 5.2.1.40 and Section 5.2.2.	

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
Consumer uses	Solvent (cleaning or degreasing)	Aerosol spray degreaser/cleaner	Yes	Section 5.2.1.41. and Section 5.2.2.
	Adhesives and sealants	Single component glues and adhesives and sealants and caulks	Yes	Section 5.2.1.42. and Section 5.2.2.
	Paints and coatings	Paints and coatings use (brush cleaner)	Yes	Section 5.2.1.43. and Section 5.2.2.
		Adhesive/caulk removers	Yes	Section 5.2.1.44. and Section 5.2.2.
	Metal products not covered elsewhere	Degreasers – aerosol and non-aerosol degreasers and cleaners	Yes	Section 5.2.1.45. and Section 5.2.2.
	Automotive care products	Functional fluids for air conditioners: refrigerant, treatment, leak sealer	Yes	Section 5.2.1.46. and Section 5.2.2.
		Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner	Yes	Section 5.2.1.47. and Section 5.2.2.
	Lubricants and greases	Liquid and spray lubricants and greases	Yes	Section 5.2.1.48. and Section 5.2.2.
		Degreasers – aerosol and non-aerosol degreasers and cleaners	Yes	
	Building/ construction materials not covered elsewhere	Cold pipe insulation	Yes	Section 5.2.1.49. and Section 5.2.2.
	Arts, crafts and hobby materials	Crafting glue and cement/concrete	Yes	Section 5.2.1.50. and Section 5.2.2.
	Other uses	Anti-adhesive agent – anti-spatter welding aerosol	Yes	Section 5.2.1.51. and Section 5.2.2.
		Carbon remover and brush cleaner	Yes	Section 5.2.1.52. and Section 5.2.2.
Disposal	Disposal	Industrial pre-treatment	No	Section 5.2.1.53. and Section 5.2.2.
		Industrial wastewater treatment		
		Publicly owned treatment works (POTW)		
		Underground injection		
		Municipal landfill		

Table 5-1. Categories and Subcategories of Conditions of Use Included in the Scope of the Risk Evaluation

Life Cycle Stage	Category ^a	Subcategory ^b	Unreasonable Risk	Detailed Risk Determination
		Hazardous landfill		
		Other land disposal		
		Municipal waste incinerator		
		Hazardous waste incinerator		
		Off-site waste transfer		

^a These categories of conditions of use appear in the Life Cycle Diagram, reflect CDR codes, and broadly represent conditions of use of methylene chloride in industrial and/or commercial settings and of consumer uses.

^b These subcategories reflect more specific uses of methylene chloride.

^c Reported for the following sectors in the 2016 CDR for manufacturing of: plastic materials and resins, plastics products, miscellaneous, all other chemical product and preparation (U.S. EPA, 2016).

^d Reported for the following sectors in the 2016 CDR for manufacturing of: petrochemicals, plastic materials and resins, plastics products, miscellaneous and all other chemical products * (U.S. EPA, 2016) also including as a chemical processor for polycarbonate resins and cellulose triacetate (photographic film).

^e Consumer paint and coating remover uses are already addressed through rulemaking (see 40 CFR Part 751, Subpart B) and are outside the scope of this risk evaluation.

* Conditions of use with CBI or unknown function were evaluated and considered for the methylene chloride risk evaluation; however, the non-CBI elements of the category, subcategory, function and industrial sector were used in the analysis as these data were higher quality. This applies to: CBI function for petrochemical manufacturing, paint additives and coating additives not described by other codes for CBI industrial sector, laboratory chemicals for CBI industrial sectors, manufacturing of CBI and oil and gas drilling, extraction, and support activities.

** Although EPA has identified both industrial and commercial uses here for purposes of distinguishing scenarios in this document, the Agency interprets the authority over “any manner or method of commercial use” under TSCA section 6(a)(5) to reach both.

5.2.1 Human Health

5.2.1.1 Manufacturing – Domestic Manufacturing – Manufacturing (Domestic manufacture)

Section 6(b)(4)(A) unreasonable risk determination for domestic manufacture of methylene chloride: Does not present an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was no unreasonable risk of

non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the domestic manufacturing of methylene chloride does not present an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures from the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute inhalation exposures at the high-end for 15-minute TWA do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data from one source. The data may not be representative of exposures across the range of facilities that manufacture methylene chloride.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is no unreasonable risk of injury to health (workers and ONUs) from domestic manufacturing of methylene chloride.

5.2.1.2 Manufacturing – Import – Import (Import)

Section 6(b)(4)(A) unreasonable risk determination for import of methylene chloride: Presents an unreasonable risk of injury to health (ONUs); does not present an unreasonable risk of injury to health (workers).

For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency. For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE.

EPA's determination that the import of methylene chloride presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the

benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures, do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data collected at one repackaging facility. Methylene chloride may be imported into the United States in bulk containers and may be repackaged into smaller containers for resale. The monitoring data may not be representative of exposures across the range of facilities that import methylene chloride.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (ONUs) from the import of methylene chloride.

5.2.1.3 Processing – Processing as a reactant – Intermediate in industrial gas manufacturing; intermediate for pesticide, fertilizer, and other agricultural chemical manufacturing; use in petrochemical manufacturing; intermediate for other chemicals (Processing as a reactant)

Section 6(b)(4)(A) unreasonable risk determination for processing of methylene chloride as a reactant: Does not present an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency, point estimate, and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the processing of methylene chloride as a reactant does not present an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and

cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures from the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposure at the point estimate and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data reflective of current operations provided by one fluorochemical manufacturing facility; there is uncertainty regarding how well the data represent activities at all processing facilities.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is no unreasonable risk of injury to health (workers and ONUs) from processing of methylene chloride as a reactant.

5.2.1.4 Processing – Incorporation into formulation, mixture, or reaction products – Solvents for cleaning or degreasing; solvents which become part of product formulation or mixture; propellants and blowing agents for all other chemical products and preparation manufacturing; propellants and blowing agents for plastic product manufacturing; paints and coating additives not described by other codes; laboratory chemicals for all other chemical product and preparation manufacturing; laboratory chemicals for other industrial sectors; processing aid, not otherwise listed for petrochemical manufacturing; adhesive and sealant chemicals in adhesive manufacturing; oil and gas drilling, extraction, and support activities (Processing into a formulation, mixture, or reaction product)

Section 6(b)(4)(A) unreasonable risk determination for processing of methylene chloride into a formulation, mixture, or reaction product: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the processing of methylene chloride into a formulation, mixture, or reaction product presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal monitoring data from one source. The data may not be representative of exposures across the range of facilities that process methylene chloride into formulation, mixture or reaction product.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from the processing of methylene chloride into a formulation, mixture, or reaction product.

5.2.1.5 Processing – Repackaging – Solvents (which become part of product formulation or mixture) for all other chemical product and preparation manufacturing; all other chemical product and preparation manufacturing (Repackaging)

Section 6(b)(4)(A) unreasonable risk determination for repackaging of methylene chloride:
Presents an unreasonable risk of injury to health (ONUs); does not present an unreasonable risk of injury to health (workers).

For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency. For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic

(liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE.

EPA's determination that the repackaging of methylene chloride presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data collected at one repackaging facility. The data may not be representative of exposures across the range of facilities that repackaging methylene chloride.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (ONUs) from the repackaging of methylene chloride.

5.2.1.6 Processing – Recycling – Recycling (Recycling)

Section 6(b)(4)(A) unreasonable risk determination for recycling of methylene chloride: Does not present an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the recycling of methylene chloride does not present an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data provided by two sources. The data may not be representative of exposures across the range of facilities that recycle methylene chloride.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is no unreasonable risk of injury to health (workers and ONUs) from the recycling of methylene chloride.

5.2.1.7 Distribution in Commerce – Distribution – Distribution

Section 6(b)(4)(A) unreasonable risk determination for distribution in commerce of methylene chloride: Does not present an unreasonable risk of injury to health (workers and ONUs).

For the purposes of the unreasonable risk determination, distribution in commerce of methylene chloride is the transportation associated with the moving of methylene chloride in commerce. The loading and unloading activities are associated with other conditions of use. EPA assumes transportation of methylene chloride is in compliance with existing regulations for the transportation of hazardous materials, and emissions are therefore minimal (with the exception of spills and leaks, which are outside the scope of the risk evaluation). Based on the limited emissions from the transportation of chemicals, EPA determines there is no unreasonable risk of injury to health (workers and ONUs) from the distribution in commerce of methylene chloride.

5.2.1.8 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (e.g., open-top, closed-loop) (Solvent for batch vapor degreasing)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as solvent for batch vapor degreasing: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, and of cancer from chronic inhalation exposures at the high-end.

EPA's determination that the industrial and commercial use of methylene chloride as solvent for batch vapor degreasing presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- The inhalation exposures were assessed using modeling data by performing near-field and far-field inhalation concentrations in the open-top vapor degreasing (OTVD) scenario for workers and ONUs. Uncertainties in the analysis include the unknown methodology used by industries to estimate the emission data used in the model and the representativeness of the air concentrations generated by the model toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as solvent for batch vapor degreasing.

5.2.1.9 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (e.g., conveyORIZED, web cleaner) (Solvent for in-line vapor degreasing)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as solvent for in-line vapor degreasing: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures, at the central tendency and high-end.

EPA's determination that the industrial and commercial use of methylene chloride as solvent for in-line vapor degreasing presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end support an unreasonable risk determination. The risk estimates at the central tendency of non-cancer effects from acute inhalation exposures when assuming use of respirators with APF of 50 approximate the benchmark and support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Inhalation exposures were assessed using modeling data by performing near-field and far-field inhalation concentrations in the conveyORIZED vapor degreasing scenario for both workers and ONUs. Uncertainties in the analysis include the unknown methodology used by industries to estimate the emission data used in the model and the representativeness of the air concentrations generated by the model toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as solvent for in-line vapor degreasing.

5.2.1.10 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Cold cleaner (Solvent for cold cleaning)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as solvent for cold cleaning: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as solvent for cold cleaning presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data from one source in published literature. The data may not be representative of exposures across the range of facilities that use methylene chloride as solvent for cold cleaning
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as solvent for cold cleaning.

5.2.1.11 **Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner (Solvent for aerosol spray degreaser/cleaner)**

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as solvent for aerosol spray degreaser/cleaner: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as solvent for aerosol spray degreaser/cleaner presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride as solvent for aerosol spray degreasers/cleaners.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride as solvent for aerosol spray degreaser/cleaner.

5.2.1.12 Industrial/Commercial Use – Adhesives and sealants – Single component glues and adhesives and sealants and caulks (Adhesives, sealants and caulks)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in adhesives, sealants and caulks: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in adhesives, sealants and caulks presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis including uncertainties related to the exposures for ONUs:

- The workers considered included the “sprayer” of the methylene chloride adhesive; the “non-sprayers” that handle the methylene chloride adhesive or spend the majority of their shift working in an area where spraying occurs; and worker exposure during an unknown method of application.
- For workers (sprayers), when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination. The high-end risk estimates of non-cancer effects from acute inhalation exposures when assuming use of respirators with APF of 50 approximate the benchmark and support an unreasonable risk determination.
- For workers (sprayers), when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination.
- For workers (non-sprayers), when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end do not support an unreasonable risk determination, and when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination.
- For workers (unknown application method), when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers (unknown application method), without assuming use of PPE, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination.
- For workers (sprayers, non-sprayers, and unknown application method), when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.

- For workers (sprayers, non-sprayers, and unknown application methods), the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposure was assessed using monitoring data for both spray and non-spray industrial adhesive applications for workers. For some monitoring data, the method of application could not be determined, and these are included as unknown application method. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in adhesives, sealants and caulks.

5.2.1.13 Industrial/Commercial Use – Paints and coatings use including commercial paint and coating removers – Paints and coatings use (Paints and coatings)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene in paints and coatings: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, and of cancer from chronic inhalation at the high-end, without assuming use of respirators. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in paints and coatings presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in paints and coatings

- For workers, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data for both spray/coating operations and unknown application method operations. Uncertainties in the analysis include the representativeness of the inhalation air concentration data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in paints and coatings.

5.2.1.14 Industrial/Commercial Use – Paints and coatings including commercial paint and coating removers – Commercial paint and coating removers, including furniture refinisher (Paint and coating removers)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene in paint and coating removers: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, and of cancer from chronic inhalation at the central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency, and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in paint and coating removers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Appendix L; section 4.2.2.1.12) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene

chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- Ten different exposures scenarios were used to evaluate the industrial and commercial use of methylene chloride in paint and coating removers: professional contractors, automotive refinishing, furniture refinishing, art restoration and conservation, aircraft paint stripping, graffiti removal, non-specific workplace settings – immersion of stripping of wood, non-specific workplace settings – immersion of stripping of metal and wood, non-specific workplace settings – unknown, and one Department of Defense-specific scenario.
- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in paint and coating removers.
- For workers, when assuming use of gloves with PF of 10, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data as outlined in the 2014 Risk Assessment on Paint Stripping Use for Methylene Chloride and additional data provided by the Department of Defense.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in paint and coating removers.

5.2.1.15 Industrial/Commercial Use – Paints and coatings including commercial paint and coating removers – Adhesive/caulk remover (Adhesive and caulk removers)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in adhesive and caulk removers: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures, at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in adhesive and caulk removers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 10, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data. EPA did not find specific industry information exposure data for adhesive and caulk removers. Based on worker activities, EPA assumes that the use of adhesive and caulk removers is similar to paint stripping by professional contractors. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in adhesive and caulk removers.

5.2.1.16 Industrial/Commercial Use – Metal products not covered elsewhere – Degreasers – aerosol degreasers and cleaners (Metal aerosol degreasers)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as a metal aerosol degreaser: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that

there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in metal aerosol degreasers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in metal aerosol degreasers.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in metal aerosol degreasers.

5.2.1.17 Industrial/Commercial Use – Metal products not covered elsewhere – Degreasers – non-aerosol degreasers and cleaners (Metal non-aerosol degreasers)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in metal non-aerosol degreasers: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in metal non-aerosol degreasers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using methylene chloride in metal non-aerosol degreasing.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in metal non-aerosol degreasers.

5.2.1.18 Industrial/Commercial Use – Fabric, textile and leather products not covered elsewhere – Textile finishing and impregnating/surface treatment products (Finishing products for fabric, textiles and leather)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in finishing products for fabric, textiles, and leather: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) inhalation exposures at the high-end, and of non-cancer effects from chronic (liver) inhalation exposure at the central tendency and high-end, without assuming use of respirators. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in finishing products for fabric, textiles and leather presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis including uncertainties related to the exposures for ONUs:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in finishing products for fabric, textile and leather products.
- For workers, when assuming use of gloves with PF of 10, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic inhalation and chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of non-cancer effects from acute inhalation exposures and cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data for workers from OSHA inspections at apparel manufacturing sites. Uncertainties in the analysis include the lack of specific worker activity for the monitoring data and the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in finishing products for fabric, textiles and leather.

5.2.1.19 Industrial/Commercial Use – Automotive care products – Functional fluids for air conditioners: refrigerant, treatment, leak sealer (Automotive care products (functional fluids for air conditioners))

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in automotive care products (functional fluids for air conditioners): Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in automotive care products (functional fluids for air conditioners) presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10, and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of

injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in automotive care products (functional fluids for air conditioners).

5.2.1.20 Industrial/Commercial Use – Automotive care products – Interior car care – spot remover (Automotive care products (interior care))

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in automotive care products (interior care): Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in automotive care products (interior care) presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in automotive care products (interior care).
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in automotive care products interior car care.

5.2.1.21 Industrial/Commercial Use – Automotive care products – Degreasers: gasket remover, transmission cleaners, carburetor cleaner, brake quieter/cleaner (Automotive care products (degreasers))

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in automotive care products (degreasers): **Presents unreasonable risk of injury to health (workers)**; does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in automotive care products (degreasers) presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in automotive care products (degreasers).
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in automotive care products (degreasers).

5.2.1.22 Industrial/Commercial Use – Apparel and footwear care products – Post-market waxes and polishes applied to footwear (Apparel and footwear care products)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in apparel and footwear care products: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency and high-end.

EPA's determination that the industrial and commercial use of methylene chloride in apparel and footwear care products presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in apparel and footwear care products.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in apparel and footwear care products.

5.2.1.23 Industrial/Commercial Use – Laundry and dishwashing products – Spot remover for apparel and textiles (Spot removers for apparel and textiles)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in spot removers for apparel and textiles: Presents an unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation at the high-end, without assuming use of respirators. In addition, for workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) dermal exposures at the central tendency and high-end, without assuming use of gloves. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in spot removers for apparel and textiles presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- EPA does not assume workers use any type of respirator or gloves during industrial and commercial use of methylene chloride in spot removers for apparel and textiles.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data for methylene chloride-containing products during use as a spot cleaner. EPA used OSHA data for Industrial Launderers and Dry Cleaning and Laundry Services. Uncertainties in the analysis include the lack of specific worker activity for the monitoring data and the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using methylene chloride in spot removers for apparel and textiles.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in spot removers for apparel and textiles.

5.2.1.24 Industrial/Commercial Use – Lubricant and greases – Liquid lubricants and greases (Liquid lubricants and greases)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in liquid lubricants and greases: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in liquid lubricants and greases presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using methylene chloride in liquid lubricants and greases.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in liquid lubricants and greases.

5.2.1.25 Industrial/Commercial Use – Lubricants and greases – Spray lubricants and greases (Spray lubricants and greases)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in spray lubricants and greases: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in spray lubricants and greases presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in spray lubricants and greases.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in spray lubricants and greases.

5.2.1.26 Industrial/Commercial Use – Lubricants and greases – Degreasers – Aerosol degreasers and cleaners (Aerosol degreasers and cleaners)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in aerosol degreasers and cleaners: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in aerosol degreasers and cleaners presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in aerosol degreasers and cleaners.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in aerosol degreasers and cleaners.

5.2.1.27 Industrial/Commercial Use – Lubricants and greases – Non-aerosol degreasers and cleaners (Non-aerosol degreasers and cleaners)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in non-aerosol degreasers and cleaners: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in non-aerosol degreasers and cleaners presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposure was assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using methylene chloride in non-aerosol degreasers and cleaners.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of

injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in non-aerosol degreasers and cleaners.

5.2.1.28 Industrial/Commercial Use – Building/construction materials not covered elsewhere – Cold pipe insulation (Cold pipe insulations)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride in cold pipe insulation: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in cold pipe insulations presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in cold pipe insulations.
- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in cold pipe insulations.

5.2.1.29 Industrial/Commercial Use – Solvents (which become part of product formulation or mixture) – All other chemical product and preparation manufacturing (Solvent that becomes part of a formulation or mixture)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as solvent that becomes part of a formulation or mixture: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as solvent that becomes part of a formulation or mixture presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data. The data may not be representative of exposures across the range of facilities that process methylene chloride as solvent which becomes part of formulation or mixture.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as solvent that becomes part of a formulation or mixture.

5.2.1.30 Industrial/Commercial Use – Processing aid not otherwise listed – In multiple manufacturing sectors (Processing aid)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as a processing aid: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures, at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as processing aid presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data from six studies. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using methylene chloride as processing aid.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of

injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as processing aid.

5.2.1.31 Industrial/Commercial Use – Propellants and blowing agents – Flexible polyurethane foam manufacturing (Propellant and blowing agent)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as propellant and blowing agent: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures, at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as propellant and blowing agent presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal monitoring data samples from several sources, and cover activities such as application of mold release, foam manufacturing (blowing), blending, and sawing in the foam or plastic industry and tractor trailer construction. As described in Section 2.4.1.2.15, regulations (Final National Emissions Standards for Hazardous Air Pollutants (NESHAP) for Area Sources: Polyurethane Foam Production and Fabrication (72 FR 38864)) have limited the use of methylene chloride in polyurethane foam production and fabrication and some sources provided only

concentration ranges rather than discrete data points. Other uncertainties include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.

- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as propellant and blowing agent.

5.2.1.32 Industrial/Commercial Use – Other uses – Laboratory chemicals - all other chemical product and preparation manufacturing (Laboratory chemical)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as laboratory chemical: Does not present an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver), or of cancer from chronic inhalation at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as laboratory chemical does not present an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposures do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 20 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal monitoring data samples. Uncertainties in the analysis include the representativeness of the monitoring data toward

the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.

- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is no unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride as laboratory chemical.

5.2.1.33 Industrial/Commercial Use – Other uses – Electrical equipment, appliance, and component manufacturing (Electrical equipment, appliance, and component manufacturing)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride for electrical equipment, appliance, and component manufacturing: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride for electrical equipment, appliance, and component manufacturing presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account

for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.

- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride for electrical equipment, appliance, and component manufacturing.

5.2.1.34 Industrial/Commercial Use – Other uses – Plastic and rubber products (plastic product manufacturing) (Plastic and rubber products manufacturing)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride for plastic and rubber products manufacturing: Presents an unreasonable risk of injury to health (ONUs); does not present an unreasonable risk of injury to health (workers).

For ONUs, EPA found that there was unreasonable risk of non-cancer effects from chronic (liver) inhalation exposures. For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation exposures at the high-end, when assuming use of PPE, and from chronic dermal exposures at the central tendency and high-end, without assuming use of PPE.

EPA's determination that the industrial and commercial use of methylene chloride for plastic and rubber products manufacturing presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end do not support an unreasonable risk determination. When assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.

- For ONUs, the high-end risk estimates of non-cancer effects from acute inhalation exposures approximate the benchmark and do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished to calculate risk estimates of cancer; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from chronic inhalation exposures when determining ONUs' unreasonable risk of cancer. For non-cancer effects, EPA was able to calculate different risk estimates for workers and ONUs and the high-end risk estimates were used.
- Inhalation exposures were assessed using personal monitoring data samples, and the data may or may not be reflective of exposures to ONUs. Uncertainties in the analysis also include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (ONUs) from industrial and commercial use of methylene chloride for plastic and rubber products manufacturing.

5.2.1.35 Industrial/Commercial Use – Other uses – Plastic and rubber products (cellulose triacetate film production) (Cellulose triacetate film production)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride for cellulose triacetate film production: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency and high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and of cancer from chronic inhalation exposures, at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in cellulose triacetate film production presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the central tendency and high-end support an unreasonable risk determination.

- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the central tendency and high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites using of methylene chloride in cellulose triacetate film production.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in cellulose triacetate film production.

5.2.1.36 Industrial/Commercial Use – Other uses – Anti-adhesive agent – anti-spatter welding aerosol (Anti-spatter welding aerosol)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as anti-spatter welding aerosol: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures, and cancer from chronic inhalation exposures at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride as anti-spatter welding aerosol presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride as anti-spatter welding aerosol.

- For workers, when assuming use of gloves with PF of 5 and 10, the risk estimates of non-cancer effects from acute and chronic dermal exposure do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. Inhalation exposures were additionally assessed using modeled data by performing near-field and far-field inhalation concentrations for aerosol degreasing for both workers and ONUs, which support the conclusions in the monitoring data.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride as anti-spatter welding aerosol.

5.2.1.37 Industrial/Commercial Use – Other uses – Oil and gas drilling, extraction, and support activities (Oil and gas drilling, extraction, and support activities)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride for oil and gas drilling, extraction, and support activities: **Presents an unreasonable risk of injury to health (workers and ONUs).**

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride for oil and gas drilling, extraction, and support activities presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the

risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.

- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride for oil and gas drilling, extraction, and support activities.

5.2.1.38 Industrial/Commercial Use – Other uses – Toys, playground, and sporting equipment – including novelty articles (Toys, playground and sporting equipment)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as other uses for toys, playground and sporting equipment: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in toys, playground and sporting equipment presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.

- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in toys, playground and sporting equipment.

5.2.1.39 Industrial/Commercial Use – Other uses – Lithographic printing cleaner (Lithographic printing plate cleaner)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride as a lithographic printing plate cleaner: Presents unreasonable risk of injury to health (workers); does not present an unreasonable risk of injury to health (ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from chronic (liver) inhalation exposure at the central tendency and high end, and non-cancer effects from acute (CNS) inhalation and cancer from chronic inhalation at the high-end, without assuming use of respirators. For ONUs, EPA found that there was no an unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver), or of cancer from chronic inhalation at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in lithographic printing plate cleaner presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene

chloride, the exposures for the condition of use, and the uncertainties in the analysis including uncertainties related to the exposures for ONUs:

- EPA does not assume workers use any type of respirator during industrial and commercial use of methylene chloride in lithographic printing.
- For workers, when assuming use of gloves with PF of 10, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk. The risk estimates at the central tendency of non-cancer effects from chronic inhalation exposures approximate the benchmark and do not support an unreasonable risk determination.
- Inhalation exposures were assessed using data primarily from a 1985 EPA assessment. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers) from industrial and commercial use of methylene chloride in lithographic printing plate cleaner.

5.2.1.40 Industrial/Commercial Use – Other uses – Carbon remover, wood floor cleaner, brush cleaner (Carbon remover, wood floor cleaner and brush cleaner)

Section 6(b)(4)(A) unreasonable risk determination for industrial and commercial use of methylene chloride for carbon remover, wood floor cleaner and brush cleaner: Presents an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the high-end, even when assuming use of PPE. For ONUs, EPA found that there was unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures at the central tendency.

EPA's determination that the industrial and commercial use of methylene chloride in carbon remover, wood floor cleaner, and brush cleaner presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of

methylene chloride, the exposures for the condition of use and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 50, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end support an unreasonable risk determination.
- For workers, when assuming use of respirators with APF of 25, the risk estimates of cancer from chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5, 10 and 20 the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- For workers, the risk estimates of cancer from chronic dermal exposures do not support an unreasonable risk determination.
- For ONUs, the risk estimates of cancer from chronic inhalation exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using monitoring data compiled by EPA from miscellaneous industrial and commercial settings. Uncertainties in the analysis include the representativeness of the monitoring data toward the true distribution of inhalation concentrations for the industries and sites covered by this condition of use.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (workers and ONUs) from industrial and commercial use of methylene chloride in carbon remover, wood floor cleaner, and brush cleaner.

5.2.1.41 Consumer Use – Solvents (for cleaning or degreasing) - Aerosol spray degreaser/cleaner (Solvent in Aerosol degreasers/cleaners)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride as solvent in aerosol degreasers/cleaners: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures and dermal exposures at the low, medium, and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride as solvent in aerosol degreasers/cleaners presents an unreasonable risk is based on the comparison of the risk

estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride as solvent in aerosol degreaser/cleaner were based on modeled risk estimates of seven products: brake cleaner, carburetor cleaner, engine cleaner, gasket remover, carbon remover, coil cleaner, and electronics cleaner.
- Inhalation exposures to consumers and bystanders were evaluated by three products modeled with 27 scenarios, three products modeled with 18 scenarios, and one product modeled with nine scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data for six products and absorption modeled data for one product. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride as solvent in aerosol degreasers/cleaners.

5.2.1.42 Consumer Use – Adhesives and sealants – Single component glues and adhesives and sealants and caulks (Adhesives and sealants)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in adhesives and sealants: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride in adhesives and sealants presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in adhesive and sealants were based on modeled risk estimates of two products: adhesives and sealants.

- Inhalation exposures to consumers and bystanders were evaluated by two products, one modeled with 27 scenarios and one modeled with 18 scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using absorption modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in adhesives and sealants.

5.2.1.43 Consumer Use – Paints and coatings– Paints and coatings (Brush Cleaners for paints and coatings)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in brush cleaners for paints and coatings: Present unreasonable risk of injury to health (consumers); does not present unreasonable risk of injury to health (bystanders)

For consumers, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute dermal exposures at the high intensity use. For bystanders, EPA found that there was no unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures.

EPA's determination that the consumer use of methylene chloride in brush cleaners for paints and coatings presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in brush cleaners for paints and coatings were based on modeled risk estimates of one product: brush cleaner.
- Risk estimates of non-cancer effects from acute inhalation exposures do not support an unreasonable risk determination.
- Inhalation exposures to consumers and bystanders were evaluated with nine different scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data. The magnitude of dermal exposures depends on several

factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers) from the consumer use of methylene chloride in brush cleaners for paints and coatings.

5.2.1.44 Consumer Use – Paints and coatings - Adhesive/caulk remover (Adhesive and caulk removers)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in adhesive and caulk removers: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use, and dermal exposures at the low, medium, and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at high intensity use.

EPA's determination that the consumer use of methylene chloride in adhesive and caulk removers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in adhesive and caulk removers were based on modeled risk estimates of one product: adhesive remover.
- Inhalation exposures to consumers and bystanders were evaluated with 18 different scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk for the consumer use of methylene chloride in adhesive and caulk removers.

5.2.1.45 Consumer Use – Metal products not covered elsewhere - Degreasers – aerosol and non-aerosol degreasers (Metal degreasers)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in metal degreasers: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures and dermal exposures at the low, medium, and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride in metal degreasers presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride as metal degreasers were based on modeled risk estimates of three products: carbon remover, coil cleaner, electronics cleaner.
- Inhalation exposures to consumers and bystanders were evaluated by two products modeled with 18 scenarios and one product modeled with nine scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data for two products and absorption modeled data for one product. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in metal degreasers.

5.2.1.46 Consumer Use – Automotive care products - Functional fluids for air conditioners: refrigerant, treatment, leak sealer (Automotive care products (functional fluids for air conditioners))

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in automotive care products (functional fluids for air conditioners): Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use, and dermal exposures at the low, medium, and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the high intensity use.

EPA's determination that the consumer use of methylene chloride in automotive care products (functional fluids for air conditioners) presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in automotive care products functional fluids for air conditioners were based on modeled risk estimates of two products: automotive AC leak sealer and automotive AC refrigerant.
- Inhalation exposures to consumers and bystanders were evaluated by two products, one modeled with 18 scenarios and one modeled with three scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using absorption modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in automotive care products (functional fluids for air conditioners).

5.2.1.47 Consumer Use – Automotive care products - Degreasers: gasket remover, transmission cleaners, carburetor (Automotive care products (degreasers))

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in automotive care products (degreasers): Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the low, medium, and high intensity use, and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride in automotive care products (degreasers) presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in automotive care products for degreasers were based on modeled risk estimates of four products: brake cleaner, carburetor cleaner, engine cleaner, gasket remover.
- Inhalation exposures to consumers and bystanders were evaluated by three products modeled with 27 scenarios and one product modeled with 18 scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in automotive care products (degreasers).

5.2.1.48 Consumer Use – Lubricants and greases – Liquid and spray lubricants and greases; degreasers – Aerosol and non-aerosol degreasers and cleaners (Lubricants and greases)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in lubricants and greases: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the low, medium, and high intensity use, and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride in lubricants and greases presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in lubricants and greases were modeled for four products: brake cleaner, carburetor cleaner, engine cleaner, gasket remover.
- Inhalation exposures to consumers and bystanders were evaluated by three products modeled with 27 scenarios and one product modeled with 18 scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in lubricants and greases.

5.2.1.49 Consumer Use – Building/ construction materials not covered elsewhere – Cold pipe insulation (Cold pipe insulation)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in cold pipe insulation: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute inhalation at the low, medium, and high intensity use and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the medium and high intensity use.

EPA's determination that the consumer use of methylene in cold pipe insulation presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in cold pipe insulation were based on modeled risk estimates of one product: cold pipe insulation spray.
- Inhalation exposures to consumers and bystanders were evaluated with 18 different scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.

- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using absorption modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in cold pipe insulation.

5.2.1.50 Consumer Use – Arts, crafts and hobby materials - Crafting glue and cement/concrete (Arts, crafts and hobby materials glue)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in arts, crafts, and hobby materials glue: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the medium and high intensity use and dermal exposure at medium and high intensity use. For bystanders, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute inhalation exposures at the high intensity use.

EPA's determination that the consumer use of methylene chloride in arts, crafts, and hobby materials glue presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in arts, crafts and hobby materials glue were based on modeled risk estimates of one product: adhesives.
- Inhalation exposures to consumers and bystanders were evaluated with 18 different scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using absorption modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of

injury to health (consumers and bystanders) from the consumer use of methylene chloride in arts, crafts and hobby materials glue.

5.2.1.51 Consumer Use – Other Uses - Anti-adhesive agent - anti-spatter welding aerosol (Anti-spatter welding aerosol)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in an anti-spatter welding aerosol: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation at the low, medium, and high intensity use, and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the medium and high intensity use.

EPA's determination that the consumer use of methylene chloride in an anti-spatter welding aerosol presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in an anti-spatter welding aerosol were based on modeled risk estimates of one product: weld spatter protectant.
- Inhalation exposures to consumers and bystanders were evaluated with nine different scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using absorption modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in an anti-spatter welding aerosol.

5.2.1.52 Consumer Use – Other Uses – Carbon Remover and brush cleaner (Carbon remover and other brush cleaner)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of methylene chloride in carbon removers and other brush cleaners: Presents an unreasonable risk of injury to health (consumers and bystanders).

For consumers, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation at the low, medium, and high intensity use, and dermal exposures at the medium and high intensity use. For bystanders, EPA found that there was an unreasonable risk of non-cancer effects (CNS) from acute inhalation exposure at the medium and high intensity use.

EPA's determination that the use of methylene chloride in carbon removers and other brush cleaners presents an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects to the benchmarks (Table 4-3) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis:

- Risk estimates for the consumer use of methylene chloride in carbon removers and other brush cleaners were based on modeled risk estimates of two products: carbon remover and brush cleaner.
- Inhalation exposures to consumers and bystanders were evaluated by two products modeled with 18 scenarios and one product modeled with nine scenarios. The magnitude of inhalation exposures depends on several factors, including the concentration of methylene chloride in products used, use patterns (including frequency, duration, amount of product used, room of use, and local ventilation), and application methods.
- Consumer dermal exposures result from direct contact with the product or from vapor or mist deposition onto the skin while using the product. Dermal exposures were assessed using permeability modeled data. The magnitude of dermal exposures depends on several factors, including skin surface area, product volume, concentration of methylene chloride in product used, and dermal exposure duration. The potential for dermal permeation of methylene chloride is limited by physical-chemical properties of methylene chloride.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is unreasonable risk of injury to health (consumers and bystanders) from the consumer use of methylene chloride in carbon removers and other brush cleaners.

5.2.1.53 Disposal – Disposal – Industrial pre-treatment; industrial wastewater treatment; publicly owned treatment works (POTW); underground injection; municipal landfill; hazardous landfill; other land disposal; municipal waste incinerator; hazardous waste incinerator; off-site waste transfer (Disposal)

Section 6(b)(4)(A) unreasonable risk determination for disposal of methylene chloride: Does not present an unreasonable risk of injury to health (workers and ONUs).

For workers, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation and dermal exposures at the central tendency and high-end, when assuming use of PPE. In addition, for workers, EPA found that there was no unreasonable risk of cancer from chronic inhalation and dermal exposures at the central tendency and high-end, without assuming use of PPE. For ONUs, EPA found that there was no unreasonable risk of non-cancer effects from acute (CNS) and chronic (liver) inhalation exposures and of cancer from chronic inhalation exposures at the central tendency.

EPA's determination that the disposal of methylene chloride does not present an unreasonable risk is based on the comparison of the risk estimates for non-cancer effects and cancer to the benchmarks (Table 4-2) and other considerations. As explained in Section 5.1., EPA considered the health effects of methylene chloride, the exposures for the condition of use, and the uncertainties in the analysis, including uncertainties related to the exposures for ONUs:

- For workers, when assuming use of respirators with APF of 25, the risk estimates of non-cancer effects from acute and chronic inhalation exposures at the high-end do not support an unreasonable risk determination. Similarly, when assuming use of gloves with PF of 5 and 20, the risk estimates of non-cancer effects from acute and chronic dermal exposures do not support an unreasonable risk determination.
- Based on EPA's analysis, the data for worker and ONU inhalation exposures could not be distinguished; however, ONU inhalation exposures are assumed to be lower than inhalation exposures for workers directly handling the chemical substance. To account for this uncertainty, EPA considered the workers' central tendency risk estimates from inhalation exposures when determining ONUs' unreasonable risk.
- Inhalation exposures were assessed using personal breathing zone monitoring data provided by two sources. The data may not be representative of exposures across the range of disposal facilities.
- Dermal exposures were assessed using modeled data.

In summary, the risk estimates, the health effects of methylene chloride, the exposures, and consideration of uncertainties support EPA's determination that there is no unreasonable risk of injury to health (workers and ONUs) from the disposal of methylene chloride.

5.2.2 Environment

Section 6(b)(4)(A) unreasonable risk determination for **all conditions** of use of methylene chloride: Does not present an unreasonable risk of injury **to the environment** (aquatic, sediment dwelling and terrestrial organisms).

For all conditions of use, the RQ values (Table 4-4 and 4-5) do not support an unreasonable risk determination in water for acute and chronic exposures to methylene chloride for amphibians, fish, and aquatic invertebrates. To characterize the exposure to methylene chloride by aquatic organisms, modeled data were used to represent surface water concentrations near facilities actively releasing methylene chloride to surface water, and monitored concentrations were used to represent ambient water concentrations of methylene chloride. EPA considered the biological relevance of the species to determine the concentrations of concern for the location of surface

water concentration data to produce RQs, as well as frequency and duration of the exposure. Some site-specific RQs, calculated from modeled release data from facilities conducting recycling, disposal, and waste water treatment plant activities are greater than or equal to one. Uncertainties related to these particular estimates are discussed in section 4.2.2. Uncertainties in the analysis include limitations in data, since monitoring data were not available near facilities where methylene chloride is released, and TRI does not capture release data for facilities with fewer than ten employees. As an additional uncertainty, the model does not consider chemical fate or hydrologic transport properties and may not consider dilution in static water bodies. As described in section 4.4.6, additional analysis indicated that model outputs, rather than monitoring estimates, may best represent concentrations found at the point of discharge from the facilities.

The toxicity of methylene chloride to sediment-dwelling invertebrates is similar to the toxicity to aquatic invertebrates. Methylene chloride is most likely present in the pore waters and not absorbed to the sediment organic matter because methylene chloride has low partitioning to organic matter. The concentrations in sediment pore water are similar to or less than the concentrations in the overlying water, and concentrations in the deeper part of sediment are lower than the concentrations in the overlying water. Therefore, for sediment dwelling organisms the risk estimates, based on the highest ambient surface water concentration, do not support an unreasonable risk determination to sediment-dwelling organisms from acute or chronic exposures. There is uncertainty due to the lack of ecotoxicity studies specifically for sediment-dwelling organisms and limited sediment monitoring data

Based on its physical-chemical properties, methylene chloride does not partition to or accumulate in soil. Therefore, the physical chemical properties of methylene chloride do not support an unreasonable risk determination to terrestrial organisms.

5.3 Changes to the Unreasonable Risk Determination from Draft Risk Evaluation to Final Risk Evaluation

In this final risk evaluation, EPA made changes to the unreasonable risk determination for methylene chloride following the publication of the draft risk evaluation, as a result of the analysis following peer review and public comments. There are two changes: removal of the industrial and commercial use of methylene chloride for functional fluids in pharmaceutical and medicine manufacturing, because it is not a condition of use under TSCA; and, for consumer uses, clearer unreasonable risk determinations for conditions of use evaluated with multiple exposure scenarios. Details of both these changes are below.

While use of methylene chloride as a functional fluid in a closed system during pharmaceutical manufacturing was included in the problem formulation and draft risk evaluation, upon further analysis of the details of this process, EPA has determined that this use falls outside TSCA's definition of "chemical substance." Under TSCA § 3(2)(B)(vi), the definition of "chemical substance" does not include any food, food additive, drug, cosmetic, or device (as such terms are defined in section 201 of the Federal Food, Drug, and Cosmetic Act) when manufactured, processed, or distributed in commerce for use as a food, food additive, drug, cosmetic, or

device. EPA has found that methylene chloride use as a functional fluid in a closed system during pharmaceutical manufacturing entails use as an extraction solvent in the purification of pharmaceutical products, and has concluded that this use falls within the aforementioned definitional exclusion and is not a “chemical substance” under TSCA.

EPA uses representative Occupational Exposure Scenarios and Consumer Exposure Scenarios to generate risk estimates. Sometimes the same Exposure Scenario is used for several conditions of use, and sometimes unreasonable risk determinations are based on multiple exposure scenarios. EPA makes an unreasonable risk determination for each condition of use in the Problem Formulation. For consumer uses, in some instances more than one Consumer Exposure Scenario (e.g., consumer use as solvent in aerosol degreasers/cleaners has seven) is an appropriate representative for a consumer condition of use. Earlier, in the Draft Risk Evaluation, EPA assigned each Consumer Exposure Scenario to a condition of use, which, in some cases, resulted in multiple preliminary unreasonable risk determinations for a single condition of use (e.g., consumer use in metal degreasers had three unreasonable risk determinations). In this Final Risk Evaluation, EPA adheres to the conditions of use as they were presented in the Problem Formulation; as a result, in some cases a single determination may be informed by multiple risk estimates from multiple Consumer Exposure Scenarios. Therefore, whereas the draft Risk Evaluation presented 29 consumer risk determinations on 12 conditions of use, the Final Evaluation shows only the 12. Overall, the Draft Risk Evaluation had 71 unreasonable risk determinations, whereas the Final Risk Evaluation determination has 53 unreasonable risk determinations. The exposure scenarios supporting the unreasonable risk determinations for the conditions of use are listed in the detailed description of each consumer use and listed in Table 5-2.

Table 5-2. Crosswalk of Consumer Use Unreasonable Risk Determinations

Unreasonable Risk Determinations in Final Risk Evaluation	Unreasonable Risk Determinations in Draft Risk Evaluation
<ul style="list-style-type: none"> • Consumer use as solvent in aerosol degreasers/cleaners 	<ul style="list-style-type: none"> • As a solvent in an aerosol spray degreaser/cleaner (brake cleaner) • As a solvent in an aerosol spray degreaser/cleaner (carbon remover) • Consumer use as a solvent in an aerosol spray degreaser/cleaner (carburetor cleaner) • As a solvent in an aerosol spray degreaser/cleaner (coil cleaner) • As a solvent in an aerosol spray degreaser/cleaner (electronics cleaner) • As a solvent in an aerosol spray degreaser/cleaner (engine cleaner) • As a solvent in an aerosol spray degreaser/cleaner (gasket remover)
<ul style="list-style-type: none"> • Consumer use in adhesives and sealants 	<ul style="list-style-type: none"> • As an adhesive and sealant for single component glues and adhesives and sealants and caulks (adhesives) • As an adhesive and sealant for single component glues and adhesives and sealants and caulks (sealants)
<ul style="list-style-type: none"> • Consumer use in brush cleaners for paints and coatings 	<ul style="list-style-type: none"> • Consumer use as a brush cleaner for paints and coatings
<ul style="list-style-type: none"> • Consumer use in adhesive and caulk removers 	<ul style="list-style-type: none"> • As an adhesive/caulk remover
<ul style="list-style-type: none"> • Consumer use in metal degreasers 	<ul style="list-style-type: none"> • As a metal product not covered elsewhere in aerosol and non-aerosol degreasers (carbon remover) • As a metal product not covered elsewhere in aerosol and non-aerosol degreasers (coil cleaner)

Unreasonable Risk Determinations in Final Risk Evaluation	Unreasonable Risk Determinations in Draft Risk Evaluation
	<ul style="list-style-type: none"> As a metal product not covered elsewhere in aerosol and non-aerosol degreaser (electronics cleaner)
<ul style="list-style-type: none"> Consumer use in automotive care products (functional fluids for air conditioners) 	<ul style="list-style-type: none"> As an automotive care product for functional fluids for air conditioners: refrigerant, treatment, leak sealer (automotive air conditioning leak sealer) As an automotive care product for functional fluids for air conditioners: refrigerant, treatment, leak sealer (automotive air conditioning refrigerant)
<ul style="list-style-type: none"> Consumer use in automotive care products (degreasers) 	<ul style="list-style-type: none"> As an automotive care product in degreasers (brake cleaner) As an automotive care product in degreasers (carburetor cleaner) As an automotive care product in degreasers (engine cleaner) As an automotive care product in degreasers (gasket remover)
<ul style="list-style-type: none"> Consumer use in lubricants and greases 	<ul style="list-style-type: none"> As a lubricant and grease in degreasers (brake cleaner) As a lubricant and grease in degreasers (carburetor cleaner) As a lubricant and grease in degreasers (engine cleaner) As a lubricant and grease in degreasers (gasket remover)
<ul style="list-style-type: none"> Consumer use in cold pipe insulation 	<ul style="list-style-type: none"> As a building construction material not covered elsewhere for cold pipe insulation
<ul style="list-style-type: none"> Consumer use in arts, crafts, and hobby materials glue 	<ul style="list-style-type: none"> As an arts, crafts, and hobby materials for crafting glue and cement/concrete
<ul style="list-style-type: none"> Consumer use in an anti-spatter welding aerosol 	<ul style="list-style-type: none"> As other uses for anti-adhesive agent – anti-spatter welding aerosol
<ul style="list-style-type: none"> Consumer use in carbon removers and other brush cleaners 	<ul style="list-style-type: none"> Consumer use as a brush cleaner for other uses As other uses for carbon remover

5.4 Unreasonable Risk Determination Conclusion

5.4.1 5.4.1 No Unreasonable Risk Determinations

TSCA section 6(b)(4) requires EPA to conduct risk evaluations to determine whether chemical substances present unreasonable risk under their conditions of use. In conducting risk evaluations, “EPA will determine whether the chemical substance presents an unreasonable risk of injury to health or the environment under each condition of use within the scope of the risk evaluation...” 40 CFR 702.47. Pursuant to TSCA section 6(i)(1), a determination of “no unreasonable risk” shall be issued by order and considered to be final agency action.

EPA has determined that the following conditions of use of methylene chloride do not present an unreasonable risk of injury to health or the environment:

- Manufacturing (Domestic Manufacture) (Section 5.2.1.1, Section 5.2.2, Section 4, Section 3, and Section 2.4.1.2.1)

- Processing: as a reactant (Section 5.2.1.3, Section 5.2.2, Section 4, Section 3, and Section 2.4.1.2.2)
- Processing: recycling (Section 5.2.1.6, Section 5.2.2, Section 4, Section 3, and Section 2.4.1.2.2)
- Distribution in commerce (Section 5.2.1.7, Section 5.2.2, Section 4, Section 3)
- Industrial and commercial use as laboratory chemical (Section 5.2.1.32, Section 5.2.2, Section 4, Section 3, and Section 2.4.1.2.16)
- Disposal (Section 5.2.1.53, Section 5.2.2, Section 4, Section 3, and Section 2.4.1.2.21)

This subsection of the final risk evaluation therefore constitutes the order required under TSCA section 6(i)(1), and the “no unreasonable risk” determinations in this subsection are considered to be final agency action effective on the date of issuance of this order. All assumptions that went into reaching the determinations of no unreasonable risk for these conditions of use, including any considerations excluded for these conditions of use, are incorporated into this order.

The support for each determination of “no unreasonable risk” is set forth in Section 5.2 of the final risk evaluation, “Detailed Unreasonable Risk Determinations by Condition of Use.” This subsection also constitutes the statement of basis and purpose required by TSCA section 26(f).

5.4.2 Unreasonable Risk Determinations

EPA has determined that the following conditions of use of methylene chloride present an unreasonable risk of injury to health but do not present unreasonable risk of injury to the environment:

- Manufacturing (Import)
- Processing: incorporation into a formulation, mixture, or reaction products
- Processing: repackaging
- Industrial and commercial use as solvent for batch vapor degreasing
- Industrial and commercial use as solvent for in-line vapor degreasing
- Industrial and commercial use as solvent for cold cleaning
- Industrial and commercial use as solvent for aerosol spray degreaser/cleaner
- Industrial and commercial use in adhesives, sealants and caulks
- Industrial and commercial use in paints and coatings
- Industrial and commercial use in paint and coating removers

- Industrial and commercial use in adhesive and caulk removers
- Industrial and commercial use in metal aerosol degreasers
- Industrial and commercial use in metal non-aerosol degreasers
- Industrial and commercial use in finishing products for fabric, textiles and leather
- Industrial and commercial use in automotive care products (functional fluids for air conditioners)
- Industrial and commercial use in automotive care products (interior car care)
- Industrial and commercial use in automotive care products (degreasers)
- Industrial and commercial use in apparel and footwear care products
- Industrial and commercial use in spot removers for apparel and textiles
- Industrial and commercial use in liquid lubricants and greases
- Industrial and commercial use in spray lubricants and greases
- Industrial and commercial use in aerosol degreasers and cleaners
- Industrial and commercial use in non-aerosol degreasers and cleaners
- Industrial and commercial use in cold pipe insulations
- Industrial and commercial use as solvent that becomes part of a formulation or mixture
- Industrial and commercial use as a processing aid
- Industrial and commercial use as propellant and blowing agent
- Industrial and commercial use for electrical equipment, appliance, and component manufacturing
- Industrial and commercial use for plastic and rubber products manufacturing
- Industrial and commercial use in cellulose triacetate film production
- Industrial and commercial use as anti-spatter welding aerosol
- Industrial and commercial use for oil and gas drilling, extraction, and support activities
- Industrial and commercial use in toys, playground and sporting equipment
- Industrial and commercial use in lithographic printing plate cleaner
- Industrial and commercial use in carbon remover, wood floor cleaner, and brush cleaner
- Consumer use as solvent in aerosol degreasers/cleaners
- Consumer use in adhesives and sealants

- Consumer use in brush cleaners for paints and coatings
- Consumer use adhesive and caulk removers
- Consumer use in metal degreasers
- Consumer use in automotive care products (functional fluids for air conditioners)
- Consumer use in automotive care products (degreasers)
- Consumer use in lubricants and greases
- Consumer use in cold pipe insulation
- Consumer use in arts, crafts, and hobby materials glue
- Consumer use in an anti-spatter welding aerosol
- Consumer use in carbon removers and other brush cleaners

EPA will initiate TSCA section 6(a) risk management actions on these conditions of use as required under TSCA section 6(c)(1). Pursuant to TSCA section 6(i)(2), the “unreasonable risk” determinations for these conditions of use are not considered final agency action.

REFERENCES

- . (2012a). Fatality Assessment and Control Evaluation (FACE) Report for California: A Maintenance Worker Dies from Exposure to Dichloromethane (Methylene Chloride) While Stripping the Floor of a Baptismal Font in a Church, FACE-12-CA-002. GRA and I: 8.
- . (2012b). Fatality Assessment and Control Evaluation (FACE) Report for Iowa: Bathtub Refinishing Technician Died from Inhalation of Paint Stripper Vapors (pp. 15). (NTIS/13520087).
- (IPCS), IPoCS. (1996). Environmental Health Criteria 164. Methylene Chloride Second Edition. Geneva, Switzerland: International Programme on Chemical Safety (IPCS).
<http://www.inchem.org/documents/ehc/ehc/ehc164.htm>
- (NIOSH), NIOSaH. (2002a). In-depth survey report: control of perchloroethylene (PCE) in vapor degreasing operations, site #1. (EPHB 256-19b). Cincinnati, Ohio: National Institute for Occupational Safety and Health (NIOSH).
- (NIOSH), NIOSaH. (2002b). In-depth survey report: control of perchloroethylene (PCE) in vapor degreasing operations, site #4. (EPHB 256-18b). Cincinnati, Ohio: National Institute for Occupational Safety and Health (NIOSH).
- Abernethy, S; Bobra, AM; Shiu, WY; Wells, PG; Mackay, D. (1986). Acute lethal toxicity of hydrocarbons and chlorinated hydrocarbons to two planktonic crustaceans the key role of organism-water partitioning. *Aquat Toxicol* AMST: 163-174.
- Adgate, JL; Church, TR; Ryan, AD; Ramachandran, G; Fredrickson, AL; Stock, TH; Morandi, MT; Sexton, K. (2004). Outdoor, indoor, and personal exposure to VOCs in children. *Environ Health Perspect* 112: 1386-1392. <http://dx.doi.org/10.1289/ehp.7107>
- Ahrenholz, SH. (1980). Health hazard evaluation report no. HHE 80-18-691, Looart Press Incorporate, Colorado Springs, Colorado. (HHE 80-18-691). Cincinnati, OH: National Institute for Occupational Safety and Health.
- AISE. (2012). AISE SPERC fact sheet - wide dispersive use of cleaning and maintenance products. International Association for Soaps Detergents and Maintenance Products. <https://www.aise.eu/our-activities/regulatory-context/reach/environmental-exposure-assessment.aspx>
- Aiso, S; Take, M; Kasai, T; Senoh, H; Umeda, Y; Matsumoto, M; Fukushima, S. (2014a). Inhalation carcinogenicity of dichloromethane in rats and mice. *Inhal Toxicol* 26: 435-451.
https://heronet.epa.gov/heronet/index.cfm/reference/download/reference_id/4238148
- Aiso, S; Take, M; Kasai, T; Senoh, H; Umeda, Y; Matsumoto, M; Fukushima, S. (2014b). Supplement: Inhalation carcinogenicity of dichloromethane in rats and mice [Supplemental Data]. *Inhal Toxicol* 26: 435-451.
- Alexander, HC; Mccarty, WM; Bartlett, EA. (1978). Toxicity of perchloroethylene, trichloroethylene, 1,1,1-trichloroethane, and methylene chloride to fathead minnows. *Bull Environ Contam Toxicol* 20: 344-352. <http://dx.doi.org/10.1007/BF01683531>
- Alexeeff, GV; Kilgore, WW. (1983). Learning impairment in mice following acute exposure to dichloromethane and carbon tetrachloride. *J Toxicol Environ Health* 11: 569-581.
<http://dx.doi.org/10.1080/15287398309530368>

- Allen, J; Kligerman, A; Campbell, J; Westbrook-Collins, B; Erexson, G; Kari, F; Zeiger, E. (1990). Cytogenetic analyses of mice exposed to dichloromethane. *Environ Mol Mutagen* 15: 221-228. <http://dx.doi.org/10.1002/em.2850150409>
- Allred, EN; Bleecker, ER; Chaitman, BR; Dahms, TE; Gottlieb, SO; Hackney, JD; Hayes, D; Pagano, M; Selvester, RH; Walden, SM; Warren, J. (1989a). Acute effects of carbon monoxide exposure on individuals with coronary artery disease (pp. 1-79). (ISSN 1041-5505). Boston, MA: Health Effects Institute.
- Allred, EN; Bleecker, ER; Chaitman, BR; Dahms, TE; Gottlieb, SO; Hackney, JD; Pagano, M; Selvester, RH; Walden, SM; Warren, J. (1989b). Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Engl J Med* 321: 1426-1432. <http://dx.doi.org/10.1056/NEJM198911233212102>
- Allred, EN; Bleecker, ER; Chaitman, BR; Dahms, TE; Gottlieb, SO; Hackney, JD; Pagano, M; Selvester, RH; Walden, SM; Warren, J. (1991). Effects of carbon monoxide on myocardial ischemia. *Environ Health Perspect* 91: 89-132. <http://dx.doi.org/10.1289/ehp.919189>
- Andersen, ME; Black, MB; Campbell, JL; Pendse, SN; Clewell, HJ; Pottenger, LH; Bus, JS; Dodd, DE; Kemp, DC; McMullen, PD. (2017). Combining transcriptomics and PBPK modeling indicates a primary role of hypoxia and altered circadian signaling in dichloromethane carcinogenicity in mouse lung and liver. *Toxicol Appl Pharmacol* 332: 149-158. <http://dx.doi.org/10.1016/j.taap.2017.04.002>
- Andersen, ME; Clewell, HJ, III; Gargas, ML; Macnaughton, MG; Reitz, RH; Nolan, RJ; Mckenna, MJ. (1991). Physiologically based pharmacokinetic modeling with dichloromethane, its metabolite, carbon monoxide, and blood carboxyhemoglobin in rats and humans. *Toxicol Appl Pharmacol* 108: 14-27.
- Anderson, EW; Andelman, RJ; Strauch, JM; Fortuin, NJ; Knelson, JH. (1973). Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris: a study in ten patients with ischemic heart disease. *Ann Intern Med* 79: 46-50. <http://dx.doi.org/10.7326/0003-4819-79-1-46>
- Ando, T; Otsuka, S; Nishiyama, M; Senoo, K; Watanabe, MM; Matsumoto, S. (2003). Toxic Effects of Dichloromethane and Trichloroethylene on the Growth of Planktonic Green Algae, *Chlorella vulgaris* NIES227, *Selenastrum capricornutum* NIES35, and *Volvox steinii* NIES545. *18*: 43-46.
- Anundi, H; Lind, ML; Friis, L; Itkes, N; Langworth, S; Edling, C. (1993). High exposures to organic solvents among graffiti removers. *Int Arch Occup Environ Health* 65: 247-251. <http://dx.doi.org/10.1007/BF00381198>
- Aranyi, C; O'Shea, WJ; Graham, JA; Miller, FJ. (1986). The effects of inhalation of organic chemical air contaminants on murine lung host defenses. *Fundam Appl Toxicol* 6: 713-720. [http://dx.doi.org/10.1016/0272-0590\(86\)90184-3](http://dx.doi.org/10.1016/0272-0590(86)90184-3)
- Aronow, WS; Harris, CN; Isbell, MW; Rokaw, SN; Imparato, B. (1972). Effect of freeway travel on angina pectoris. *Ann Intern Med* 77: 669-676.
- Astrand, I; Ovrum, P; Carlsson, A. (1975). Exposure to methylene chloride: I. Its concentration in alveolar air and blood during rest and exercise and its metabolism. *Scand J Work Environ Health* 1: 78-94.
- ATSDR. (2000). Toxicological profile for methylene chloride [ATSDR Tox Profile]. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. <http://www.atsdr.cdc.gov/toxprofiles/tp14.pdf>

- ATSDR. (2010). Addendum to the toxicological profile for methylene chloride [ATSDR Tox Profile]. Atlanta, GA.
http://www.atsdr.cdc.gov/toxprofiles/methylene_chloride_addendum.pdf
- Aviado, DM; Zakhari, S; Watanabe, T. (1977). Methylene chloride. In DM Aviado (Ed.), (pp. 19-36). Cleveland, OH: CRC Press, Inc.
<https://www.taylorfrancis.com/books/9781351075015/chapters/10.1201/9781351075015-1>
- Bale, AS; Barone, S; Scott, CS; Cooper, GS. (2011). A review of potential neurotoxic mechanisms among three chlorinated organic solvents [Review]. *Toxicol Appl Pharmacol* 255: 113-126. <http://dx.doi.org/10.1016/j.taap.2011.05.008>
- Ballantyne, B; Gazzard, MF; Swanston, DW. (1976). The ophthalmic toxicology of dichloromethane. *Toxicology* 6: 173-187. [http://dx.doi.org/10.1016/0300-483X\(76\)90019-6](http://dx.doi.org/10.1016/0300-483X(76)90019-6)
- Barry, KH; Zhang, Y; Lan, Q; Zahm, SH; Holford, TR; Leaderer, B; Boyle, P; Hosgood, HD; Chanock, S; Yeager, M; Rothman, N; Zheng, T. (2011). Genetic variation in metabolic genes, occupational solvent exposure, and risk of non-hodgkin lymphoma. *Am J Epidemiol* 173: 404-413.
- Bell, BP; Franks, P; Hildreth, N; Melius, J. (1991). Methylene chloride exposure and birthweight in Monroe County, New York. *Environ Res* 55: 31-39.
- Benignus, VA; Bushnell, PJ; Boyes, WK. (2011). Estimated rate of fatal automobile accidents attributable to acute solvent exposure at low inhaled concentrations. *Risk Anal* 31: 1935-1948. <http://dx.doi.org/10.1111/j.1539-6924.2011.01622.x>
- Bianchi, E; Lessing, G; Brina, KR; Angeli, L; Andriguetti, NB; Peruzzo, J. R.; Do Nascimento, CA; Spilki, FR; Ziulkoski, AL; da Silva, LB. (2017). Monitoring the Genotoxic and Cytotoxic Potential and the Presence of Pesticides and Hydrocarbons in Water of the Sinos River Basin, Southern Brazil. *Arch Environ Contam Toxicol* 72: 321-334.
<http://dx.doi.org/10.1007/s00244-016-0334-0>
- Birge, WJ; Black, JA; Kuehne, RA. (1980). Effects of Organic Compounds on Amphibian Reproduction. 39 p. (NTIS PB80-147523).
- Black, JA; Birge, WJ; McDonnell, WE; Westerman, AG; Ramey, BA; Bruser, DM. (1982). The aquatic toxicity of organic compounds to embryo-larval stages of fish and amphibians. (Research Report No. 133). Lexington, KY: University of Kentucky.
- Bornschein, RL; Hastings, L; Manson, JM. (1980). Behavioral toxicity in the offspring of rats following maternal exposure to dichloromethane. *Toxicol Appl Pharmacol* 52: 29-37.
[http://dx.doi.org/10.1016/0041-008X\(80\)90244-6](http://dx.doi.org/10.1016/0041-008X(80)90244-6)
- Bos, PM; Zeilmaker, MJ; van Eijkeren, JC. (2006). Application of physiologically based pharmacokinetic modeling in setting acute exposure guideline levels for methylene chloride. *Toxicol Sci* 91: 576-585. <http://dx.doi.org/10.1093/toxsci/kfj176>
- Boublík, T; Fried, V; Hála, E. (1984). The vapour pressures of pure substances: Selected values of the temperature dependence of the vapour pressures of some pure substances in the normal and low pressure region (2nd Revised ed.). Amsterdam, The Netherlands: Elsevier Science Publishers.
- Brack, W; Rottler, H. (1994). Toxicity testing of highly volatile chemicals with green algae: A new assay. 1: 223-228.

- Braus-Stromeyer, SA; Hermann, R; Cook, AM; Leisinger, T. (1993). Dichloromethane as the sole carbon source for an acetogenic mixed culture and isolation of a fermentative, dichloromethane-degrading bacterium. *Appl Environ Microbiol* 59: 3790-3797.
- Brender, JD; Shinde, MU; Zhan, FB; Gong, X; Langlois, PH. (2014). Maternal residential proximity to chlorinated solvent emissions and birth defects in offspring: a case-control study. *Environ Health* 13: 96. <http://dx.doi.org/10.1186/1476-069X-13-96>
- Buccafusco, RJ; Ells, SJ; LeBlanc, GA. (1981). Acute toxicity of priority pollutants to bluegill (*Lepomis macrochirus*). *Bull Environ Contam Toxicol* 26: 446-452. <http://dx.doi.org/10.1007/BF01622118>
- Burek, JD; Nitschke, KD; Bell, TJ; Wackerle, DL; Childs, RC; Beyer, JE; Dittenber, DA; Rampy, LW; McKenna, MJ. (1984). Methylene chloride: A two-year inhalation toxicity and oncogenicity study in rats and hamsters. *Fundam Appl Toxicol* 4: 30-47. <http://dx.doi.org/10.1093/toxsci/4.1.30>
- Callen, DF; Wolf, CR; Philpot, RM. (1980). Cytochrome P-450 mediated genetic activity and cytotoxicity of seven halogenated aliphatic hydrocarbons in *Saccharomyces cerevisiae*. *Mutat Res* 77: 55-63. [http://dx.doi.org/10.1016/0165-1218\(80\)90120-2](http://dx.doi.org/10.1016/0165-1218(80)90120-2)
- Cantor, KP; Stewart, PA; Brinton, LA; Dosemeci, M. (1995). Occupational exposures and female breast cancer mortality in the United States. *J Occup Environ Med* 37: 336-348.
- CARB. (2000). Initial statement of reasons for the proposed airborne toxic control measure for emissions of chlorinated toxic air contaminants from automotive maintenance and repair activities.
- Carlsson, A; Hultengren, M. (1975). Exposure to methylene chloride: III metabolism of ¹⁴C-labelled methylene chloride in rat. *Scand J Work Environ Health* 1: 104-108.
- Carton, M; Barul, C; Menvielle, G; Cyr, D; Sanchez, M; Pilorget, C; Trétarre, B; Stücker, I; Luce, D; Group, IS. (2017). Occupational exposure to solvents and risk of head and neck cancer in women: a population-based case-control study in France. *BMJ Open* 7: e012833.
- Casanova, M; Bell, DA; Heck, H. (1997). Dichloromethane metabolism to formaldehyde and reaction of formaldehyde with nucleic acids in hepatocytes of rodents and humans with and without glutathione S-transferase T1 and M1 genes. *Fundam Appl Toxicol* 37: 168-180. <http://dx.doi.org/10.1093/toxsci/37.2.168>
- Casanova, M; Conolly, RB; Heck, H. (1996). DNA-protein cross-links (DPX) and cell proliferation in B6C3F1 mice but not Syrian golden hamsters exposed to dichloromethane: Pharmacokinetics and risk assessment with DPX as dosimeter. *Fundam Appl Toxicol* 31: 103-116. <http://dx.doi.org/10.1006/faat.1996.0081>
- Casanova, M; Deyo, DF; Heck, H. (1992). Dichloromethane (methylene chloride): metabolism to formaldehyde and formation of DNA-protein cross-links in B6C3F1 mice and Syrian golden hamsters [Letter]. *Toxicol Appl Pharmacol* 114: 162-165. [http://dx.doi.org/10.1016/0041-008X\(92\)90109-6](http://dx.doi.org/10.1016/0041-008X(92)90109-6)
- CDC. (2012). Fatal exposure to methylene chloride among bathtub refinishers - United States, 2000-2011. *MMWR Morb Mortal Wkly Rep* 61: 119-122.
- CDC. (2019). Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables, January 2019, Volume 1. Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey. https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Jan2019-508.pdf

- Chaigne, B; Lasfargues, G; Marie, I; Hüttenberger, B; Lavigne, C; Marchand-Adam, S; Maillot, F; Diot, E. (2015). Primary Sjögren's syndrome and occupational risk factors: A case-control study. *J Autoimmun* 60: 80-85.
- Chan, CC; Vainer, L; Martin, JW; Williams, DT. (1990). Determination of organic contaminants in residential indoor air using an adsorption-thermal desorption technique. *J Air Waste Manag Assoc* 40: 62-67.
- Chen, CY; Kao, CY; Lin, PJ; Shiesh, SC. (2013). Carbon monoxide may enhance bile secretion by increasing glutathione excretion and Mrp2 expression in rats. *J Chin Med Assoc* 76: 258-264. <http://dx.doi.org/10.1016/j.jcma.2013.02.001>
- Cherrie, JW; Semple, S; Brouwer, D. (2004). Gloves and Dermal Exposure to Chemicals: Proposals for Evaluating Workplace Effectiveness. *Ann Occup Hyg* 48: 607-615. <http://dx.doi.org/10.1093/annhyg/meh060>
- Cherry, N; Venables, H; Waldron, HA. (1983). The acute behavioural effects of solvent exposure. *Occup Med (Lond)* 33: 13-18.
- Chin, JY; Godwin, C; Parker, E; Robins, T; Lewis, T; Harbin, P; Batterman, S. (2014). Levels and sources of volatile organic compounds in homes of children with asthma. *Indoor Air* 24: 403-415. <http://dx.doi.org/10.1111/ina.12086>
- Christensen, KY; Vizcaya, D; Richardson, H; Lavoué, J; Aronson, K; Siemiatycki, J. (2013). Risk of selected cancers due to occupational exposure to chlorinated solvents in a case-control study in Montreal. *J Occup Environ Med* 55: 198-208.
- Christof, O; Seifert, R; Michaelis, W. (2002). Volatile halogenated organic compounds in European estuaries. *Biogeochemistry* 59: 143-160.
- Chung, CW; Morandi, MT; Stock, TH; Afshar, M. (1999). Evaluation of a passive sampler for volatile organic compounds at ppb concentrations, varying temperatures, and humidities with 24-h exposures. 2. Sampler performance. *Environ Sci Technol* 33: 3666-3671. <http://dx.doi.org/10.1021/es990613f>
- Clark, DG; Tinston, DJ. (1982). Acute inhalation toxicity of some halogenated and non-halogenated hydrocarbons. *Hum Exp Toxicol* 1: 239-247. <http://dx.doi.org/10.1177/096032718200100306>
- Cocco, P; Heineman, EF; Dosemeci, M. (1999). Occupational risk factors for cancer of the central nervous system (CNS) among US women. *Am J Ind Med* 36: 70-74.
- Cone Mills Corp. (1981a). HEALTH & SAFETY STUDY REPORT (EPA 40 CFR PART 716). (OTS: OTS0205907; 8EHQ Num: NA; DCN: 878210299; TSCATS RefID: 16553; CIS: NA).
- Cone Mills Corp. (1981b). Survey results of personal exposure monitoring with cover letter [TSCA Submission]. (OTS: OTS0205909; 8EHQ Num: NA; DCN: 878210294; TSCATS RefID: 16734; CIS: NA).
- Costantini, AS; Benvenuti, A; Vineis, P; Kriebel, D; Tumino, R; Ramazzotti, V; Rodella, S; Stagnaro, E; Crosignani, P; Amadori, D; Mirabelli, D; Sommani, L; Belletti, I; Troschel, L; Romeo, L; Miceli, G; Tozzi, G; Mendico, I; Maltoni, S; Miligi, L. (2008). Risk of leukemia and multiple myeloma associated with exposure to benzene and other organic solvents: Evidence from the Italian Multicenter Case-control study. *Am J Ind Med* 51: 803-811.
- Crebelli, R; Benigni, R; Franekic, J; Conti, G; Conti, L; Carere, A. (1988). Induction of chromosome malsegregation by halogenated organic solvents in *Aspergillus nidulans*:

- Unspecific or specific mechanism? *Mutat Res* 201: 401-411.
[http://dx.doi.org/10.1016/0027-5107\(88\)90027-9](http://dx.doi.org/10.1016/0027-5107(88)90027-9)
- Crebelli, R; Carere, A; Leopardi, P; Conti, L; Fassio, F; Raiteri, F; Barone, D; Ciliutti, P; Cinelli, S; Vericat, JA. (1999). Evaluation of 10 aliphatic halogenated hydrocarbons in the mouse bone marrow micronucleus test. *Mutagenesis* 14: 207-215.
<http://dx.doi.org/10.1093/mutage/14.2.207>
- David, RM; Clewell, HJ; Gentry, PR; Covington, TR; Morgott, DA; Marino, DJ. (2006). Revised assessment of cancer risk to dichloromethane II. Application of probabilistic methods to cancer risk determinations. *Regul Toxicol Pharmacol* 45: 55-65.
<http://dx.doi.org/10.1016/j.yrtph.2005.12.003>
- Defense Occupational and Environmental Health Readiness System - Industrial Hygiene (DOEHRS-IH). (2018). Email between DOD and EPA: RE: [Non-DoD Source] Update: DoD exposure data for EPA risk evaluation - EPA request for additional information. Washington, D.C.: U.S. Department of Defense.
- Dell, LD; Mundt, KA; Mcdonald, M; Tritschler, JP; Mundt, DJ. (1999). Critical review of the epidemiology literature on the potential cancer risks of methylene chloride [Review]. *Int Arch Occup Environ Health* 72: 429-442. <http://dx.doi.org/10.1007/s004200050396>
- Demarini, DM; Shelton, ML; Warren, SH; Ross, TM; Shim, JY; Richard, AM; Pegram, RA. (1997). Glutathione S-transferase-mediated induction of GC->AT transitions by halomethanes in Salmonella. *Environ Mol Mutagen* 30: 440-447.
[http://dx.doi.org/10.1002/\(SICI\)1098-2280\(1997\)30:4<440::AID-EM9>3.0.CO;2-M](http://dx.doi.org/10.1002/(SICI)1098-2280(1997)30:4<440::AID-EM9>3.0.CO;2-M)
- Devereux, TR; Foley, JF; Maronpot, RR; Kari, F; Anderson, MW. (1993). Ras proto-oncogene activation in liver and lung tumors from B6C3F1 mice exposed chronically to methylene chloride. *Carcinogenesis* 14: 795-801. <http://dx.doi.org/10.1093/carcin/14.5.795>
- Di Toro, DM. (1984). Probability Model of Stream Quality Due to Runoff. ASCE. *J Environ Eng* 110: 607-628.
- Dierickx, PJ. (1993). Comparison between fish lethality data and the in vitro cytotoxicity of lipophilic solvents to cultured fish cells in a two-compartment model. *Chemosphere* 27: 1511-1518.
- Dill, DC; Murphy, PG; Mayes, MA. (1987). Toxicity of methylene-chloride to life stages of the fathead minnow, *Pimephales promelas* Rafinesque. *Bull Environ Contam Toxicol* 39: 869-876. <http://dx.doi.org/10.1007/BF01855868>
- Dilling, WL; Tefertiller, NB; Kallos, GJ. (1975). Evaporation rates and reactivities of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. *Environ Sci Technol* 9: 833-838. <http://dx.doi.org/10.1021/es60107a008>
- Dillon, D; Edwards, I; Combes, R; Mcconville, M; Zeiger, E. (1992). The role of glutathione in the bacterial mutagenicity of vapour phase dichloromethane. *Environ Mol Mutagen* 20: 211-217. <http://dx.doi.org/10.1002/em.2850200310>
- Divincenzo, GD; Kaplan, CJ. (1981). Uptake, metabolism, and elimination of methylene chloride vapor by humans. *Toxicol Appl Pharmacol* 59: 130-140.
[http://dx.doi.org/10.1016/0041-008X\(81\)90460-9](http://dx.doi.org/10.1016/0041-008X(81)90460-9)
- Divincenzo, GD; Yanno, FJ; Astill, BD. (1972). Human and canine exposures to methylene chloride vapor. *Am Ind Hyg Assoc J* 33: 125-135.
<http://dx.doi.org/10.1080/0002889728506622>

- Dodson, RE; Levy, JI; Spengler, JD; Shine, JP; Bennett, DH. (2008). Influence of basements, garages, and common hallways on indoor residential volatile organic compound concentrations. *Atmos Environ* 42: 1569-1581.
<http://dx.doi.org/10.1016/j.atmosenv.2007.10.088>
- Doherty, AT; Ellard, S; Parry, EM; Parry, JM. (1996). An investigation into the activation and deactivation of chlorinated hydrocarbons to genotoxins in metabolically competent human cells. *Mutagenesis* 11: 247-274. <http://dx.doi.org/10.1093/mutage/11.3.247>
- Dosemeci, M; Cocco, P; Chow, WH. (1999). Gender differences in risk of renal cell carcinoma and occupational exposures to chlorinated aliphatic hydrocarbons. *Am J Ind Med* 36: 54-59.
- Dow Chem Co. (1988). INITIAL SUBMISSION: EVALUATION OF THE ACUTE NEUROPHARMACOLOGIC EFFECTS OF DICHLOROMETHANE IN RATS (FINAL REPORT) WITH ATTACHMENTS AND COVER LETTER DATED 050792. (OTS: OTS0537278; 8EHQ Num: 8EHQ-0592-3826; DCN: 88-920002468; TSCATS RefID: 423282; CIS: NA).
- Duclos, Y; Blanchard, M; Chesterikoff, A; Chevreuil, M. (2000). Impact of paris waste upon the chlorinated solvent concentrations of the river Seine (France). *Water Air Soil Pollut* 117: 273-288. <http://dx.doi.org/10.1023/A:1005165126290>
- Durkee, J. (2014). Cleaning with solvents: Methods and machinery. In *Cleaning with solvents: Methods and machinery*. Oxford, UK: Elsevier Inc.
<https://www.sciencedirect.com/book/9780323225205/cleaning-with-solvents-methods-and-machinery>
- Dzul-Caamal, R; Olivares-Rubio, HF; López-Tapia, P; Vega-López, A. (2013). Pro-oxidant and antioxidant response elicited by CH₂Cl₂, CHCl₃ and BrCHCl₂ in *Goodea gracilis* using non-invasive methods. *Comp Biochem Physiol A Mol Integr Physiol* 165: 515-527.
<http://dx.doi.org/10.1016/j.cbpa.2013.03.005>
- E I Dupont Denemours & Co Inc. (1987a). DAPHNIA MAGNA STATIC ACUTE 48-HOUR EC50 OF METHYLENE CHLORIDE (SANITIZED). (OTS: OTS0514009; 8EHQ Num: NA; DCN: 86-880000119S; TSCATS RefID: 305184; CIS: NA).
- E I Dupont Denemours & Co Inc. (1987b). FLOW-THROUGH ACUTE 96-HOUR LC50 OF METHYLENE CHLORIDE TO RAINBOW TROUT (SANITIZED). (OTS: OTS0514008; 8EHQ Num: NA; DCN: 86-880000118S; TSCATS RefID: 305182; CIS: NA).
- Echa. (2013). SpERC Fact Sheet – Formulation & (re)packing of substances and mixtures – Industrial (Solvent-borne).
- Enander, RT; Cohen, HJ; Gute, DM; Brown, LC; Desmaris, AM; Missaghian, R. (2004). Lead and methylene chloride exposures among automotive repair technicians. *J Occup Environ Hyg* 1: 119-125. <http://dx.doi.org/10.1080/15459620490275911>
- EPA, US. (1985). OCCUPATIONAL EXPOSURE AND ENVIRONMENTAL RELEASE ASSESSMENT OF METHYLENE CHLORIDE CONTRACT NO 68-02-3935. (OTS: OTS0505611; 8EHQ Num: 48503 B2-10; DCN: 45-8503010; TSCATS RefID: 30192; CIS: NA).
- EPA, US. (1996). Technical Support Document for the Round Two Sewage Sludge Pollutants. (822R96003). <http://nepis.epa.gov/exe/ZyPURL.cgi?Dockkey=20003N5O.txt>

- EPA, US. (1998). Guidelines for ecological risk assessment [EPA Report]. (EPA/630/R-95/002F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. <https://www.epa.gov/risk/guidelines-ecological-risk-assessment>
- EPA, US. (2002). A review of the reference dose and reference concentration processes. (EPA/630/P-02/002F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. <https://www.epa.gov/sites/production/files/2014-12/documents/rfd-final.pdf>
- EPA, US. (2003a). Attachment 1-3 Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs): Evaluation of Dermal Contact and Inhalation Exposure Pathways for the Purpose of Setting Eco-SSLs. (OSWER9285755E). Washington, DC: U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. https://www.epa.gov/sites/production/files/2015-09/documents/ecossl_attachment_1-3.pdf
- EPA, US. (2003b). Guidance for developing ecological soil screening levels (Eco-SSLs): Review of background concentration for metals - Attachment 1-4 [EPA Report]. (OSWER Directive 92857-55). Washington, DC.
- EPA, US. (2005a). Guidelines for carcinogen risk assessment [EPA Report] (pp. 1-166). (EPA/630/P-03/001F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf
- EPA, US. (2005b). Notice of availability; Documents entitled: Guidelines for carcinogen risk assessment and supplemental guidance for assessing susceptibility from early-life exposure to carcinogens. Fed Reg 70: 17765-17817.
- EPA, US. (2009). Risk assessment guidance for superfund volume I: Human health evaluation manual (Part F, supplemental guidance for inhalation risk assessment): Final [EPA Report]. (EPA/540/-R-070/002). Washington, DC. <https://www.epa.gov/risk/risk-assessment-guidance-superfund-rags-part-f>
- EPA, US. (2011a). Exposure factors handbook: 2011 edition (final) [EPA Report]. (EPA/600/R-090/052F). Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=236252>
- EPA, US. (2011b). Highlights of the exposure factors handbook (Final Report). (EPA/600/R-10/030). Washington, DC.
- EPA, US. (2012a). Benchmark dose technical guidance. (EPA/100/R-12/001). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. <https://www.epa.gov/risk/benchmark-dose-technical-guidance>
- EPA, US. (2012b). Sustainable futures: P2 framework manual [EPA Report]. (EPA/748/B-12/001). Washington DC. <http://www.epa.gov/sustainable-futures/sustainable-futures-p2-framework-manual>
- EPA, US. (2013a). ChemSTEER user guide - Chemical screening tool for exposures and environmental releases. Washington, D.C. https://www.epa.gov/sites/production/files/2015-05/documents/user_guide.pdf
- EPA, US. (2013b). Interpretive assistance document for assessment of discrete organic chemicals. Sustainable futures summary assessment [EPA Report]. Washington, DC. http://www.epa.gov/sites/production/files/2015-05/documents/05-iad_discretos_june2013.pdf

- EPA, US. (2013c). Toxicological review of Methanol (Noncancer) (CASRN 67-56-1) in support of summary information on the Integrated Risk Information System (IRIS) [EPA Report]. (EPA/635/R-11-001F). Washington, DC.
- EPA, US. (2014a). Framework for human health risk assessment to inform decision making. Final [EPA Report]. (EPA/100/R-14/001). Washington, DC: U.S. Environmental Protection, Risk Assessment Forum. <https://www.epa.gov/risk/framework-human-health-risk-assessment-inform-decision-making>
- EPA, US. (2014b). Guidance for applying quantitative data to develop data-derived extrapolation factors for interspecies and intraspecies extrapolation [EPA Report]. (EPA/100/R-14/002F). Washington, DC: Risk Assessment Forum, Office of the Science Advisor. <https://www.epa.gov/sites/production/files/2015-01/documents/ddef-final.pdf>
- EPA, US. (2017). Consumer Exposure Model (CEM) version 2.0: User guide. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. https://www.epa.gov/sites/production/files/2017-06/documents/cem_2.0_user_guide.pdf
- EPA, US. (2018a). 2014 National Emissions Inventory Report. <https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data>
- EPA, US. (2019a). Draft Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies. (Docket EPA-HQ-OPPT-2019-0236).
- EPA, US. (2019b). Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment. Docket # EPA-HQ-OPPT-2016-0742.
- EPA, US. (2019c). Risk evaluation for methylene chloride (dichloromethane, DCM): Systematic review supplemental file: Data quality evaluation of environmental releases and occupational exposure common sources . Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019d). Risk evaluation for methylene chloride (dichloromethane, DCM): Systematic review supplemental file: Data quality evaluation of environmental releases and occupational exposure data. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019e). Risk Evaluation for Methylene Chloride Systematic Review Supplemental File: Data Extraction Tables for Environmental Fate and Transport Studies. Draft Report.
- EPA, US. (2019f). Risk Evaluation for Methylene Chloride Systematic Review Supplemental File: Data Quality Evaluation of Environmental Fate and Transport Studies. Draft Report.
- EPA, US. (2019g). Risk Evaluation for Methylene Chloride, Supplemental File: Information on Consumer Exposure Assessment. Draft Report.
- EPA, US. (2019h). Risk Evaluation for Methylene Chloride, Supplemental File: Methylene Chloride Benchmark Dose and PBPK Modeling Report. U.S. Environmental Protection Agency.
- EPA, US. (2019i). Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Input Parameters. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019j). Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Outputs. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019k). Risk Evaluation for Methylene Chloride, Supplemental Information on Surface Water Exposure Assessment. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.

- EPA, US. (2019l). Risk Evaluation for Methylene Chloride, Supplemental Information: Risk Calculator for Consumer Dermal Exposures. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019m). Risk Evaluation for Methylene Chloride, Supplemental Information: Risk Calculator for Consumer Inhalation Exposures. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019n). Risk Evaluation for Methylene Chloride, Supplemental Information: Risk Calculator for Occupational Exposures. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019o). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction of Human Health Hazard Studies. U.S. Environmental Protection Agency.
- EPA, US. (2019p). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction Tables for Consumer and Environmental Exposure Studies. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019q). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation for Data Sources on Consumer and Environmental Exposure. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019r). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2019s). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Epidemiological Studies. U.S. Environmental Protection Agency.
- EPA, US. (2019t). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Human Controlled Experiments. U.S. Environmental Protection Agency.
- EPA, US. (2019u). Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal Studies. U.S. Environmental Protection Agency.
- EPA, US. (2020a). Memorandum: NIOSH/BLS Respirator Usage in Private Sector Firms [Personal Communication]. <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2019-0500-0029>
- EPA, US. (2020b). Risk Evaluation for Methylene Chloride, Supplemental Information: Consumer Risk Calculator Dermal. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, US. (2020c). Risk Evaluation for Methylene Chloride, Supplemental Information: Consumer Risk Calculator Inhalation. Docket # EPA-HQ-OPPT-2016-0742. Washington, DC.
- EPA, USEPAUS. (1994). Guidelines for Statistical Analysis of Occupational Exposure Data: Final. United States Environmental Protection Agency :: U.S. EPA.
- Erickson, MD; Harris, BSH; Pellizzari, ED; Tomer, KB; Waddell, RD. (1980). Acquisition and chemical analysis of mother's milk for selected toxic substances. Research Triangle Park, NC: Research Triangle Inst. <https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=PB81231029>

- Fairfax, R; Porter, E. (2006). OSHA compliance issues - Evaluation of worker exposure to TDI, MOCA, and methylene chloride. *J Occup Environ Hyg* 3: D50-D53.
<http://dx.doi.org/10.1080/15459620600671688>
- Finkel, A, .M. (2017). [Comment letter of Adam M. Finkel regarding Docket ID No. EPA-HQ-OPPT-2016-0231-0536. Available online at
<https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0231-0536>
- Fisk, G; Whittaker, S. (2018). Summary of 2007 – 2016 Washington Poison Center Methylene Chloride Exposure Calls in King County. Washington Poison Center.
- Foley, JF; Tuck, PD; Ton, TV; Frost, M; Kari, F; Anderson, MW; Maronpot, RR. (1993). Inhalation exposure to a hepatocarcinogenic concentration of methylene chloride does not induce sustained replicative DNA synthesis in hepatocytes of female B6C3F1 mice. *Carcinogenesis* 14: 811-817. <http://dx.doi.org/10.1093/carcin/14.5.811>
- Foster, JR; Green, T; Smith, LL; Lewis, RW; Hext, PM; Wyatt, I. (1992). Methylene chloride--an inhalation study to investigate pathological and biochemical events occurring in the lungs of mice over an exposure period of 90 days. *Fundam Appl Toxicol* 18: 376-388.
<http://dx.doi.org/10.1093/toxsci/18.3.376>
- Foster, JR; Green, T; Smith, LL; Tittensor, S; Wyatt, I. (1994). Methylene chloride: an inhalation study to investigate toxicity in the mouse lung using morphological, biochemical and Clara cell culture techniques. *Toxicology* 91: 221-234. [http://dx.doi.org/10.1016/0300-483X\(94\)90011-6](http://dx.doi.org/10.1016/0300-483X(94)90011-6)
- Frasch, HF; Bunge, AL. (2015). The transient dermal exposure II: post-exposure absorption and evaporation of volatile compounds. *J Pharm Sci* 104: 1499-1507.
<http://dx.doi.org/10.1002/jps.24334>
- Fuxe, K; Andersson, K; Hansson, T; Agnati, LF; Eneroth, P; Gustafsson, JA. (1984). Central catecholamine neurons and exposure to dichloromethane. Selective changes in amine levels and turnover in tel- and diencephalic DA and NA nerve terminal systems and in the secretion of anterior pituitary hormones in the male rat. *Toxicology* 29: 293-305.
[http://dx.doi.org/10.1016/0300-483X\(84\)90161-6](http://dx.doi.org/10.1016/0300-483X(84)90161-6)
- Gamberale, F; Annwall, G; Hultengren, M. (1975). Exposure to methylene chloride: II. Psychological functions. *Scand J Work Environ Health* 1: 95-103.
- Garcia, E; Hurley, S; Nelson, DO; Hertz, A; Reynolds, P. (2015). Hazardous air pollutants and breast cancer risk in California teachers: a cohort study. *Environ Health* 14: 14.
- Geiger, DL; Poirier, SH; Brooke, LT; Call, DJ. (1986). Acute toxicities of organic chemicals to fathead minnows (*Pimephales promelas*): Volume III. Superior, WI: University of Wisconsin-Superior, Center for Lake Superior Environmental Studies.
- General Electric Co. (1976a). DICHLOROMETHANE FOURTEEN DAY RANGE FINDING STUDY IN RATS. (OTS: OTS0205887; 8EHQ Num: NA; DCN: 878210707; TSCATS RefID: 16714; CIS: NA).
- General Electric Co. (1976b). DICHLOROMETHANE NINETY DAY ORAL TOXICITY STUDY IN DOGS. (OTS: OTS0205887; 8EHQ Num: NA; DCN: 878210709; TSCATS RefID: 16716; CIS: NA).
- General Electric Co. (1989). MORBIDITY STUDY OF OCCUPATIONAL EXPOSURE TO METHYLENE CHLORIDE USING A COMPUTERIZED SURVEILLANCE SYSTEM (FINAL REPORT) WITH COVER LETTER DATED 073189. (OTS: OTS0521036; 8EHQ Num: NA; DCN: 86-890001420; TSCATS RefID: 404504; CIS: NA).

- General Electric Co. (1990). MORBIDITY STUDY OF OCCUPATIONAL EXPOSURE TO METHYLENE CHLORIDE USING A COMPUTERIZED SURVEILLANCE SYSTEM (FINAL REPORT) WITH COVER SHEETS AND LETTER DATED 041190. (OTS: OTS0522984; 8EHQ Num: NA; DCN: 86-900000421; TSCATS RefID: 406678; CIS: NA).
- General Electric Company. (1976). Dichloromethane: Reproduction and ninety day oral toxicity study in rats. (878210710). Mattawan, MI: International Research and Development Corporation.
- Gibbs, GW; Amsel, J; Soden, K. (1996). A cohort mortality study of cellulose triacetate-fiber workers exposed to methylene chloride. *J Occup Environ Med* 38: 693-697.
- Gilbert, D; Goyer, M; Lyman, W; Magil, G; Walker, P; Wallace, D; Wechsler, A; Yee, J. (1982). An exposure and risk assessment for tetrachloroethylene. (EPA-440/4-85-015). Washington, DC: U.S. Environmental Protection Agency, Office of Water Regulations and Standards. <http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=2000LLOH.txt>
- Gocke, E; King, MT; Eckhardt, K; Wild, D. (1981). [Mutagenicity of cosmetics ingredients licensed by the European communities]. *Mutat Res* 90: 91-109. [http://dx.doi.org/10.1016/0165-1218\(81\)90072-0](http://dx.doi.org/10.1016/0165-1218(81)90072-0)
- Gold, LS; Stewart, PA; Milliken, K; Purdue, M; Severson, R; Seixas, N; Blair, A; Hartge, P; Davis, S; De Roos, AJ. (2010). The relationship between multiple myeloma and occupational exposure to six chlorinated solvents. *Occup Environ Med* 68: 391-399.
- Gossett, JM. (1985). Anaerobic degradation of C1 and C2 chlorinated hydrocarbons. (ESL-TR-85-38). Tyndal AFB, FL: Air Force Engineering & Services Center.
- Graves, RJ; Callander, RD; Green, T. (1994a). The role of formaldehyde and S-chloromethylglutathione in the bacterial mutagenicity of methylene chloride. *Mutat Res* 320: 235-243. [http://dx.doi.org/10.1016/0165-1218\(94\)90050-7](http://dx.doi.org/10.1016/0165-1218(94)90050-7)
- Graves, RJ; Coutts, C; Eyton-Jones, H; Green, T. (1994b). Relationship between hepatic DNA damage and methylene chloride-induced hepatocarcinogenicity in B6C3F1 mice. *Carcinogenesis* 15: 991-996. <http://dx.doi.org/10.1093/carcin/15.5.991>
- Graves, RJ; Coutts, C; Green, T. (1995). Methylene chloride-induced DNA damage: An interspecies comparison. *Carcinogenesis* 16: 1919-1926. <http://dx.doi.org/10.1093/carcin/16.8.1919>
- Graves, RJ; Green, T. (1996). Mouse liver glutathione S-transferase mediated metabolism of methylene chloride to a mutagen in the CHO/HPRT assay. *Mutat Res Genet Toxicol* 367: 143-150. [http://dx.doi.org/10.1016/0165-1218\(95\)00087-9](http://dx.doi.org/10.1016/0165-1218(95)00087-9)
- Graves, RJ; Trueman, P; Jones, S; Green, T. (1996). DNA sequence analysis of methylene chloride-induced HPRT mutations in Chinese hamster ovary cells: Comparison with the mutation spectrum obtained for 1,2-dibromoethane and formaldehyde. *Mutagenesis* 11: 229-233. <http://dx.doi.org/10.1093/mutage/11.3.229>
- Green, T. (1983). The metabolic activation of dichloromethane and chlorofluoromethane in a bacterial mutation assay using *Salmonella typhimurium*. *Mutat Res Genet Toxicol* 118: 227-288. [http://dx.doi.org/10.1016/0165-1218\(83\)90211-2](http://dx.doi.org/10.1016/0165-1218(83)90211-2)
- Gregus, Z. (2008). Chapter 3: Mechanisms of Toxicity. In CD Klaassen (Ed.), (7th ed., pp. 45-106). New York, NY: McGraw Hill Medical Publishing Division.
- Haber, LT; Maier, A; Gentry, PR; Clewell, HJ; Dourson, ML. (2002). Genetic polymorphisms in assessing interindividual variability in delivered dose [Review]. *Regul Toxicol Pharmacol* 35: 177-197. <http://dx.doi.org/10.1006/rtph.2001.1517>

- Hall, AH; Rumack, BH. (1990). Methylene chloride exposure in furniture-stripping shops: Ventilation and respirator use practices. *J Occup Med* 32: 33-37.
- Halogenated Solvents Industry Alliance, I. (2018). [Comment letter of Halogenated Solvents Industry Alliance, Inc. (HSIA) regarding Docket ID No. EPA-HQ-OPPT-2016-0742-0103]. Available online at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0103>
- Hansch, C; Leo, A; Hoekman, D. (1995). Exploring QSAR: Hydrophobic, electronic, and steric constants. In C Hansch; A Leo; DH Hoekman (Eds.), *Exploring QSAR: Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society.
- Hardin, BD; Manson, JM. (1980). Absence of dichloromethane teratogenicity with inhalation exposure in rats. *Toxicol Appl Pharmacol* 52: 22-28. [http://dx.doi.org/10.1016/0041-008X\(80\)90243-4](http://dx.doi.org/10.1016/0041-008X(80)90243-4)
- Haun, CC; Harris, ES; Darmer, KI, Jr. (1971). Continuous animal exposure to methylene chloride. In *Proceedings of the annual conference on environmental toxicology (2nd held at Fairborn, Ohio on 31 August, 1 and 2 September 1971 (pp. 125-135)*. (AMRL-TR-71-120, paper no. 10). Wright-Patterson AFB, OH: Aerospace Medical Research Laboratory. <https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=AD751432>
- Haun, CC; Vernot, EH; Darmer, KI, Jr; Diamond, SS. (1972). Continuous animal exposure to low levels of dichloromethane. In *Proceedings of the annual conference on environmental toxicology (3rd held in Fairborn, Ohio, on 25-27 October 1972 (pp. 199-208)*. (AMRL-TR-72-130, paper no. 12). Wright-Patterson AFB, OH: Aerospace Medical Research Laboratory.
- Hazleton Laboratories. (1983). 24-month oncogenicity study of methylene chloride in mice: Final report. (45-8303005). New York, NY: National Coffee Association. <https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=OTS0505606>
- Health Canada. (1993). Canadian Environmental Protection Act priority substances list assessment report: Dichloromethane. (NTIS/02990019_2). Ottawa, Canada: Canada Communication Group.
- Hearne, FT; Pifer, JW. (1999). Mortality study of two overlapping cohorts of photographic film base manufacturing employees exposed to methylene chloride. *J Occup Environ Med* 41: 1154-1169.
- Hegi, ME; Söderkvist, P; Foley, JF; Schoonhoven, R; Swenberg, JA; Kari, F; Maronpot, R; Anderson, MW; Wiseman, RW. (1993). Characterization of p53 mutations in methylene chloride-induced lung tumors from B6C3F1 mice. *Carcinogenesis* 14: 803-810. <http://dx.doi.org/10.1093/carcin/14.5.803>
- Heineman, EF; Cocco, P; Gomez, MR; Dosemeci, M; Stewart, PA; Hayes, RB; Zahm, SH; Thomas, TL; Blair, A. (1994). Occupational exposure to chlorinated aliphatic hydrocarbons and risk of astrocytic brain cancer. *Am J Ind Med* 26: 155-169.
- Heitmuller, PT; Hollister, TA; Parrish, PR. (1981). Acute toxicity of 54 industrial chemicals to sheepshead minnows (*Cyprinodon variegatus*). *Bull Environ Contam Toxicol* 27: 596-604. <http://dx.doi.org/10.1007/BF01611069>
- Heppel, LA; Neal, PA. (1944). Toxicology of dichloromethane (methylene chloride): II: Its effect upon running activity in the male rat. *J Ind Hyg Toxicol* 26: 17-21.
- Hirata, T; Cho, YM; Toyoda, T; Akagi, JI; Suzuki, I; Nishikawa, A; Ogawa, K. (2016). Lack of in vivo mutagenicity of 1,2-dichloropropane and dichloromethane in the livers of gpt

- delta rats administered singly or in combination. *J Appl Toxicol* 37: 683-691.
<http://dx.doi.org/10.1002/jat.3416>
- hoechst celanese corp. (1992). SUPPLEMENT: MORTALITY OR WORKERS EMPLOYED AT A CELLULOSE ACETATE & TRIACETATE FIBERS PLANT IN CUMBERLAND, MD (FINAL REPORT) WITH COVER LETTER DATED 061792. (OTS: OTS0516635-3; 8EHQ Num: 8EHQ-0692-0772; DCN: 89-920000119; TSCATS RefID: 427311; CIS: NA).
- Holbrook, MT. (2003). Methylene chloride. In *Kirk-Othmer Encyclopedia of Chemical Technology* (4th ed.). New York, NY: John Wiley & Sons.
<http://dx.doi.org/10.1002/0471238961.1305200808151202.a02.pub2>
- Horvath, AL. (1982). Halogenated hydrocarbons: Solubility-miscibility with water. New York, NY: Marcel Dekker, Inc.
- Hossaini, R; Chipperfield, MP; Saiz-Lopez, A; Harrison, JJ; von Glasow, R; Sommariva, R; Atlas, E; Navarro, M; Montzka, SA; Feng, W; Dhomse, S; Harth, C; Muehle, J; Lunder, C; O'Doherty, S; Young, D; Reimann, S; Vollmer, MK; Krummel, PB; Bernath, PF. (2015). Growth in stratospheric chlorine from short-lived chemicals not controlled by the Montreal Protocol. *Geophys Res Lett* 42: 4573-4580.
<http://dx.doi.org/10.1002/2015GL063783>
- Hsdb. (2012). Dichloromethane. <https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~w1qCJ9:1>
- HSL. (2007). Protective glove selection for workers using NMP containing products -Graffiti removal. (HSL/2007/41). United Kingdom: Health and Safety Laboratory.
http://www.hse.gov.uk/research/hsl_pdf/2007/hsl0741.pdf
- Hu, Y; Kabler, SL; Tennant, AH; Townsend, AJ; Kligerman, AD. (2006). Induction of DNA-protein crosslinks by dichloromethane in a V79 cell line transfected with the murine glutathione-S-transferase theta 1 gene. *Mutat Res Genet Toxicol Environ Mutagen* 607: 231-239. <http://dx.doi.org/10.1016/j.mrgentox.2006.04.013>
- Hughes, NJ; Tracey, JA. (1993). A case of methylene chloride (nitromors) poisoning, effects on carboxyhaemoglobin levels. *Hum Exp Toxicol* 12: 159-160.
- IARC. (2016). Dichloromethane [IARC Monograph]. In *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. Lyon, France.
<http://monographs.iarc.fr/ENG/Monographs/vol110/mono110-04.pdf>
- Iarc. (2019). Carcinogenicity of night shift work. [http://dx.doi.org/10.1016/S1470-2045\(19\)30455-3](http://dx.doi.org/10.1016/S1470-2045(19)30455-3)
- ICIS. (2005). Chemical profile: Methylene chloride.
<https://www.icis.com/resources/news/2005/12/02/580954/chemical-profile-methylene-chloride/>
- IHS Markit. (2016). *Chemical Economics Handbook: Chlorinated Methanes*.
<https://www.ihs.com/products/chlorinatedmethanes->
- Infante-Rivard, C; Siemiatycki, J; Lakhani, R; Nadon, L. (2005). Maternal exposure to occupational solvents and childhood leukemia. *Environ Health Perspect* 113: 787-792.
- Isaacs, K. (2014). The consolidated human activity database - master version (CHAD-Master) technical memorandum. Washington, DC: U.S. Environmental Protection Agency, National Exposure Research Laboratory.
https://www.epa.gov/sites/production/files/2015-02/documents/chadmaster_091814_1.pdf

- Jensen, AA. (1983). Chemical contaminants in human milk [Review]. *Residue Rev* 89: 1-128.
- Jones, DR; Abel, S; Effland, W; Matzner, R; Parker, R. (1998). An Index Reservoir for Use in Assessing Drinking Water Exposure: Chapter IV in Proposed Methods for Basin-Scale Estimation of Pesticide Concentrations in Flowing Water and Reservoirs for Tolerance Reassessment. Presented to the FIFRA Science Advisory Panel on July 29, 1998. Jones, D.R.; Abel, S.; Effland, W.; Matzner, R.; Parker, R. <https://www.epa.gov/sap/fifra-scientific-advisory-panel-meetings>
- Jongen, WMF; Alink, GM; Koeman, JH. (1978). Mutagenic effect of dichloromethane on *Salmonella typhimurium*. *Mutat Res-Fundam Mol Mech Mutagen* 56: 245-248. [http://dx.doi.org/10.1016/0027-5107\(78\)90191-4](http://dx.doi.org/10.1016/0027-5107(78)90191-4)
- Jongen, WMF; Harmsen, EGM; Alink, GM; Koeman, JH. (1982). The effect of glutathione conjugation and microsomal oxidation on the mutagenicity of dichloromethane in *S. typhimurium*. *Mutat Res-Fundam Mol Mech Mutagen* 95: 183-189. [http://dx.doi.org/10.1016/0027-5107\(82\)90256-1](http://dx.doi.org/10.1016/0027-5107(82)90256-1)
- Jongen, WMF; Lohman, PHM; Kottenhagen, MJ; Alink, GM; Berends, F; Koeman, JH. (1981). Mutagenicity testing of dichloromethane in short-term mammalian test systems. *Mutat Res-Fundam Mol Mech Mutagen* 81: 203-213. [http://dx.doi.org/10.1016/0027-5107\(81\)90035-X](http://dx.doi.org/10.1016/0027-5107(81)90035-X)
- Kalkbrenner, AE; Daniels, JL; Chen, JC; Poole, C; Emch, M; Morrissey, J. (2010). Perinatal exposure to hazardous air pollutants and autism spectrum disorders at age 8. *Epidemiology* 21: 631-641.
- Kanada, M; Miyagawa, M; Sato, M; Hasegawa, H; Honma, T. (1994). Neurochemical profile of effects of 28 neurotoxic chemicals on the central nervous system in rats (1) Effects of oral administration on brain contents of biogenic amines and metabolites. *Ind Health* 32: 145-164. <http://dx.doi.org/10.2486/indhealth.32.145>
- Kanegsberg, B; Kanegsberg, E. (2011). Handbook for critical cleaning, cleaning agents and systems (2nd ed.). Boca Raton, FL: CRC Press.
- Kanno, J; Foley, JF; Kari, F; Anderson, MW; Maronpot, RR. (1993). Effect of methylene chloride inhalation on replicative DNA synthesis in the lungs of female B6C3F1 mice. *Environ Health Perspect* 101: 271-276.
- Kari, FW; Foley, JF; Seilkop, SK; Maronpot, RR; Anderson, MW. (1993). Effect of varying exposure regimens on methylene chloride-induced lung and liver tumors in female B6C3F1 mice. *Carcinogenesis* 14: 819-826. <http://dx.doi.org/10.1093/carcin/14.5.819>
- Kayser, MF; Vuilleumier, S. (2001). Dehalogenation of dichloromethane by dichloromethane dehalogenase/glutathione S-transferase leads to formation of DNA adducts. *J Bacteriol* 183: 5209-5212.
- Kelly, M. (1988). Case reports of individuals with oligospermia and methylene chloride exposures. *Reprod Toxicol* 2: 13-17. [http://dx.doi.org/10.1016/S0890-6238\(88\)80004-2](http://dx.doi.org/10.1016/S0890-6238(88)80004-2)
- Khudoley, VV; Mizgireuv, I; Pliss, GB. (1987). The study of mutagenic activity of carcinogens and other chemical agents with *Salmonella typhimurium* assays: Testing of 126 compounds. *Arch Geschwulstforsch* 57: 453-462.
- Kim, EY; Lee, MY; Hwang, SY; Kang, I, nC. (2010). Biomarker analysis of rat livers exposed to different toxic pollutants (VOCs and PAHs) using an antibody array. *BioChip Journal* 4: 173-178. <http://dx.doi.org/10.1007/s13206-010-4302-x>
- Kim, JK; Eun, JW; Bae, HJ; Shen, Q; Park, SJ; Kim, HS; Park, S; Ahn, YM; Park, WS; Lee, JY; Nam, SW. (2013). Characteristic molecular signatures of early exposure to volatile

- organic compounds in rat liver. *Biomarkers* 18: 706-715.
<http://dx.doi.org/10.3109/1354750X.2013.847121>
- Kirschman, JC; Brown, NM; Coots, RH; Morgareidge, K. (1986). Review of investigations of dichloromethane metabolism and subchronic oral toxicity as the basis for the design of chronic oral studies in rats and mice. *Food Chem Toxicol* 24: 943-949.
[http://dx.doi.org/10.1016/0278-6915\(86\)90322-4](http://dx.doi.org/10.1016/0278-6915(86)90322-4)
- Kitchin, KT; Brown, JL. (1989). Biochemical effects of three carcinogenic chlorinated methanes in rat liver. *Teratog Carcinog Mutagen* 9: 61-69.
<http://dx.doi.org/10.1002/tcm.1770090108>
- Kjellstrand, P; Holmquist, B; Jonsson, I; Romare, S; Mansson, L. (1985). Effects of organic solvents on motor activity in mice. *Toxicology* 35: 35-46. [http://dx.doi.org/10.1016/0300-483X\(85\)90130-1](http://dx.doi.org/10.1016/0300-483X(85)90130-1)
- Kleinman, MT; Davidson, DM; Vandagriff, RB; Caiozzo, VJ; Whittenberger, JL. (1989). Effects of short-term exposure to carbon monoxide in subjects with coronary artery disease. *Arch Environ Occup Health* 44: 361-369. <http://dx.doi.org/10.1080/00039896.1989.9935908>
- Kleinman, MT; Leaf, DA; Kelly, E; Caiozzo, V; Osann, K; O'Niell, T. (1998). Urban angina in the mountains: effects of carbon monoxide and mild hypoxemia on subjects with chronic stable angina. *Arch Environ Occup Health* 53: 388-397.
<http://dx.doi.org/10.1080/00039899809605726>
- Kolodner, K; Cameron, L; Gittlesohn, A. (1990). Morbidity study of occupational exposure to methylene chloride using a computerized surveillance system (final report) with cover sheets and letter dated 041190. (86900000421). Baltimore, MD: Johns Hopkins School of Hygiene and Public Health.
<https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=OTS0522984>
- Kozena, L; Frantik, E; Vodickova, A. (1990). Methylene chloride dose not impair vigilance performance at blood levels simulating limit exposure. *Activitas Nervosa Superior* 32: 35-37. https://heronet.epa.gov/heronet/index.cfm/reference/download/reference_id/29233
- Kramer, VC; Schnell, DJ; Nickerson, KW. (1983). Relative toxicity of organic solvents to *Aedes aegypti* larvae. *J Invertebr Pathol* 42: 285-287. [http://dx.doi.org/10.1016/0022-2011\(83\)90076-9](http://dx.doi.org/10.1016/0022-2011(83)90076-9)
- Kramers, PGN; Mout, HCA; Bissumbar, B; Mulder, CR. (1991). Inhalation exposure in *Drosophila* mutagenesis assays: Experiments with aliphatic halogenated hydrocarbons, with emphasis on the genetic activity profile of 1,2-dichloroethane. *Mutat Res* 252: 17-33. [http://dx.doi.org/10.1016/0165-1161\(91\)90248-7](http://dx.doi.org/10.1016/0165-1161(91)90248-7)
- Krausova, VI; Robb, FT; Gonzalez, JM. (2006). Biodegradation of dichloromethane in an estuarine environment. *Hydrobiologia* 559: 77-83. <http://dx.doi.org/10.1007/s10750-004-0571-5>
- Kubulus, D; Mathes, A; Pradarutti, S; Raddatz, A; Heiser, J; Pavlidis, D; Wolf, B; Bauer, I; Rensing, H. (2008). Hemin arginate-induced heme oxygenase 1 expression improves liver microcirculation and mediates an anti-inflammatory cytokine response after hemorrhagic shock. *Shock* 29: 583-590.
<http://dx.doi.org/10.1097/SHK.0b013e318157e526>
- Kuhn, R; Pattard, M; Pernak, KD; Winter, A. (1989). Results of the harmful effects of selected water pollutants (anilines, phenols, aliphatic compounds) to *Daphnia magna*. *Water Res* 23: 495-499. [http://dx.doi.org/10.1016/0043-1354\(89\)90141-3](http://dx.doi.org/10.1016/0043-1354(89)90141-3)

- Kumagai, S; Sobue, T; Makiuchi, T; Kubo, S; Uehara, S; Hayashi, T; Sato, KK; Endo, G. (2016). Relationship between cumulative exposure to 1,2-dichloropropane and incidence risk of cholangiocarcinoma among offset printing workers. *Occup Environ Med* 73: 545-552.
- Landi, S; Naccarati, A; Ross, MK; Hanley, NM; Dailey, L; Devlin, RB; Vasquez, M; Pegram, RA; DeMarini, DM. (2003). Induction of DNA strand breaks by trihalomethanes in primary human lung epithelial cells. *Mutat Res Genet Toxicol Environ Mutagen* 538: 41-50. [http://dx.doi.org/10.1016/S1383-5718\(03\)00086-X](http://dx.doi.org/10.1016/S1383-5718(03)00086-X)
- Lanes, SF; Cohen, A; Rothman, KJ; Dreyer, NA; Soden, KJ. (1990). Mortality of cellulose fiber production workers. *Scand J Work Environ Health* 16: 247-251.
- Lanes, SF; Rothman, KJ; Dreyer, NA; Soden, KJ. (1993). Mortality update of cellulose fiber production workers. *Scand J Work Environ Health* 19: 426-428.
- Lapertot, ME; Pulgarin, C. (2006). Biodegradability assessment of several priority hazardous substances: Choice, application and relevance regarding toxicity and bacterial activity. *Chemosphere* 65: 682-690. <http://dx.doi.org/10.1016/j.chemosphere.2006.01.046>
- Lash, AA; Becker, CE; So, Y; Shore, M. (1991). Neurotoxic effects of methylene chloride: Are they long lasting in humans? *Occup Environ Med* 48: 418-426.
- Laurence, C; Nicolet, P; Dalati, MT; Abboud, JLM; Notario, R. (1994). The empirical treatment of solvent-solute interactions: 15 years of pi. *J Phys Chem* 98: 5807-5816. <http://dx.doi.org/10.1021/j100074a003>
- Leblanc, GA. (1980). Acute toxicity of priority pollutants to water flea (*Daphnia magna*). *Bull Environ Contam Toxicol* 24: 684-691. <http://dx.doi.org/10.1007/BF01608174>
- Lefevre, PA; Ashby, J. (1989). Evaluation of dichloromethane as an inducer of DNA synthesis in the B6C3F1 mouse liver. *Carcinogenesis* 10: 1067-1072. <http://dx.doi.org/10.1093/carcin/10.6.1067>
- Leighton, DT, Jr; Calo, JM. (1981). Distribution coefficients of chlorinated hydrocarbons in dilute air-water systems for groundwater contamination applications. *Journal of Chemical and Engineering Data* 26: 382-585. <http://dx.doi.org/10.1021/je00026a010>
- Leuschner, F; Neumann, BW; Huebscher, F. (1984). Report on subacute toxicological studies with dichloromethane in rats and dogs by inhalation. *Arzneimittelforschung* 34: 1772-1774.
- Li, CY; Sung, FC. (1999). A review of the healthy worker effect in occupational epidemiology [Review]. *Occup Med (Lond)* 49: 225-229.
- Lindstrom, AB; Proffitt, D; Fortune, CR. (1995). Effects of modified residential construction on indoor air quality. *Indoor Air* 5: 258-269. <http://dx.doi.org/10.1111/j.1600-0668.1995.00005.x>
- Love, JR; Kern, M. (1981). Health hazard evaluation report no. HETA-81-065-938, METRO Bus Maintenance Shop, Washington, DC. (HETA-81-065-938). Cincinnati, OH: National Institute for Occupational Safety and Health. <https://www.cdc.gov/niosh/hhe/reports/pdfs/81-65-938.pdf>
- Ma, H; Zhang, H; Wang, L; Wang, J; Chen, J. (2014). Comprehensive screening and priority ranking of volatile organic compounds in Daliao River, China. *Environ Monit Assess* 186: 2813-2821. <http://dx.doi.org/10.1007/s10661-013-3582-8>
- Macisaac, J; Harrison, R; Krishnaswami, J; McNary, J; Suchard, J; Boysen-Osborn, M; Cierpich, H; Styles, L; Shusterman, D. (2013). Fatalities due to dichloromethane in paint strippers: a continuing problem. *Am J Ind Med* 56: 907-910. <http://dx.doi.org/10.1002/ajim.22167>

- Maltoni, C; Cotti, G; Perino, G. (1988). Long-term carcinogenicity bioassays on methylene chloride administered by ingestions to Sprague-Dawley rats and Swiss mice and by inhalation to Sprague-Dawley rats. *Ann N Y Acad Sci* 534: 352-366. <http://dx.doi.org/10.1111/j.1749-6632.1988.tb30122.x>
- Mansouri, K; Grulke, CM; Judson, RS; Williams, AJ. (2018). OPERA models for predicting physicochemical properties and environmental fate endpoints. 10: 10. <http://dx.doi.org/10.1186/s13321-018-0263-1>
- Marino, DJ; Clewell, HJ; Gentry, PR; Covington, TR; Hack, CE; David, RM; Morgott, DA. (2006). Revised assessment of cancer risk to dichloromethane: Part I Bayesian PBPK and dose-response modeling in mice. *Regul Toxicol Pharmacol* 45: 44-54. <http://dx.doi.org/10.1016/j.yrtph.2005.12.007>
- Marquart, H; Franken, R; Goede, H; Fransman, W; Schinkel, J. (2017). Validation of the dermal exposure model in ECETOC TRA. *Annals of Work Exposures and Health* 61: 854-871. <http://dx.doi.org/10.1093/annweh/wxx059>
- Marquis, O; Millery, A; Guittonneau, S; Miaud, C. (2006). Solvent toxicity to amphibian embryos and larvae. *Chemosphere* 63: 889-892. <http://dx.doi.org/10.1016/j.chemosphere.2005.07.063>
- Marsch, GA; Botta, S; Martin, MV; McCormick, WA; Guengerich, FP. (2004). Formation and mass spectrometric analysis of DNA and nucleoside adducts by S-(1-acetoxymethyl)glutathione and by glutathione S-transferase-mediated activation of dihalomethanes. *Chem Res Toxicol* 17: 45-54. <http://dx.doi.org/10.1021/tx034156z>
- Mattei, F; Guida, F; Matrat, M; Cenée, S; Cyr, D; Sanchez, M; Radoi, L; Menvielle, G; Jellouli, F; Carton, M; Bara, S; Marrer, E; Luce, D; Stücker, I. (2014). Exposure to chlorinated solvents and lung cancer: results of the ICARE study. *Occup Environ Med* 71: 681-689.
- Mattsson, JL; Albee, RR; Eisenbrandt, DL. (1990). Neurotoxicologic evaluation of rats after 13 weeks of inhalation exposure to dichloromethane or carbon monoxide. *Pharmacol Biochem Behav* 36: 671-681. [http://dx.doi.org/10.1016/0091-3057\(90\)90273-K](http://dx.doi.org/10.1016/0091-3057(90)90273-K)
- Mccammon, CS. (1990). Health Hazard Evaluation Report HETA 89-199-2033, Enseco, Inc., Rocky Mountain Analytical Laboratory, Arvada, Colorado. (NTIS/02971023_a). Mccammon, CS.
- Melin, ES; Puhakka, JA; Strand, SE; Rockne, KJ; Ferguson, JF. (1996). Fluidized-bed enrichment of marine ammonia-to-nitrite oxidizers and their ability to degrade chloroaliphatics. *Int Biodeterior Biodegradation* 38: 9-18. [http://dx.doi.org/10.1016/S0964-8305\(96\)00004-2](http://dx.doi.org/10.1016/S0964-8305(96)00004-2)
- Mennear, JH; McConnell, EE; Huff, JE; Renne, RA; Giddens, E. (1988). Inhalation toxicology and carcinogenesis studies of methylene chloride (dichloromethane) in F344/N rats and B6C3F1 mice. *Ann N Y Acad Sci* 534: 343-351. <http://dx.doi.org/10.1111/j.1749-6632.1988.tb30121.x>
- Miligi, L; Costantini, AS; Benvenuti, A; Kriebel, D; Bolejack, V; Tumino, R; Ramazzotti, V; Rodella, S; Stagnaro, E; Crosignani, P; Amadori, D; Mirabelli, D; Sommani, L; Belletti, I; Trochel, L; Romeo, L; Miceli, G; Tozzi, GA; Mendico, I; Vineis, P. (2006). Occupational exposure to solvents and the risk of lymphomas. *Epidemiology* 17: 552-561.
- Miller, MA; Bhatt, V; Kasting, GB. (2005). Dose and airflow dependence of benzyl alcohol disposition on skin. *J Pharm Sci* 95: 281-291. <http://dx.doi.org/10.1002/jps.20513>

- Mimaki, S; Totsuka, Y; Suzuki, Y; Nakai, C; Goto, M; Kojima, M; Arakawa, H; Takemura, S; Tanaka, S; Marubashi, S; Kinoshita, M; Matsuda, T; Shibata, T; Nakagama, H; Ochiai, A; Kubo, S; Nakamori, S; Esumi, H; Tsuchihara, K. (2016). Hypermutation and unique mutational signatures of occupational cholangiocarcinoma in printing workers exposed to haloalkanes. *Carcinogenesis* 37: 817-826. <http://dx.doi.org/10.1093/carcin/bgw066>
- Mirsalis, JC; Tyson, CK; Steinmetz, KL; Loh, EK; Hamilton, CM; Bakke, JP; Spalding, JW. (1989). Measurement of unscheduled DNA synthesis and S-phase synthesis in rodent hepatocytes following in vivo treatment: Testing of 24 compounds. *Environ Mol Mutagen* 14: 155-164. <http://dx.doi.org/10.1002/em.2850140305>
- Morales-Suárez-Varela, MM; Olsen, J; Villeneuve, S; Johansen, P; Kaerlev, L; Llopis-González, A; Wingren, G; Hardell, L; Ahrens, W; Stang, A; Merletti, F; Gorini, G; Aurrekoetxea, JJ; Févotte, J; Cyr, D; Guénel, P. (2013). Occupational exposure to chlorinated and petroleum solvents and mycosis fungoides. *J Occup Environ Med* 55: 924-931.
- Moser, VC; Cheek, BM; Macphail, RC. (1995). A multidisciplinary approach to toxicological screening: III. Neurobehavioral toxicity. *J Toxicol Environ Health A* 45: 173-210. <http://dx.doi.org/10.1080/15287399509531988>
- Moutsopoulos, HM; Zerva, LV. (1990). Anti-Ro (SSA)/La (SSB) antibodies and Sjögren's syndrome. *Clin Rheumatol* 1990: 123-130.
- NAC/AEGL. (2008a). Carbon monoxide - acute exposure guideline levels (AEGLs). Washington, DC: National Advisory Committee for Acute Exposure Guideline Levels.
- Nac/Aegl. (2008b). Methylene chloride - interim acute exposure guideline levels (AEGLs). Washington, DC: National Advisory Committee for Acute Exposure Guideline Levels.
- Narotsky, MG; Kavlock, RJ. (1995). A multidisciplinary approach to toxicological screening: II. Developmental toxicity. *J Toxicol Environ Health* 45: 145-171. <http://dx.doi.org/10.1080/15287399509531987>
- Neta, G; Stewart, PA; Rajaraman, P; Hein, MJ; Waters, MA; Purdue, MP; Samanic, C; Coble, JB; Linet, MS; Inskip, PD. (2012). Occupational exposure to chlorinated solvents and risks of glioma and meningioma in adults. *Occup Environ Med* 69: 793-801.
- NICNAS. (2016). Human health Tier II assessment for methane, dichloro. <https://www.nicnas.gov.au/search?query=75-09-2&collection=nicnas-meta&f.IMAP+assessment+Tier%7CB=Tier+II>
- NIH. (2016). Report on carcinogens: Dichloromethane [NTP]. In Report on carcinogens: Fourteenth Edition (14th ed.). Washington, DC: National Toxicology Program. <https://ntp.niehs.nih.gov/pubhealth/roc/index-1.html#C>
- Niosh. (1976). A Guide to Industrial Respiratory Protection. (HEW Pub. 76-189). Cincinnati, OH: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health.
- NIOSH. (1985). Health hazard evaluation report no. HETA-84-214-1633, Sheldahl, Inc., Northfield, Minnesota. (HETA- 84-214-1633). Cincinnati, OH. <https://www.cdc.gov/niosh/hhe/reports/pdfs/1984-0214-1633.pdf>
- NIOSH. (1994). Methylene chloride - IDLH documentation. Cincinnati, OH: National Institutes for Occupational Safety and Health. <http://www.cdc.gov/niosh/idlh/75092.html>
- Niosh. (1998). Methylene Chloride: Method 1005, Issue 3. <https://www.cdc.gov/niosh/docs/2003-154/pdfs/1005.pdf>

- Niosh. (2002a). In-depth survey report: Control of perchloroethylene (PCE) in vapor degreasing operations, site #2. (EPHB 256-16b). CDC.
<https://www.cdc.gov/niosh/surveyreports/pdfs/256-16b.pdf>
- NIOSH. (2002b). In-depth survey report: Control of perchloroethylene exposure (PCE) in vapor degreasing operations, site #3. (EPHB 256-17b). CDC.
<https://www.cdc.gov/niosh/surveyreports/pdfs/ECTB-256-17b.pdf>
- NIOSH. (2003). Respirator Usage in Private Sector Firms. Washington D.C.: United States Department of Labor, Bureau of Labor Statistics and National Institute for Occupational Safety and Health. <https://www.cdc.gov/niosh/docs/respsurv/>
- NIOSH. (2011a). Fatality assessment and control evaluation (FACE) report for Michigan: Tub refinisher died due to methylene chloride overexposure while stripping a bathtub (pp. 21).
- Niosh. (2011b). NIOSH pocket guide to chemical hazards: Methylene chloride.
<http://www.cdc.gov/niosh/npg/npgd0414.html>
- Nitschke, KD; Burek, JD; TJ, B; Kociba, RJ; Rampy, LW; McKenna, MJ. (1988a). Methylene chloride: A 2-year inhalation toxicity and oncogenicity study in rats. *Fundam Appl Toxicol* 11: 48-59. [http://dx.doi.org/10.1016/0272-0590\(88\)90269-2](http://dx.doi.org/10.1016/0272-0590(88)90269-2)
- Nitschke, KD; Eisenbrandt, DL; Lomax, LG; Rao, KS. (1988b). Methylene chloride: Two-generation inhalation reproductive study in rats. *Fundam Appl Toxicol* 11: 60-67.
[http://dx.doi.org/10.1016/0272-0590\(88\)90270-9](http://dx.doi.org/10.1016/0272-0590(88)90270-9)
- Nrc. (1996). Spacecraft maximum allowable concentrations for selected airborne contaminants. Washington, D.C.: National Academy Press. <http://dx.doi.org/10.17226/5170>
- NRC. (2001). Standing operating procedures for developing acute exposure guideline levels (AEGs) for hazardous chemicals. Washington, DC: National Academy Press.
<http://www.epa.gov/oppt/aegl/pubs/sop.pdf>
- Nrc. (2008). Spacecraft maximum allowable concentrations for selected airborne contaminants: Volume 5. Washington, DC: National Academies Press.
http://www.nap.edu/catalog.php?record_id=12529
- Nrc. (2010). Acute exposure guideline levels for selected airborne chemicals. In *Acute Exposure Guideline Levels for Selected Airborne Chemicals*. Washington, D.C.: The National Academies Press. <http://dx.doi.org/10.17226/12770>
- NTP. (1986). NTP Toxicology and Carcinogenesis Studies of Dichloromethane (Methylene Chloride) (CAS No. 75-09-2) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). 306: 1-208.
- O'Neil, MJ. (2013). The Merck index: An encyclopedia of chemicals, drugs, and biologicals. In MJ O'Neil (Ed.), (15th ed.). Cambridge, UK: Royal Society of Chemistry.
- OECD. (2011). SIDS initial assessment profile: Dichloromethane (methylene chloride) [OECD SIDS]. (CoCAM 1, October 10-12, 2011). Paris, France: Organization for Economic Co-operation and Development. <http://webnet.oecd.org/hpv/UI/handler.axd?id=B8EA971C-0C2C-4976-8706-A9A68033DAA0>
- Oehha. (2000). Public health goals for chemicals in drinking water: Dichloromethane (methylene chloride, DCM). Sacramento, CA: California Environmental Protection Agency.
https://oehha.ca.gov/media/downloads/water/chemicals/phg/dcm_0.pdf
- Oehha. (2008a). Acute reference exposure level (REL) and toxicity summary for methylene chloride. Sacramento, CA: Office of Environmental Health Hazard Assessment, State of California Environmental Protection Agency.
http://oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=187

- OEHHA. (2008b). TSD for noncancer RELs - Appendix D.3 Chronic RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines (OEHHA 1999). Sacramento, CA: California Environmental Protection Agency. <http://oehha.ca.gov/media/downloads/crnrr/appendixd3final.pdf>
- Oettingen, WF; Powell, CC; Sharpless, NE; Alford, WC; Pecora, LJ. (1950). Comparative studies of the toxicity and pharmacodynamic action of chlorinated methanes with special reference to their physical and chemical characteristics. *Arch Int Pharmacodyn Ther* 81: 17-34.
- Olin Chemicals. (1977). ENVIRONMENTAL HYGIENE SURVEY OF THE OLIN CHEMICALS BROOK PARK, OHIO PLANT. (OTS: OTS0515276; 8EHQ Num: NA; DCN: 86-870000838; TSCATS RefID: 308130; CIS: NA).
- Olin Corp. (1979). INDUSTRIAL HYGIENE SURVEY CORP PROTECTION AREA WITH COVER LETTER & MEMO. (OTS: OTS0215011; 8EHQ Num: NA; DCN: 878220192; TSCATS RefID: 18805; CIS: NA).
- Olvera-Bello, AE; Estrada-Muñiz, E; Elizondo, G; Vega, L. (2010). Susceptibility to the cytogenetic effects of dichloromethane is related to the glutathione S-transferase theta phenotype. *Toxicol Lett* 199: 218-224. <http://dx.doi.org/10.1016/j.toxlet.2010.09.002>
- Osha. (1990). Methylene Chloride: Method 80 [Website]. <https://www.osha.gov/dts/sltc/methods/organic/org080/org080.html>
- OSHA. (1997a). Final rules: Occupational exposure to methylene chloride. Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration. https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=FEDERAL_REGISTRATION&p_id=13600
- OSHA. (1997b). Occupational exposure to methylene chloride. *Fed Reg* 62: 1493-1619.
- OSHA. (2019). Dichloromethane Sampling Results, 2012-2016 [Database].
- OSHA; NIOSH. (2013). Hazard alert methylene chloride hazards for bathtub refinishers. (DHHS (NIOSH) Publication Number 2013-110). Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration Office of Training and Education. https://www.osha.gov/dts/hazardalerts/methylene_chloride_hazard_alert.html
- Ott, MG; Skory, LK; Holder, BB; Bronson, JM; Williams, PR. (1983a). Health evaluation of employees occupationally exposed to methylene chloride. *Scand J Work Environ Health* 9: 1-38.
- Ott, MG; Skory, LK; Holder, BB; Bronson, JM; Williams, PR. (1983b). Health evaluation of employees occupationally exposed to methylene chloride: Clinical laboratory evaluation. *Scand J Work Environ Health* 9(1): 17-25.
- Park, SK; Lee, MY. (2014). Profiling of the dichloromethane-induced proteome expression changes. *J Environ Biol* 35: 377-382.
- Pegram, RA; Andersen, ME; Warren, SH; Ross, TM; Claxton, LD. (1997). Glutathione S-transferase-mediated mutagenicity of trihalomethanes in *Salmonella typhimurium*: Contrasting results with bromodichloromethane off chloroform. *Toxicol Appl Pharmacol* 144: 183-188. <http://dx.doi.org/10.1006/taap.1997.8123>
- Peijnenburg, W; Eriksson, L; De Groot, A; Sjöström, M; Verboom, H. (1998). The kinetics of reductive dehalogenation of a set of halogenated aliphatic hydrocarbons in anaerobic sediment slurries. *Environ Sci Pollut Res Int* 5: 12-16. <http://dx.doi.org/10.1007/BF02986368>

- Pelch, KE; Bolden, AL; Kwiatkowski, CF. (2019). Environmental Chemicals and Autism: A Scoping Review of the Human and Animal Research. *Environ Health Perspect* 127: 46001.
https://heronet.epa.gov/heronet/index.cfm/reference/download/reference_id/5489075
- Pellizzari, ED; Hartwell, TD; Harris, BS, III; Waddell, RD; Whitaker, DA; Erickson, MD. (1982). Purgeable organic compounds in mother's milk. *Bull Environ Contam Toxicol* 28: 322-328. <http://dx.doi.org/10.1007/BF01608515>
- Peterson, JE. (1978). Modeling the uptake, metabolism and excretion of dichloromethane by man. *Am Ind Hyg Assoc J* 39: 41-47. <http://dx.doi.org/10.1080/0002889778507711>
- Preisser, AM; Budnik, LT; Hampel, E; Baur, X. (2011). Surprises perilous: toxic health hazards for employees unloading fumigated shipping containers. *Sci Total Environ* 409: 3106-3113. <http://dx.doi.org/10.1016/j.scitotenv.2011.04.053>
- Processing Magazine. (2015). Global methylene chloride market value to reach \$892.9m by 2020. <http://www.processingmagazine.com/global-methylene-chloride-market-value-reach-892-9m-2020/>
- Purdue, MP; Stewart, PA; Friesen, MC; Colt, JS; Locke, SJ; Hein, MJ; Waters, MA; Graubard, BI; Davis, F; Ruterbusch, J; Schwartz, K; Chow, WH; Rothman, N; Hofmann, JN. (2016). Occupational exposure to chlorinated solvents and kidney cancer: a case-control study. *Occup Environ Med* 74: 268-274.
- Putz, VR; Johnson, BL; Setzer, JV. (1979). A comparative study of the effects of carbon monoxide and methylene chloride on human performance. *J Environ Pathol Toxicol* 2: 97-112.
- Quinlan, CL; Perevoschikova, IV; Goncalves, RL; Hey-Mogensen, M; Brand, MD. (2013). Chapter 12: The determination and analysis of site-specific rates of mitochondrial reactive oxygen species production. *Methods Enzymol* 526: 189-217.
<http://dx.doi.org/10.1016/B978-0-12-405883-5.00012-0>
- Radican, L; Blair, A; Stewart, P; Wartenberg, D. (2008). Mortality of aircraft maintenance workers exposed to trichloroethylene and other hydrocarbons and chemicals: Extended follow-up. *J Occup Environ Med* 50: 1306-1319.
- Raje, R; Basso, M; Tolen, T; Greening, M. (1988). Evaluation of in vivo mutagenicity of low-dose methylene chloride in mice. *Int J Toxicol* 7: 699-703.
<http://dx.doi.org/10.3109/10915818809019544>
- Ratney, RS; Wegman, DH; Elkins, HB. (1974). In vivo conversion of methylene chloride to carbon monoxide. *Arch Environ Occup Health* 28: 223-226.
- Rayburn, JR; Fisher, WS. (1999). Developmental toxicity of copper chloride, methylene chloride, and 6-aminonicotinamide to embryos of the grass shrimp *Palaemonetes pugio*. *Environ Toxicol Chem* 18: 950-957.
- Rebert, CS; Matteucci, MJ; Pryor, GT. (1989). Acute effects of inhaled dichloromethane on the EEG and sensory-evoked potentials of Fischer-344 rats. *Pharmacol Biochem Behav* 34: 619-629. [http://dx.doi.org/10.1016/0091-3057\(89\)90568-6](http://dx.doi.org/10.1016/0091-3057(89)90568-6)
- Reh, CM; Lushniak, BD. (1990). Health hazard evaluation report no. HETA 87-350-2084, Trailmobile, Inc., Charleston, Illinois. (HETA 87-350-2084). Cincinnati, OH: National Institute for Occupational Safety and Health.
- Rice, D; Barone, S, Jr. (2000). Critical periods of vulnerability for the developing nervous system: Evidence from humans and animal models [Review]. *Environ Health Perspect*

- 108: 511-533.
https://heronet.epa.gov/heronet/index.cfm/reference/download/reference_id/20837
- Riley, EC; Fassett, DW; Sutton, WL. (1966). Methylene chloride vapor in expired air of human subjects. *Am Ind Hyg Assoc J* 27: 341-348.
<http://dx.doi.org/10.1080/00028896609342839>
- Roberts, AL; Lyall, K; Hart, JE; Laden, F; Just, AC; Bobb, JF; Koenen, KC; Ascherio, A; Weisskopf, MG. (2013). Perinatal Air Pollutant Exposures and Autism Spectrum Disorder in the Children of Nurses' Health Study II Participants. *Environ Health Perspect* 121: 978-984.
- Rodriguez-Arnaiz, R. (1998). Biotransformation of several structurally related 2B compounds to reactive metabolites in the somatic w/w+ assay of *Drosophila melanogaster*. *Environ Mol Mutagen* 31: 390-401. [http://dx.doi.org/10.1002/\(SICI\)1098-2280\(1998\)31:4<390::AID-EM12>3.0.CO;2-7](http://dx.doi.org/10.1002/(SICI)1098-2280(1998)31:4<390::AID-EM12>3.0.CO;2-7)
- Roldán-Arjona, T; Pueyo, C. (1993). Mutagenic and lethal effects of halogenated methanes in the Ara test of *Salmonella typhimurium*: Quantitative relationship with chemical reactivity. *Mutagenesis* 8: 127-131. <http://dx.doi.org/10.1093/mutage/8.2.127>
- Rosengren, LE; Kjellstrand, P; Aurell, A; Haglid, KG. (1986). Irreversible effects of dichloromethane on the brain after long term exposure: A quantitative study of DNA and the glial cell marker proteins S-100 and GFA. *Br J Ind Med* 43: 291-299.
- Rossberg, M; Lendle, W; Pfleiderer, G; Togel, A; Torkelson, TR; Beutel, K. (2011). Chloromethanes. In *Ullman's Encyclopedia of Industrial Chemistry* (7 ed.). New York, NY: John Wiley & Sons.
- Ruder, AM; Yiin, JH; Waters, MA; Carreon, T; Hein, MJ; Butler, MA; Calvert, GM; Davis-King, KE; Schulte, PA; Mandel, JS; Morton, RF; Reding, DJ; Rosenman, KD; Stewart, PA; Grp, BCCS. (2013). The Upper Midwest Health Study: gliomas and occupational exposure to chlorinated solvents. *Occup Environ Med* 70: 73-80.
- Russo, J. (2015). Significance of rat mammary tumors for human risk assessment. *Toxicol Pathol* 43: 145-170.
- Samoiloff, MR; Schulz, S; Jordan, Y; Denich, K; Arnott, E. (1980). A rapid simple long-term toxicity assay for aquatic contaminants using the nematode *Panagrellus redivivus*. *Can J Fish Aquat Sci* 37: 1167-1174. <http://dx.doi.org/10.1139/f80-149>
- Sanchez-Fortun, S; Sanz, F; Santa-Maria, A; Ros, JM; De Vicente, ML; Encinas, MT; Vinagre, E; Barahona, MV. (1997). Acute sensitivity of three age classes of *Artemia salina* larvae to seven chlorinated solvents. *Bull Environ Contam Toxicol* 59: 445-451.
<http://dx.doi.org/10.1007/s001289900498>
- Sasaki, YF; Saga, A; Akasaka, M; Ishibashi, S; Yoshida, K; Su, YQ; Matsusaka, N; Tsuda, S. (1998a). Detection in vivo genotoxicity of haloalkanes and haloalkenes carcinogenic to rodents by the alkaline single cell gel electrophoresis (comet) assay in multiple mouse organs. *Mutat Res* 419: 13-20. [http://dx.doi.org/10.1016/S1383-5718\(98\)00114-4](http://dx.doi.org/10.1016/S1383-5718(98)00114-4)
- Sasaki, YF; Saga, A; Akasaka, M; Ishibasi, S; Yoshida, K; Su, QY; Matsusaka, N; Tsuda, S. (1998b). Detection of in vivo genotoxicity of haloalkanes and haloalkenes carcinogenic to rodents by the alkaline single cell gel electrophoresis (comet) assay in multiple mouse organs. *Mutat Res Genet Toxicol Environ Mutagen* 419: 13-20.
[http://dx.doi.org/10.1016/S1383-5718\(98\)00114-4](http://dx.doi.org/10.1016/S1383-5718(98)00114-4)

- Savolainen, H; Kurppa, K; Pfaffli, P; Kivisto, H. (1981). Dose-related effects of dichloromethane on rat brain in short-term inhalation exposure. *Chem Biol Interact* 34: 315-322.
[http://dx.doi.org/10.1016/0009-2797\(81\)90103-4](http://dx.doi.org/10.1016/0009-2797(81)90103-4)
- Savolainen, H; Pfaffli, P; Tengén, M; Vainio, H. (1977). Biochemical and behavioural effects of inhalation exposure to tetrachlorethylene and dichlormethane. *J Neuropathol Exp Neurol* 36: 941-949.
- Sax, SN; Bennett, DH; Chillrud, SN; Kinney, PL; Spengler, JD. (2004). Differences in source emission rates of volatile organic compounds in inner-city residences of New York City and Los Angeles. *J Expo Anal Environ Epidemiol* 14: S95-109.
<http://dx.doi.org/10.1038/sj.jea.7500364>
- Schenk, L; Rauma, M; Fransson, MN; Johanson, G. (2018). Percutaneous absorption of thirty-eight organic solvents in vitro using pig skin. *PLoS ONE* 13: e0205458.
<http://dx.doi.org/10.1371/journal.pone.0205458>
- Schf. (2020). U.S. deaths from methylene chloride.
- Schwarzenbach, RP; Gschwend, PM; Imboden, DM. (2003). *Environmental Organic Chemistry* (2 ed.). Hoboken, New Jersey: John Wiley & Sons.
- Schwetz, BA; Leong, BKJ; Gehring, PJ. (1975). The effect of maternally inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. *Toxicol Appl Pharmacol* 32: 84-96.
[http://dx.doi.org/10.1016/0041-008X\(75\)90197-0](http://dx.doi.org/10.1016/0041-008X(75)90197-0)
- Seidler, A; Möhner, M; Berger, J; Mester, B; Deeg, E; Elsner, G; Nieters, A; Becker, N. (2007). Solvent exposure and malignant lymphoma: A population-based case-control study in Germany. *J Occup Med Toxicol* 2: 2.
- Serota, DG; Thakur, AK; Ulland, BM; Kirschman, JC; Brown, NM; Coots, RH; Morgareidge, K. (1986a). A two-year drinking-water study of dichloromethane in rodents: I. Rats. *Food Chem Toxicol* 24: 951-958. [http://dx.doi.org/10.1016/0278-6915\(86\)90323-6](http://dx.doi.org/10.1016/0278-6915(86)90323-6)
- Serota, DG; Thakur, AK; Ulland, BM; Kirschman, JC; Brown, NM; Coots, RH; Morgareidge, K. (1986b). A two-year drinking-water study of dichloromethane in rodents: II. Mice. *Food Chem Toxicol* 24: 959-963. [http://dx.doi.org/10.1016/0278-6915\(86\)90324-8](http://dx.doi.org/10.1016/0278-6915(86)90324-8)
- Sexton, K; Mongin, SJ; Adgate, JL; Pratt, GC; Ramachandran, G; Stock, TH; Morandi, MT. (2007). Estimating volatile organic compound concentrations in selected microenvironments using time-activity and personal exposure data. *J Toxicol Environ Health A* 70: 465-476. <http://dx.doi.org/10.1080/15287390600870858>
- Sheldon, T; Richardson, CR; Elliott, BM. (1987). Inactivity of methylene chloride in the mouse bone marrow micronucleus assay. *Mutagenesis* 2: 57-59.
- Shell Oil. (1986). TEN DAY INHALATION TOXICITY STUDY TO INVESTIGATE THE EFFECTS ON RAT AND MOUSE LIVER AND LUNG WITH METHYLENE CHLORIDE. (OTS: OTS0514365; 8EHQ Num: NA; DCN: 86-880000287; TSCATS RefID: 305688; CIS: NA). Shell Oil Co.
- Sheps, DS; Adams, KF, Jr.; Bromberg, PA; Goldstein, GM; O'Neil, JJ; Horstman, D; Koch, G. (1987). Lack of effect of low levels of carboxyhemoglobin on cardiovascular function in patients with ischemic heart disease. *Arch Environ Occup Health* 42: 108-116.
<http://dx.doi.org/10.1080/00039896.1987.9935805>
- Siemiatycki, J, . (1991). Risk factors for cancer in the workplace. In J Siemiatycki (Ed.). Boca Raton, FL: CRC Press.

- Silver, SR; Pinkerton, LE; Fleming, DA; Jones, JH; Allee, S; Luo, L; Bertke, SJ. (2014). Retrospective Cohort Study of a Microelectronics and Business Machine Facility. *Am J Ind Med* 57: 412-424.
- Singh, HB; Salas, LJ; Stiles, RE. (1983). Selected man-made halogenated chemicals in the air and oceanic environment. *J Geophys Res* 88: 3675-3683.
- Soden, KJ. (1993). An evaluation of chronic methylene chloride exposure. *J Occup Med* 35: 282-286.
- Steiman, R; Seiglemurandi, F; Guiraud, P; Benoitguyod, JL. (1995). TESTING OF CHLORINATED SOLVENTS ON MICROFUNGI. *Environ Toxicol Water Qual* 10: 283-285.
- Stewart, RD; Fisher, TN; Hosko, MJ; Peterson, JE; Baretta, ED; Dodd, HC. (1972). Experimental human exposure to methylene chloride. *Arch Environ Occup Health* 25: 342-348. <http://dx.doi.org/10.1080/00039896.1972.10666184>
- Stewart, RD; Hake, CL; Wu, A. (1976). Use of breath analysis to monitor methylene chloride exposure. *Scand J Work Environ Health* 2: 57-70.
- Stull, JO; Thomas, RW; James, LE. (2002). A comparative analysis of glove permeation resistance to paint stripping formulations. *AIHA J* 63: 62-71. [http://dx.doi.org/10.1202/0002-8894\(2002\)063<0062:ACAOGP>2.0.CO;2](http://dx.doi.org/10.1202/0002-8894(2002)063<0062:ACAOGP>2.0.CO;2)
- Suzuki, T; Yanagiba, Y; Suda, M; Wang, RS. (2014). Assessment of the genotoxicity of 1,2-dichloropropane and dichloromethane after individual and co-exposure by inhalation in mice. *J Occup Health* 56: 205-214.
- Tabak, HH; Quave, SA; Mashni, CI; Barth, EF. (1981). Biodegradability studies with organic priority pollutant compounds. *J Water Pollut Control Fed* 53: 1503-1518.
- Talbott, EO; Marshall, LP; Rager, JR; Arena, VC; Sharma, RK; Stacy, SL. (2015). Air toxics and the risk of autism spectrum disorder: the results of a population based case-control study in southwestern Pennsylvania. *Environ Health* 14: 80.
- Taskinen, H; Lindbohm, ML; Hemminki, K. (1986). Spontaneous abortions among women working in the pharmaceutical industry. *Br J Ind Med* 43: 199-205.
- Ten Berge, WF; Zwart, A; Appelman, LM. (1986). Concentration-time mortality response relationship of irritant and systemically acting vapours and gases. *J Hazard Mater* 13: 301-309. [http://dx.doi.org/10.1016/0304-3894\(86\)85003-8](http://dx.doi.org/10.1016/0304-3894(86)85003-8)
- Texaco Inc. (1993). I.h. monit. for pentane, ethyl ether, chloroform, acetone, t-butyl alcohol, carbon tetrachloride, total hydrocarbons, gasoline, isooctane, hexane, methylene chloride & toluene. (OTS: OTS0537774; 8EHQ Num: NA; DCN: 86-930000338; TSCATS RefID: 423786; CIS: NA).
- Thiébaud, H; Merlin, G; Capovilla, MP; Blake, G. (1994). Fate of a volatile chlorinated solvent in indoor aquatic microcosms: Sublethal and static exposure to [¹⁴C]dichloromethane. *Ecotoxicol Environ Saf* 28: 71-81. <http://dx.doi.org/10.1006/eesa.1994.1035>
- Thier, R; Taylor, JB; Pemble, SE; Humphreys, WG; Persmark, M; Ketterer, B; Guengerich, FP. (1993). Expression of mammalian glutathione S-transferase 5-5 in *Salmonella typhimurium* TA1535 leads to base-pair mutations upon exposure to dihalomethanes. *Proc Natl Acad Sci USA* 90: 8576-8580.
- Thier, R; Wiebel, FA; Hinkel, A; Burger, A; Brüning, T; Morgenroth, K; Senge, T; Wilhelm, M; Schulz, TG. (1998). Species differences in the glutathione transferase GSTT1-1 activity towards the model substrates methyl chloride and dichloromethane in liver and kidney. *Arch Toxicol* 72: 622-629. <http://dx.doi.org/10.1007/s002040050552>

- Thilagar, AK; Kumaroo, V. (1983). Induction of chromosome damage by methylene chloride in CHO cells. *DNA Repair* 116: 361-367. [http://dx.doi.org/10.1016/0165-1218\(83\)90074-5](http://dx.doi.org/10.1016/0165-1218(83)90074-5)
- Thomas, AA; Pinkerton, MK; Warden, JA. (1972). Effects of low level dichloromethane exposure on the spontaneous activity of mice. In *Proceedings of the Annual Conference on Environmental Toxicology (3rd) held in Fairborn, Ohio, on 25-27 October 1972* (pp. 223-226). (AMRLTR72130). Wright-Patterson AFB, OH: Aerospace Medical Research Lab. <https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=AD773766>
- TNO (CIVO). (1999). Methylene chloride: Advantages and Drawbacks of Possible Market Restrictions in the EU. In *Methylene chloride: Advantages and drawbacks of possible market restrictions in the EU STB-99-53 Final*. Brussels, Belgium: European Commission. TNO-STB. <http://ec.europa.eu/DocsRoom/documents/13039/attachments/1/translations/en/renditions/native>
- Tomenson, JA. (2011). Update of a cohort mortality study of workers exposed to methylene chloride employed at a plant producing cellulose triacetate film base. *Int Arch Occup Environ Health* 84: 889-897.
- Tomenson, JA; Bonner, SM; Heijne, CG; Farrar, DG; Cummings, TF. (1997). Mortality of workers exposed to methylene chloride employed at a plant producing cellulose triacetate film base. *Occup Environ Med* 54: 470-476.
- Trueman, RW; Ashby, J. (1987). Lack of UDS activity in the livers of mice and rats exposed to dichloromethane. *Environ Mol Mutagen* 10: 189-195. <http://dx.doi.org/10.1002/em.2850100209>
- Tsai, KP; Chen, CY. (2007). An algal toxicity database of organic toxicants derived by a closed-system technique. *Environ Toxicol Chem* 26: 1931-1939. <http://dx.doi.org/10.1897/06-612R.1>
- U.S. Coast Guard. (1984). The chemical hazards response information system (CHRIS) hazardous chemical data. Washington, DC: Department of Transportation.
- U.S. EPA. (1987). Household solvent products: A national usage survey. (EPA-OTS 560/5-87-005). Washington, DC: Office of Toxic Substances, Office of Pesticides and Toxic Substances. <https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=PB88132881>
- U.S. EPA. (1992). Guidelines for exposure assessment. *Federal Register* 57(104):22888-22938 [EPA Report]. In *Guidelines for exposure assessment*. (EPA/600/Z-92/001). Washington, DC. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263>
- U.S. EPA. (2000). Methylene chloride (dichloromethane). <https://www.epa.gov/sites/production/files/2016-09/documents/methylene-chloride.pdf>
- U.S. EPA. (2007). Technical support document for proposed rule: National emission standards for hazardous air pollutants: Paint stripping and miscellaneous surface coating operations at area sources [EPA Report] (pp. 52958-52982). (EPA-HQ-OAR-2005-0526; FRL-8466-6). Research Triangle Park, NC: OAQPS/Sector Policies and Programs Division. <https://www.regulations.gov/document?D=EPA-HQ-OAR-2005-0526-0001>
- U.S. EPA. (2011). Toxicological review of dichloromethane (methylene chloride) (CASRN 75-09-2): In support of summary information on the Integrated Risk Information System (IRIS) [EPA Report]. (EPA/635/R-10/003F). Washington, D.C. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0070tr.pdf

- U.S. EPA. (2012). Estimation Programs Interface Suite™ for Microsoft® Windows, v 4.11 [Computer Program]. Washington, DC. Retrieved from <https://www.epa.gov/tsca-screening-tools/epi-suitetm-estimation-program-interface>
- U.S. EPA. (2014). TSCA work plan chemical risk assessment, methylene chloride: paint stripping use. (740-R1-4003). Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. https://www.epa.gov/sites/production/files/2015-09/documents/dcm_opptworkplanra_final.pdf
- U.S. EPA. (2015). Update of human health ambient water quality criteria: Methylene Chloride 75-09-2. (EPA 820-R-15-057). Washington D.C.: Office of Water, Office of Science and Technology. <https://www.federalregister.gov/documents/2014/05/13/2014-10963/updated-national-recommended-water-quality-criteria-for-the-protection-of-human-health>
- U.S. EPA. (2016). Public database 2016 chemical data reporting (May 2017 release). Washington, DC: US Environmental Protection Agency, Office of Pollution Prevention and Toxics. Retrieved from <https://www.epa.gov/chemical-data-reporting>
- U.S. EPA. (2017a). Methylene chloride (DCM) (CASRN: 75-09-2) bibliography: Supplemental file for the TSCA Scope Document [EPA Report]. https://www.epa.gov/sites/production/files/2017-06/documents/dcm_comp_bib.pdf
- U.S. EPA. (2017b). Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: Methylene Chloride. Available online at <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0003>
- U.S. EPA. (2017c). Scope of the risk evaluation for methylene chloride (dichloromethane, DCM). CASRN: 75-09-2 [EPA Report]. (EPA 740-R1-7006). https://www.epa.gov/sites/production/files/2017-06/documents/mecl_scope_06-22-17.pdf
- U.S. EPA. (2017d). Strategy for conducting literature searches for methylene chloride (DCM): Supplemental document to the TSCA Scope Document. CASRN: 75-09-2 [EPA Report]. https://www.epa.gov/sites/production/files/2017-06/documents/dcm_lit_search_strategy_053017.pdf
- U.S. EPA. (2017e). Toxics Release Inventory (TRI), reporting year 2015. Retrieved from <https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools>
- U.S. EPA. (2017f). Toxics Release Inventory (TRI), reporting year 2016. Retrieved from <https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools>
- U.S. EPA. (2017g). Use and market profile for methylene chloride. Washington, D.C.: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention, Chemistry, Economics, and Sustainable Strategies Division, Economic and Policy Analysis Branch. <https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0742-0062>
- U.S. EPA. (2018a). Application of systematic review in TSCA risk evaluations. (740-P1-8001). Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. https://www.epa.gov/sites/production/files/2018-06/documents/final_application_of_sr_in_tsca_05-31-18.pdf
- U.S. EPA. (2018b). Application of systematic review in TSCA risk evaluations: DRAFT Version 1.0. (740P18001). Washington, D.C.: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention.
- U.S. EPA. (2018c). Problem formulation of the risk evaluation for methylene chloride (dichloromethane, DCM). (EPA-740-R1-7016). Washington, DC: Office of Chemical

- Safety and Pollution Prevention, United States Environmental Protection Agency.
https://www.epa.gov/sites/production/files/2018-06/documents/mecl_problem_formulation_05-31-18.pdf
- U.S. EPA. (2019a). Consumer Exposure Model (CEM) 2.1 User Guide. (EPA Contract # EP-W-12-010). Washington, DC.
- U.S. EPA. (2019b). Consumer Exposure Model (CEM) 2.1 User Guide - Appendices. (EPA Contract # EP-W-12-010). Washington, DC.
- Ukai, H; Okamoto, S; Takada, S; Inui, S; Kawai, T; Higashikawa, K; Ikeda, M. (1998). Monitoring of occupational exposure to dichloromethane by diffusive vapor sampling and urinalysis. *Int Arch Occup Environ Health* 71: 397-404.
<http://dx.doi.org/10.1007/s004200050298>
- Unocal Corporation. (1986). MEMORANDUM REGARDING UNOCAL TEMPORARY OCCUPATIONAL EXPOSURE LIMIT (TOEL) FOR DICHLOROMETHANE WITH ATTACHMENTS AND COVER LETTER DATED 110987. (OTS: OTS0513971; 8EHQ Num: NA; DCN: 86-880000080; TSCATS RefID: 304976; CIS: NA).
- Uraga-Tovar, DI; Domínguez-López, ML; Madera-Sandoval, RL; Nájera-Martínez, M; García-Latorre, E; Vega-López, A. (2014). Generation of oxyradicals (O₂ and H₂O₂), mitochondrial activity and induction of apoptosis of PBMC of *Cyprinus carpio carpio* treated in vivo with halomethanes and with recombinant HSP60 kDa and with LPS of *Klebsiella pneumoniae*. *Immunopharmacol Immunotoxicol* 36: 329-340.
<http://dx.doi.org/10.3109/08923973.2014.947034>
- USGS. (2003). A national survey of methyl tert-butyl ether and other volatile organic compounds in drinking-water sources: Results of the random survey. Reston, VA: U.S. Department of the Interior, U.S. Geological Survey.
<https://pubs.er.usgs.gov/publication/wri024079>
- USGS. (2013). Federal Standards and Procedures for the National Watershed Boundary Dataset (WBD): Techniques and Methods 11–A3 (4th ed., pp. 63). U.S. Geological Survey and U.S. Department of Agriculture, Natural Resources Conservation Service.
<https://pubs.usgs.gov/tm/11/a3/>
- Van Winkle, MR; Scheff, PA. (2001). Volatile organic compounds, polycyclic aromatic hydrocarbons and elements in the air of ten urban homes. *Indoor Air* 11: 49-64.
<http://dx.doi.org/10.1034/j.1600-0668.2001.011001049.x>
- Vandervort, R; Polakoff, PL. (1973). Health hazard evaluation report no. HHE 72-84-31, Dunham-Bush, Incorporated, West Hartford, Connecticut, Part 2. (HHE 72-84-31). Cincinnati, OH: National Institute for Occupational Safety and Health.
- Vizcaya, D; Christensen, KY; Lavoue, J; Siemiatycki, J. (2013). Risk of lung cancer associated with six types of chlorinated solvents: results from two case-control studies in Montreal, Canada. *Occup Environ Med* 70: 81-85.
- von Ehrenstein, OS; Aralis, H; Cockburn, M; Ritz, B. (2014). In Utero Exposure to Toxic Air Pollutants and Risk of Childhood Autism. *Epidemiology* 25: 851-858.
- Vulcan Chemicals. (1991). LETTER FROM VULCAN CHEMICALS TO USEPA SUBMITTING ENCLOSED INDUSTRIAL HYGIENE MONITORING REPORT ON METHYLENE CHLORIDE WITH ATTACHMENT. (OTS: OTS0529788; 8EHQ Num: NA; DCN: 86-910000869; TSCATS RefID: 417033; CIS: NA).
- Wallace, L; Nelson, W; Ziegenfus, R; Pellizzari, E; Michael, L; Whitmore, R; Zelon, H; Hartwell, T; Perritt, R; Westerdahl, D. (1991). The Los Angeles TEAM Study: personal

- exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. *J Expo Anal Environ Epidemiol* 1: 157-192.
- Wang, R; Zhang, Y; Lan, Q; Holford, TR; Leaderer, B; Zahm, SH; Boyle, P; Dosemeci, M; Rothman, N; Zhu, Y; Qin, Q; Zheng, T. (2009). Occupational exposure to solvents and risk of non-Hodgkin lymphoma in Connecticut women. *Am J Epidemiol* 169: 176-185.
- Warbrick, EV; Kilgour, JD; Dearman, RJ; Kimber, I; Dugard, PH. (2003). Inhalation exposure to methylene chloride does not induce systemic immunotoxicity in rats. *J Toxicol Environ Health A* 66: 1207-1219. <http://dx.doi.org/10.1080/15287390306410>
- Warholm, M; Alexandrie, AK; Hogberg, J; Sigvardsson, K; Rannug, A. (1994). Polymorphic distribution of glutathione transferase activity with methyl chloride in human blood. *Pharmacogenetics* 4: 307-311.
- Watanabe, K; Liberman, RG; Skipper, PL; Tannenbaum, SR; Guengerich, FP. (2007). Analysis of DNA adducts formed in vivo in rats and mice from 1,2-dibromoethane, 1,2-dichloroethane, dibromomethane, and dichloromethane using HPLC/accelerator mass spectrometry and relevance to risk estimates. *Chem Res Toxicol* 20: 1594-1600. <http://dx.doi.org/10.1021/tx700125p>
- Weinstein, RS; Boyd, DD; Back, KC. (1972). Effects of continuous inhalation of dichloromethane in the mouse: morphologic and functional observations. *Toxicol Appl Pharmacol* 23: 660-679. [http://dx.doi.org/10.1016/0041-008X\(72\)90107-X](http://dx.doi.org/10.1016/0041-008X(72)90107-X)
- Wells, GG; Waldron, HA. (1984). Methylene chloride burns. *Br J Ind Med* 41: 420.
- Wells, VE; Schrader, SM; McCammon, CS; Ward, EM; Turner, TW; Thun, MJ; Halperin, WE. (1989). Letter to the editor: Cluster of oligospermia among four men occupationally exposed to methylene chloride (MeCl) [Letter]. *Reprod Toxicol* 3: 281-282. [http://dx.doi.org/10.1016/0890-6238\(89\)90025-7](http://dx.doi.org/10.1016/0890-6238(89)90025-7)
- Westbrook-Collins, B; Allen, JW; Sharief, Y; Campbell, J. (1990). Further evidence that dichloromethane does not induce chromosome damage. *J Appl Toxicol* 10: 79-81. <http://dx.doi.org/10.1002/jat.2550100203>
- WHO. (1996a). Environmental health criteria 164: Methylene chloride, 2nd ed. Geneva, Switzerland.
- WHO. (1996b). Methylene chloride (second edition).
- WHO. (2000). Air quality guidelines for Europe (2nd ed.). Copenhagen, Denmark: World Health Organization, Regional Office for Europe. <http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/pre2009/air-quality-guidelines-for-europe>
- Wilson, JEH. (1998). Developmental Arrest in Grass Shrimp Embryos Exposed to Selected Toxicants. 60-75.
- Windham, GC; Zhang, L; Gunier, R; Croen, LA; Grether, JK. (2006). Autism spectrum disorders in relation to distribution of hazardous air pollutants in the San Francisco Bay area. *Environ Health Perspect* 114: 1438-1444.
- Winneke, G. (1974). Behavioral effects of methylene chloride and carbon monoxide as assessed by sensory and psychomotor performance. In C Xintaras; BL Johnson; I De Groot (Eds.), *Behavioral toxicology: Early detection of occupational hazards* (pp. 130-144). Cincinnati, OH: U.S. Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health.

- Winneke, G; Fodor, GG. (1976). Dichloromethane produces narcotic effect. *Occup Health Saf* 45: 34-35.
https://heronet.epa.gov/heronet/index.cfm/reference/download/reference_id/29075
- Wu, S; Zhang, H; Yu, X; Qiu, L. (2014). Toxicological Responses of *Chlorella vulgaris* to Dichloromethane and Dichloroethane. *Environ Eng Sci* 31: 9-17.
<http://dx.doi.org/10.1089/ees.2013.0038>
- Yamamoto, K; Fukushima, M; Kakutani, N; Kuroda, K. (1997). Volatile organic compounds in urban rivers and their estuaries in Osaka, Japan. *Environ Pollut* 95: 135-143.
[http://dx.doi.org/10.1016/S0269-7491\(96\)00100-5](http://dx.doi.org/10.1016/S0269-7491(96)00100-5)
- Yang, F; Zhang, J; Chu, W; Yin, D; Templeton, MR. (2014). Haloactamides versus halomethanes formation and toxicity in chloraminated drinking water. *J Hazard Mater* 274: 156-163. <http://dx.doi.org/10.1016/j.jhazmat.2014.04.008>
- Zeiger, E. (1990). Mutagenicity of 42 chemicals in *Salmonella*. *Environ Mol Mutagen* 16: 32-54.
<http://dx.doi.org/10.1002/em.2850160504>
- Zeljezic, D; Mladinic, M; Kopjar, N; Radulovic, AH. (2016). Evaluation of genome damage in subjects occupationally exposed to possible carcinogens. *Toxicol Ind Health* 32: 1570-1580.
<http://dx.doi.org/10.1177/0748233714568478>

APPENDICES

Appendix A REGULATORY HISTORY

A.1 Federal Laws and Regulations

Table Apx A-1. Federal Laws and Regulations

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
EPA Regulations		
TSCA – Section 6(a)	If EPA evaluates the risk of a chemical substance, in accordance with TSCA Section 6(b)(A), and concludes that the manufacture (including import), processing, distribution in commerce, disposal of such chemical substance, or any combination of these activities, presents an unreasonable risk of injury to human health or the environment, then EPA shall, by rule, take one or more of the actions described in TSCA Section 6(a)(1)-(7) to ensure the chemical substance no longer presents an unreasonable risk.	Prohibits the manufacture (including import), processing, and distribution in commerce of methylene chloride for consumer paint and coating removal, including distribution to and by retailers; requiring manufacturers (including importers), processors, and distributors, except for retailers, of methylene chloride for any use to provide downstream notification of these prohibitions; and requiring recordkeeping 40 CFR 751.1, effective as of May 28, 2019.
TSCA – Section 6(b)	Directs EPA to promulgate regulations to establish processes for prioritizing chemical substances and conducting risk evaluations on priority chemicals substances. In the meantime, EPA was required to identify and begin risk evaluations on	Methylene chloride is one of the 10 chemical substances on the initial list to be evaluated for unreasonable risk of injury to health or the environment (81 FR 91927 , December 19, 2016).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	10 chemical substances drawn from the 2014 update of the TSCA Work Plan for Chemical Assessments.	
TSCA – Section 8(a)	The TSCA section 8(a) CDR Rule requires manufacturers (including importers) to give EPA basic exposure-related information on the types, quantities and uses of chemical substances produced domestically and imported into the U.S.	Methylene chloride manufacturing (including importing), processing, and use information is reported under the CDR rule (76 FR 50816 , August 16, 2011).
TSCA – Section 8(b)	EPA must compile, keep current and publish a list (the TSCA Inventory) of each chemical substance manufactured, processed or imported in the U.S..	Methylene chloride was on the initial TSCA Inventory and therefore was not subject to EPA’s new chemicals review process under TSCA section 5 (60 FR 16309 , March 29, 1995).
TSCA – Section 8(d)	Provides EPA with authority to issue rules requiring producers, importers, and (if specified) processors of a chemical substance or mixture to submit lists and/or copies of ongoing and completed, unpublished health and safety studies.	One submission received in 2001 (U.S. EPA, Chemical Data Access Tool. Accessed April 24, 2017).
TSCA – Section 8(e)	Manufacturers (including importers), processors, and distributors must immediately notify EPA if they obtain information that supports the conclusion that a chemical substance or mixture presents a substantial risk of injury to health or the environment.	Sixteen submissions received 1992-1994 (U.S. EPA, ChemView . Accessed April 24, 2017).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
TSCA – Section 4	Provides EPA with authority to issue rules and orders requiring manufacturers (including importers) and processors to test chemical substances and mixtures.	Five chemical data from test rules (Section 4) from 1974 and (U.S. EPA, ChemView . Accessed April 24, 2017).
Emergency Planning and Community Right-to-Know Act (EPCRA) – Section 313	Requires annual reporting from facilities in specific industry sectors that employ 10 or more full-time equivalent employees and that manufacture, process or otherwise use a TRI-listed chemical in quantities above threshold levels. A facility that meets reporting requirements must submit a reporting form for each chemical for which it triggered reporting, providing data across a variety of categories, including activities and uses of the chemical, releases and other waste management (e.g., quantities recycled, treated, combusted) and pollution prevention activities (under section 6607 of the Pollution Prevention Act). These data include on- and off-site data as well as multimedia data (i.e., air, land and water).	Methylene chloride is a listed substance subject to reporting requirements under 40 CFR 372.65 effective as of January 01, 1987.
Federal Food, Drug, and Cosmetic Act (FFDCA) –Section 408	FFDCA governs the allowable residues of pesticides in food. Section 408 of the FFDCA provides EPA with the authority to set tolerances (rules that establish maximum allowable residue limits), or	Methylene chloride was registered as an antimicrobial, conventional chemical in 1974. In 1998, EPA removed methylene chloride from its list of pesticide product inert ingredients that are currently used in pesticide products (63)

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	<p>exemptions from the requirement of a tolerance, for pesticide residues (including inert ingredients) on food. Prior to issuing a tolerance or exemption from tolerance, EPA must determine that the pesticide residues permitted under the action are “safe.” Section 408(b) of the FFDCA defines “safe” to mean a reasonable certainty that no harm will result from aggregate, nonoccupational exposures to the pesticide. Pesticide tolerances or exemptions from tolerance that do not meet the FFDCA safety standard are subject to revocation under FFDCA section 408(d) or (e). In the absence of a tolerance or an exemption from tolerance, a food containing a pesticide residue is considered adulterated and may not be distributed in interstate commerce.</p>	<p>FR 34384). The tolerance exemptions for methylene chloride were revoked in 2002 (67 FR 16027, April 4, 2002).</p>
CAA – Section 112(b)	<p>Defines the original list of 189 HAPs. Under 112(c) of the CAA, EPA must identify and list source categories that emit HAP and then set emission standards for those listed source categories under CAA section 112(d). CAA section 112(b)(3)(A) specifies that any person may petition the Administrator to modify the list of HAP by adding or</p>	<p>Methylene chloride is listed as a HAP (42 U.S. Code section 7412) and is considered an “urban air toxic” (CAA Section 112(k)).</p>

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	deleting a substance. Since 1990, EPA has removed two pollutants from the original list leaving 187 at present.	
CAA – Section 112(d)	Directs EPA to establish, by rule, National Emission Standards for Hazardous Air Pollutants (NESHAPs) for each category or subcategory of listed major sources and area sources of HAPs (listed pursuant to Section 112(c)). The standards must require the maximum degree of emission reduction that the EPA determines is achievable by each particular source category. This is generally referred to as maximum achievable control technology (MACT).	<p>There are a number of source-specific NESHAPs for methylene chloride, including:</p> <ul style="list-style-type: none"> • Foam production and fabrication process (68 FR 18062, April 14, 2003; 72 FR 38864, July 16, 2007; 73 FR 15923, March 26, 2008; 79 FR 48073, August 15, 2014). • Aerospace (60 FR 45948, September 1, 1995). • Boat manufacturing (66 FR 44218, August 22, 2001). • Chemical manufacturing industry (agricultural chemicals and pesticides, cyclic crude and intermediate production, industrial inorganic chemicals, industrial and miscellaneous organic chemicals, inorganic pigments, plastic materials and resins, pharmaceutical production, synthetic rubber) (74 FR 56008, October 29, 2009). • Fabric printing, coating and dyeing (68 FR 32172, May 29, 2003). • Halogenated Solvent Cleaning (72 FR 25138, May 3, 2007). • Miscellaneous organic chemical production and processes (MON) (68 FR 63852, November 10, 2003). • Paint and allied products manufacturing (area sources)

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
		<p>(74 FR 63504, December 3, 2009).</p> <ul style="list-style-type: none"> • Paint stripping and miscellaneous surface coating operations (area sources) (73 FR 1738, January 9, 2008). • Paper and other web surface coating (67 FR 72330, December 4, 2002). • Pesticide active ingredient production (64 FR 33550, June 23, 1999; 67 FR 38200, June 3, 2002). • Pharmaceutical production (63 FR 50280, September 21, 1998). • POTW (64 FR 57572, October 26, 1999). • Reciprocating Internal Combustion Engines (RICE) (75 FR 51570, August 20, 2010). • Reinforced plastic composites production (68 FR 19375, April 21, 2003). • Wood preserving (area sources) (72 FR 38864, July 16, 2007).
CAA sections 112(d) and 112(f)	Risk and technology review (RTR) of section 112(d) MACT standards. Section 112(f)(2) requires EPA to conduct risk assessments for each source category subject to section 112(d) MACT standards, and to determine if additional standards are needed to reduce remaining risks. Section 112(d)(6) requires EPA to review and revise the MACT standards,	EPA has promulgated a number of RTR NESHAP (e.g., the RTR NESHAP for Halogenated Solvent Cleaning (72 FR 25138 ; May 3, 2007) and will do so, as required, for the remaining source categories with NESHAP.

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	as necessary, taking into account developments in practices, processes and control technologies.	
CAA – Section 612	Under Section 612 of the CAA, EPA’s Significant New Alternatives Policy (SNAP) program reviews substitutes for ozone-depleting substances within a comparative risk framework. EPA publishes lists of acceptable and unacceptable alternatives. A determination that an alternative is unacceptable, or acceptable only with conditions, is made through rulemaking.	Under the SNAP program, EPA listed methylene chloride as an acceptable substitute in multiple industrial end-uses, including as a blowing agent in polyurethane foam, in cleaning solvents, in aerosol solvents and in adhesives and coatings (59 FR 13044, March 18, 1994). In 2016, methylene chloride was listed as an unacceptable substitute for use as a blowing agent in the production of flexible polyurethane foam (81 FR 86778 , December 1, 2016).
CWA – Section 301(b), 304(b), 306, and 307(b)	Requires establishment of Effluent Limitations Guidelines and Standards for conventional, toxic, and nonconventional pollutants. For toxic and non-conventional pollutants, EPA identifies the best available technology that is economically achievable for that industry after considering statutorily prescribed factors and sets regulatory requirements based on the performance of that technology.	Methylene chloride is designated as a toxic pollutant under section 307(a)(1) of the CWA and as such is subject to effluent limitations. Under CWA section 304, methylene chloride is included in the list of total toxic organics (TTO) (40 CFR 413.02(i)).
CWA – Section 307(a)	Establishes a list of toxic pollutants or combination of pollutants under the CWA. The statute specifies a list of families of toxic pollutants also listed in the CFR at 40	

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	<p>CFR Part 401.15. The “priority pollutants” specified by those families are listed in 40 CFR Part 423 Appendix A. These are pollutants for which best available technology effluent limitations must be established on either a national basis through rules (Sections 301(b), 304(b), 307(b), 306) or on a case-by-case best professional judgement basis in NPDES permits, see Section 402(a)(1)(B).</p>	
SDWA – Section 1412	<p>Requires EPA to publish non-enforceable maximum contaminant level goals (MCLGs) for contaminants which 1. may have an adverse effect on the health of persons; 2. are known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern; and 3. in the sole judgement of the Administrator, regulation of the contaminant presents a meaningful opportunity for health risk reductions for persons served by public water systems. When EPA publishes an MCLG, EPA must also promulgate a National Primary Drinking Water Regulation (NPDWR) which includes either an enforceable maximum</p>	<p>Methylene chloride is subject to NPDWR under the SDWA with a MCLG of zero and an enforceable MCL of 0.005 mg/L or 5 ppb (40 CFR part 151).</p>

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	contaminant level (MCL), or a required treatment technique. Public water systems are required to comply with NPDWRs.	
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) – Sections 102(a) and 103	Authorizes EPA to promulgate regulations designating as hazardous substances those substances which, when released into the environment, may present substantial danger to the public health or welfare or the environment. EPA must also promulgate regulations establishing the quantity of any hazardous substance the release of which must be reported under Section 103. Section 103 requires persons in charge of vessels or facilities to report to the National Response Center if they have knowledge of a release of a hazardous substance above the reportable quantity threshold.	Methylene chloride is a hazardous substance under CERCLA. Releases of methylene chloride in excess of 1,000 pounds must be reported (40 CFR 302.4).
RCRA – Section 3001	Directs EPA to develop and promulgate criteria for identifying the characteristics of hazardous waste, and for listing hazardous waste, taking into account toxicity, persistence, and degradability in nature, potential for accumulation in tissue and other related factors such as flammability, corrosiveness, and other hazardous characteristics.	Methylene chloride is included on the list of hazardous wastes pursuant to RCRA 3001. RCRA Hazardous Waste Code: F001, F002, U080; see 40 CFR 261.31, 261.32. In 2013, EPA modified its hazardous waste management regulations to conditionally exclude solvent-contaminated wipes that have been cleaned and reused from the definition of solid waste under RCRA and to

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
		conditionally exclude solvent-contaminated wipes that are disposed from the definition of hazardous waste (78 FR 46448 , July 31, 2013, 40 CFR 261.4(a)(26)).
Other Federal Regulations		
Federal Hazardous Substance Act (FHSA)	Requires precautionary labeling on the immediate container of hazardous household products and allows the Consumer Product Safety Commission (CPSC) to ban certain products that are so dangerous, or the nature of the hazard is such that labeling is not adequate to protect consumers.	Certain household products that contain methylene chloride are hazardous substances required to be labelled under the FHSA (52 FR 34698 , September 14, 1987). In 2016, the Halogenated Solvents Industry Alliance petitioned the CPSC to amend the CPSC's labeling interpretation and policy on those products (81 FR 60298 , September 1, 2016). In 2018, CPSC updated the labelling policy for paint strippers containing methylene chloride (83 FR 12254 , March 21, 2018 and 83 FR 18219 , April 26, 2018)
Hazardous Materials Transportation Act (HMTA)	Section 5103 of the Act directs the Secretary of Transportation to: <ul style="list-style-type: none"> • Designate material (including an explosive, radioactive material, infectious substance, flammable or combustible liquid, solid or gas, toxic, oxidizing or corrosive material, and compressed gas) as hazardous when the Secretary determines that transporting the material in commerce may pose an 	Methylene chloride is listed as a hazardous material with regard to transportation and is subject to regulations prescribing requirements applicable to the shipment and transportation of listed hazardous materials (70 FR 34381 , June 14 2005).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	<p>unreasonable risk to health and safety or property.</p> <ul style="list-style-type: none"> Issue regulations for the safe transportation, including security, of hazardous material in intrastate, interstate and foreign commerce. 	
FFDCA	Provides the Food and Drug Administration (FDA) with authority to oversee the safety of food, drugs and cosmetics.	Methylene chloride is banned by the FDA as an ingredient in all cosmetic products (54 FR 27328 , June 29, 1989).
Occupational Safety and Health Act	Requires employers to provide their workers with a place of employment free from recognized hazards to safety and health, such as exposure to toxic chemicals, excessive noise levels, mechanical dangers, heat or cold stress or unsanitary conditions (29 U.S.C. section 651 et seq.).	In 1997, OSHA revised an existing occupational safety and health standards for methylene chloride, to include an 8-hr TWA PEL of 25 ppm and a 15-minute TWQ STEL of 125 ppm, exposure monitoring, control measures and respiratory protection (29 CFR 1910.1052 App. A).

A.2 State Laws and Regulations

Table_Apx A-2. State Laws and Regulations

State Actions	Description of Action
State PELs	California (PEL of 25 ppm and a STEL of 100) (Cal Code Regs. title 8, section 5155)
State Right-to-Know Acts	Massachusetts (454 Code Mass. Regs. section 21.00), New Jersey (8:59 N.J. Admin. Code section 9.1) and Pennsylvania (34 Pa. Code section 323).
State Drinking Water Standards and Guidelines	Arizona (14 Ariz. Admin. Register 2978, August 1, 2008), California (Cal Code Regs. Title 26, section 22-64444), Delaware (Del. Admin. Code Title 16, section 4462), Connecticut (Conn. Agencies Regs.

State Actions	Description of Action
	<p>section 19-13-B102), Florida (Fla. Admin. Code R. Chap. 62-550), Maine (10 144 Me. Code R. Chap. 231), Massachusetts (310 Code Mass. Regs. section 22.00), Minnesota (Minn R. Chap. 4720), New Jersey (7:10 N.J Admin. Code section 5.2), Pennsylvania (25 Pa. Code section 109.202), Rhode Island (14 R.I. Code R. section 180-003), Texas (30 Tex. Admin. Code section 290.104).</p>
<p>Chemicals of High Concern to Children</p>	<p>Several states have adopted reporting laws for chemicals in children's products that include methylene chloride, including Maine (38 MRSA Chapter 16-D), Minnesota (Minnesota Statutes 116.9401 to 116.9407), Oregon (Toxic-Free Kids Act, Senate Bill 478, 2015), Vermont (18 V.S.A section 1776) and Washington State (WAC 173-334-130).</p>
<p>VOC Regulations for Consumer Products</p>	<p>Many states regulate methylene chloride as a VOC. These regulations may set VOC limits for consumer products and/or ban the sale of certain consumer products as an ingredient and/or impurity. Regulated products vary from state to state, and could include contact and aerosol adhesives, aerosols, electronic cleaners, footwear or leather care products and general degreasers, among other products. California (Title 17, California Code of Regulations, Division 3, Chapter 1, Subchapter 8.5, Articles 1, 2, 3 and 4), Connecticut (R.C.S.A Sections 22a-174-40, 22a-174-41, and 22a-174-44), Delaware (Adm. Code Title 7, 1141), District of Columbia (Rules 20-720, 20-721, 20-735, 20-736, 20-737), Illinois (35 Adm Code 223), Indiana (326 IAC 8-15), Maine (Chapter 152 of the Maine Department of Environmental Protection Regulations), Maryland (COMAR 26.11.32.00 to 26.11.32.26), Michigan (R 336.1660 and R 336. 1661), New Hampshire (Env-A 4100) New Jersey (Title 7, Chapter 27, Subchapter 24), New York (6 CRR-NY III A 235), Rhode Island (Air Pollution Control Regulation No. 31) and Virginia (9VAC5 CHAPTER 45) all have VOC regulations or limits for consumer products. Some of these states also require emissions reporting.</p>
<p>Other</p>	<p>California listed methylene chloride on Proposition 65 (Cal Code Regs. title 27, section 27001) Massachusetts designated methylene chloride as a Higher Hazard Substance which will require reporting starting in 2014 (301 CMR 41.00).</p>

A.3 International Laws and Regulations

Table Apx A-3. Regulatory Actions by other Governments and Tribes

Country/ Organization	Requirements and Restrictions
Canada	Methylene chloride is on the Canadian List of Toxic Substances (CEPA 1999 Schedule 1). Canada required pollution prevention plan implementation for methylene chloride in 2003 for aircraft paint stripping; flexible polyurethane foam blowing; pharmaceuticals and chemical intermediates manufacturing and tablet coating; industrial cleaning; and adhesive formulations. The overall reduction objective of 85% was exceeded (<i>Canada Gazette</i> , Part I, Saturday, February 28, 2004; Vol. 138, No. 9, p. 409).
European Union	In 2010, a restriction of sale and use of paint removers containing 0.1% or more methylene chloride was added to Annex XVII of regulation (EC) No 1907/2006 - REACH (Registration, Evaluation, Authorization and Restriction of Chemicals). The restriction included provisions for individual member states to issue a derogation for professional uses if they have completed proper training and demonstrate they are capable of safely use the paint removers containing methylene chloride (European Chemicals Agency (ECHA) database. Accessed April 18, 2017).
Australia	Methylene chloride was assessed under Human Health Tier II of the Inventory Multi-Tiered Assessment and Prioritisation (IMAP). Uses reported include solvent in paint removers, adhesives, detergents, print developing, aerosol propellants (products not specified), cold tank degreasing and metal cleaning, as well as uses in waterproof membranes, in urethane foam and plastic manufacturing, and as an extraction solvent for spices, caffeine and hops (NICNAS, 2017, <i>Human Health Tier II assessment for Methane, dichloro-</i> . Accessed April 18, 2017).
Japan	Methylene chloride is regulated in Japan under the following legislation: Act on the Evaluation of Chemical Substances and Regulation of Their Manufacture, etc. (Chemical Substances Control Law; CSCL) <ul style="list-style-type: none"> • Act on Confirmation, etc. of Release Amounts of Specific Chemical Substances in the Environment and Promotion of Improvements to the Management Thereof • Industrial Safety and Health Act (ISHA) • Air Pollution Control Law • Water Pollution Control Law • Soil Contamination Countermeasures Act

Country/ Organization	Requirements and Restrictions
	(National Institute of Technology and Evaluation [NITE] Chemical Risk Information Platform [CHIRP]. Accessed April 17, 2017).
Basel Convention	Halogenated organic solvents (Y41) are listed as a category of waste under the Basel Convention. Although the U.S. is not currently a party to the Basel Convention, this treaty still affects U.S. importers and exporters.
OECD Control of Transboundary Movements of Wastes Destined for Recovery Operations	Halogenated organic solvents (A3150) are listed as a category of waste subject to The Amber Control Procedure under Council Decision C (2001) 107/Final.
Australia, Austria, Belgium, Canada, Denmark, EU, Finland, France, Germany, Hungary, Ireland, Israel, Japan, Latvia New Zealand, People's Republic of China, Poland, Singapore, South Korea, Spain, Sweden, Switzerland, U.K.	OES for methylene chloride (GESTIS International limit values for chemical agents (Occupational exposure limits, OELs) database. Accessed April 18, 2017).

Appendix B LIST OF SUPPLEMENTAL DOCUMENTS

List of supplemental documents:

- a. **Associated Systematic Review Data Quality Evaluation and Data Extraction Documents** – Provides additional detail and information on individual study evaluations and data extractions including criteria and scoring results.
 - a. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction Tables for Environmental Fate and Transport Studies* ([EPA, 2019e](#)).
 - b. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Physical Chemical Properties Studies* ([EPA, 2019f](#))
 - c. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data* ([EPA, 2019d](#))
 - d. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Common Sources* ([EPA, 2019c](#))
 - e. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation for Data Sources on Consumer and Environmental Exposure* ([EPA, 2019q](#))
 - f. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction Tables for Consumer and Environmental Exposure Studies* ([EPA, 2019p](#))
 - g. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies* ([EPA, 2019r](#))
 - h. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal and In Vitro Studies* ([EPA, 2019u](#))
 - i. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Epidemiological Studies* ([EPA, 2019s](#))
 - j. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Human Controlled Experiments* ([EPA, 2019t](#))

- k. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies* ([EPA, 2019a](#))
- l. *Risk Evaluation for Methylene Chloride, Systematic Review Supplemental File: Data Extraction Tables for Human Health Hazard Studies* ([EPA, 2019o](#))
- b. Associated **Supplemental Information Documents** – Provides additional details and information on exposure, hazard and risk assessments.
 - a. *Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment* ([EPA, 2019g](#))
This document provides additional details and information on the exposure assessment and analyses including modeling inputs and outputs.
 - b. *Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Input Parameters* ([EPA, 2019j](#))
 - c. *Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Outputs* ([EPA, 2019j](#))
 - d. *Risk Evaluation for Methylene Chloride, Supplemental Information on Surface Water Exposure Assessment* ([EPA, 2019k](#))
 - e. *Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment* ([EPA, 2019b](#))
This document provides additional details and information on the environmental release and occupational exposure assessment, including process information, estimates of number of sites and workers, summary of monitoring data, and exposure modeling equations, inputs and outputs.
 - f. *Risk Evaluation for Methylene Chloride, Supplemental File: Methylene Chloride Benchmark Dose and PBPK Modeling* ([EPA, 2019h](#))
This document provides details on the modeling used to estimate the PODs for the human health chronic non-cancer and cancer endpoints.
 - g. *Risk Evaluation for Methylene Chloride, Supplemental Information Risk Calculator for Occupational Exposures* ([EPA, 2019n](#))
 - h. *Risk Evaluation for Methylene Chloride, Supplemental Information Risk Calculator for Consumer Inhalation Exposures* ([EPA, 2019m](#))
 - i. *Risk Evaluation for Methylene Chloride, Supplemental Information Risk Calculator for Consumer Dermal Exposures* ([EPA, 2019l](#))

Appendix C FATE AND TRANSPORT

EPI Suite™ Model Inputs

To set up EPI Suite™ for estimating fate properties of methylene chloride, methylene chloride was identified using the “Name Lookup” function. The physical-chemical properties were input based on the values in Table 1-1. EPI Suite™ was run using default settings (i.e., no other parameters were changed or input).

The screenshot displays the EPI Suite software interface. The title bar reads "EPI Suite". The menu bar includes File, Edit, Functions, Batch Mode, Show Structure, Output, Fugacity, STP, and Help. The main window title is "EPI Suite - Welcome Screen".

On the left sidebar, there is a vertical list of model selection buttons: ADPWIN, KOWWIN, BIOWIN, MPBPVP, WSKOW, WATERNT, HENRYWIN, KOAWIN, KOCWIN, BCFBAF, HYDROWIN, BioHCwin, DERMWIN, ECOSAR, and EPI Links.

The main input area contains the following fields and values:

- Input CAS #: 000075-09-2
- Input Smiles: ClCCl
- Input Chem Name: Methane, dichloro-
- Name Lookup: (checked)
- Henry LC: 0.00325 atm-m³/mole
- Melting Point: -95 Celsius
- Boiling Point: 39.7 Celsius
- Water Solubility: 13000 mg/L
- Vapor Pressure: 435 mm Hg
- Log Kow: 1.25
- Water Depth: 1 (River) / 1 (Lake) meters
- Wind Velocity: 5 (River) / 0.5 (Lake) meters/sec
- Current Velocity: 1 (River) / 0.05 (Lake) meters/sec

On the right, there is a chemical structure diagram of methylene chloride (Cl-CH₂-Cl).

At the bottom, there is a text box with the following information:

The Estimation Programs Interface (EPI) Suite™ was developed by the US Environmental Protection Agency's Office of Pollution Prevention and Toxics and Syracuse Research Corporation (SRC). It is a screening-level tool, intended for use in applications such as to quickly screen chemicals for release potential and "bin" chemicals by priority for future work. Estimated values should not be used when experimental (measured) values are available.

EPI Suite™ cannot be used for all chemical substances. The intended application domain is organic chemicals. Inorganic and organometallic chemicals generally are outside the domain.

Important information on the performance, development and application of EPI Suite™ and the individual programs within it can be found under the Help tab. Copyright 2000-2012 United States Environmental Protection Agency for EPI Suite™ and all component programs except BioHCWIN and KOAWIN.

Figure_Apx C-1. EPI Suite Model Inputs for Estimating Methylene Chloride Fate and Transport Properties

Appendix D RELEASES TO THE ENVIRONMENT

Table_Apx D-1 presents a summary of all information on releases to water available for the assessed scenarios.

Table_Apx D-1. Water Releases Reported in 2016 TRI or DMR for Occupational Exposure Scenarios

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources ^a & Notes
OES: Polyurethane Foam							
PREGIS INNOVATIVE PACKAGING INC	WURLAND	KY	2	250	0.01	Surface Water	2016 TRI
OES: Spot Cleaner							
BOISE STATE UNIVERSITY	BOISE	ID	0.1	250	0.0002	Surface Water	2016 DMR
OES: Manufacturing							
COVESTRO LLC	BAYTOWN	TX	1	350	0.004	Surface Water	2016 TRI
EMERALD PERFORMANCE MATERIALS LLC	HENRY	IL	0.5	350	0.001	Surface Water	2016 TRI
FISHER SCIENTIFIC CO LLC	FAIR LAWN	NJ	2	350	0.01	POTW	2016 TRI
FISHER SCIENTIFIC CO LLC	BRIDGEWATER	NJ	2	350	0.01	POTW	2016 TRI
OLIN BLUE CUBE FREEPORT TX	FREEPORT	TX	58	350	0.2	Non-POTW WWT	2016 TRI
REGIS TECHNOLOGIES INC	MORTON GROVE	IL	2	350	0.01	POTW	2016 TRI
SIGMA-ALDRICH MANUFACTURING LLC	SAINT LOUIS	MO	2	350	0.01	POTW	2016 TRI
VANDERBILT CHEMICALS LLC-MURRAY DIV	MURRAY	KY	0.5	350	0.00	Non-POTW WWT	2016 TRI
E I DUPONT DE NEMOURS - CHAMBERS WORKS	DEEPWATER	NJ	76	350	0.2	Surface Water	2016 DMR
BAYER MATERIALSCIENCE BAYTOWN	BAYTOWN	TX	10	350	0.03	Surface Water	2016 DMR

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources ^a & Notes
INSTITUTE PLANT	INSTITUTE	WV	3	350	0.01	Surface Water	2016 DMR
MPM SILICONES LLC	FRIENDLY	WV	2	350	0.005	Surface Water	2016 DMR
BASF CORPORATION	WEST MEMPHIS	AR	1	350	0.003	Surface Water	2016 DMR
ARKEMA INC	PIFFARD	NY	0.3	350	0.001	Surface Water	2016 DMR
EAGLE US 2 LLC - LAKE CHARLES COMPLEX	LAKE CHARLES	LA	0.2	350	0.001	Surface Water	2016 DMR
BAYER MATERIALSCIENCE	NEW MARTINSVILLE	WV	0.2	350	0.001	Surface Water	2016 DMR
ICL-IP AMERICA INC	GALLIPOLIS FERRY	WV	0.1	350	0.0004	Surface Water	2016 DMR
KEESHAN AND BOST CHEMICAL CO., INC.	MANVEL	TX	0.02	350	0.00005	Surface Water	2016 DMR
INDORAMA VENTURES OLEFINS, LLC	SULPHUR	LA	0.01	350	0.00003	Surface Water	2016 DMR
CHEMTURA NORTH AND SOUTH PLANTS	MORGANTOWN	WV	0.01	350	0.00002	Surface Water	2016 DMR
OES: Repackaging							
CHEMISPHERE CORP	SAINT LOUIS	MO	2	250	0.01	POTW	2016 TRI
HUBBARD-HALL INC	WATERBURY	CT	144	250	1	Non-POTW WWT	2016 TRI
WEBB CHEMICAL SERVICE CORP	MUSKEGON HEIGHTS	MI	98	250	0.4	POTW	2016 TRI
RESEARCH SOLUTIONS GROUP INC	PELHAM	AL	0.09	250	0.0003	Surface Water	2016 DMR
EMD MILLIPORE CORP	CINCINNATI	OH	0.03	250	0.0001	Surface Water	2016 DMR
OES: Processing as a Reactant							
AMVAC CHEMICAL CO	AXIS	AL	213	350	0.6	Non-POTW WWT	2016 TRI
THE DOW CHEMICAL CO	MIDLAND	MI	25	350	0.1	Surface Water	2016 TRI
FMC CORPORATION	MIDDLEPORT	NY	0.1	350	0.0003	Surface Water	2016 DMR

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources ^a & Notes
OES: Processing: Formulation							
ARKEMA INC	CALVERT CITY	KY	31	300	0.1	Surface Water	2016 TRI
MCGEAN-ROHCO INC	LIVONIA	MI	113	300	0.4	POTW	2016 TRI
WM BARR & CO INC	MEMPHIS	TN	0.5	300	0.002	POTW	2016 TRI
BUCKMAN LABORATORIES INC	MEMPHIS	TN	254	300	1	POTW	2016 TRI
EUROFINS MWG OPERON LLC	LOUISVILLE	KY	5,785	300	19	POTW	2016 TRI
SOLVAY - HOUSTON PLANT	HOUSTON	TX	12	300	0.04	Surface Water	2016 DMR
HONEYWELL INTERNATIONAL INC - GEISMAR COMPLEX	GEISMAR	LA	4	300	0.01	Surface Water	2016 DMR
STEPAN CO MILLSDALE ROAD	ELWOOD	IL	2	300	0.01	Surface Water	2016 DMR
ELEMENTIS SPECIALTIES, INC.	CHARLESTON	WV	0.2	300	0.001	Surface Water	2016 DMR
OES: Plastics Manufacturing							
SABIC INNOVATIVE PLASTICS US LLC	BURKVILLE	AL	8	250	0.03	Surface Water	2016 TRI
SABIC INNOVATIVE PLASTICS MT. VERNON, LLC	MOUNT VERNON	IN	28	250	0.1	Surface Water	2016 DMR
SABIC INNOVATIVE PLASTICS US LLC	SELKIRK	NY	9	250	0.03	Surface Water	2016 DMR
EQUISTAR CHEMICALS LP	LA PORTE	TX	9	250	0.03	Surface Water	2016 DMR
CHEMOURS COMPANY FC LLC	WASHINGTON	WV	7	250	0.03	Surface Water	2016 DMR
SHINTECH ADDIS PLANT A	ADDIS	LA	3	250	0.01	Surface Water	2016 DMR
STYROLUTION AMERICA LLC	CHANNAHON	IL	0.2	250	0.001	Surface Water	2016 DMR
DOW CHEMICAL CO DALTON PLANT	DALTON	GA	0.3	250	0.001	Surface Water	2016 DMR
PREGIS INNOVATIVE PACKAGING INC	WURLAND	KY	0.02	250	0.0001	Surface Water	2016 DMR

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources ^a & Notes
OES: CTA Film Manufacturing							
KODAK PARK DIVISION	ROCHESTER	NY	29	250	0.1	Surface Water	2016 DMR
OES: Lithographic Printer Cleaner							
FORMER REXON FACILITY AKA ENJEMS MILLWORKS	WAYNE TWP	NJ	0.001	250	0.000004	Surface Water	2016 DMR
OES: Recycling and Disposal							
JOHNSON MATTHEY	WEST DEPTFORD	NJ	620	250	2	Non-POTW WWT	2016 TRI
CLEAN HARBORS DEER PARK LLC	LA PORTE	TX	522	250	2	Non-POTW WWT	2016 TRI
CLEAN HARBORS EL DORADO LLC	EL DORADO	AR	113	250	0.5	Non-POTW WWT	2016 TRI
TRADEBE TREATMENT & RECYCLING LLC	EAST CHICAGO	IN	19	250	0.1	Non-POTW WWT	2016 TRI
VEOLIA ES TECHNICAL SOLUTIONS LLC	WEST CARROLLTON	OH	2	250	0.01	POTW	2016 TRI
VEOLIA ES TECHNICAL SOLUTIONS LLC	AZUSA	CA	0.5	250	0.002	POTW	2016 TRI
VEOLIA ES TECHNICAL SOLUTIONS LLC	MIDDLESEX	NJ	115,059	250	460	99.996% Non-POTW WWT 0.004% POTW	2016 TRI
CHEMICAL WASTE MANAGEMENT	EMELLE	AL	4	250	0.01	Surface Water	2016 DMR
OILTANKING HOUSTON INC	HOUSTON	TX	1	250	0.003	Surface Water	2016 DMR
HOWARD CO ALFA RIDGE LANDFILL	MARRIOTTSVILLE	MD	0.1	250	0.0002	Surface Water	2016 DMR
CLIFFORD G HIGGINS DISPOSAL SERVICE INC SLF	KINGSTON	NJ	0.02	250	0.0001	Surface Water	2016 DMR
CLEAN WATER OF NEW YORK INC	STATEN ISLAND	NY	2	250	0.01	Surface Water	2016 DMR
FORMER CARBORUNDUM COMPLEX	SANBORN	NY	0.2	250	0.001	Surface Water	2016 DMR

Site Identity	City	State	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	Release Media	Sources ^a & Notes
OES: Other							
APPLIED BIOSYSTEMS LLC	PLEASANTON	CA	42	250	0.2	Non-POTW WWT	2016 TRI
EMD MILLIPORE CORP	JAFFREY	NH	2	250	0.01	POTW	2016 TRI
GBC METALS LLC SOMERS THIN STRIP	WATERBURY	CT	0.2	250	0.001	Surface Water	2016 DMR
HYSTER-YALE GROUP, INC	SULLIGENT	AL	0.0002	250	0.000001	Surface Water	2016 DMR
AVNET INC (FORMER IMPERIAL SCHRADER)	ELLENVILLE	NY	0.005	250	0.00002	Surface Water	2016 DMR
BARGE CLEANING AND REPAIR	CHANNELVIEW	TX	0.1	250	0.0003	Surface Water	2016 DMR
AC & S INC	NITRO	WV	0.01	250	0.00005	Surface Water	2016 DMR
MOOG INC - MOOG IN-SPACE PROPULSION ISP	NIAGARA FALLS	NY	0.003	250	0.00001	Surface Water	2016 DMR
OILTANKING JOLIET	CHANNAHON	IL	1	250	0.003	Surface Water	2016 DMR
NIPPON DYNAWAVE PACKAGING COMPANY	LONGVIEW	WA	22	250	0.1	Surface Water	2016 DMR
TREE TOP INC WENATCHEE PLANT	WENATCHEE	WA	0.01	250	0.00003	Surface Water	2016 DMR
CAROUSEL CENTER	SYRACUSE	NY	0.001	250	0.000002	Surface Water	2016 DMR

^a Sources: 2016 TRI ([U.S. EPA, 2017f](#)); 2016 DMR ([EPA, 2016](#))

Appendix E ENVIRONMENTAL EXPOSURES

Table_Apx E-1. Occurrence of Methylene Dichloride Releases (Facilities) and Monitoring Sites By HUC-8

HUC8	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
HUCs with Co-located Methylene Dichloride Releases (Facilities) and Monitoring Sites (n = 2)							
15060106	Lower Salt	666211.2	2696.1	AZ	1	5	12
15070102	Aqua Fria	1758350.5	7115.8	AZ	3	7	11
HUCs with Methylene Dichloride Releases (Facilities) Only (n = 72)							
01070003	Contoocook	488993.1	1978.9	NH	1	0	0
02030103	Hackensack-Passaic	725724.6	2936.9	NJ,NY	1	0	0
02030104	Sandy Hook-Staten Island	454261.8	1838.3	NJ,NY	2	0	0
02030105	Raritan	707463.2	2863.0	NJ	4	0	0
02040206	Cohansey-Maurice	764587.9	3094.2	DE,NJ	1	0	0
02020007	Rondout	760490.1	3077.6	NJ,NY	1	0	0
02020006	Middle Hudson	1554773.3	6291.9	MA,NY	1	0	0
02030102	Bronx	120544.9	487.8	CT,NY	1	0	0
02030202	Southern Long Island	1255171.2	5079.5	NJ,NY,RI	2	0	0
04130001	Oak Orchard-Twelve mile	685684.0	2774.9	CN,NY	1	0	0
04130003	Lower Genesee	682891.3	2763.6	NY	2	0	0
04140201	Seneca	2214337.6	8961.1	NY	1	0	0
05060002	Lower Scioto	1392040.5	5633.4	KY,OH	1	0	0
05090202	Little Miami	1125043.6	4552.9	OH	1	0	0
05080002	Lower Great Miami, Indiana, Ohio	883871.2	3576.9	IN,OH	2	0	0
03150201	Upper Alabama	1530362.5	6193.2	AL	1	0	0
03150202	Cahaba	1167292.7	4723.9	AL	1	0	0
03160204	Mobile-Tensaw	583840.0	2362.7	AL	1	0	0
06030002	Wheeler Lake	1851599.9	7493.2	AL,TN	1	0	0
03160108	Noxubee	907700.0	3673.3	AL,MS	1	0	0
08010211	Horn Lake-Nonconnah	178697.3	723.2	MS,TN	1	0	0
08010100	Lower Mississippi-Memphis	702312.8	2842.2	AR,IL,KY,MO,MS,TN	2	0	0
15020016	Lower Little Colorado	1532516.1	6201.9	AZ	1	0	0

HUC8	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
15050301	Upper Santa Cruz	1680515.5	6800.8	AZ,MX	1	0	0
12040104	Buffalo-San Jacinto	756769.3	3062.5	TX	4	0	0
12040203	North Galveston Bay	228393.2	924.3	TX	2	0	0
12040204	West Galveston Bay	776232.4	3141.3	TX	1	0	0
12070104	Lower Brazos	1051241.4	4254.2	TX	1	0	0
18010102	Mad-Redwood	910412.8	3684.3	CA	1	0	0
18020155	Paynes Creek-Sacramento River	271113.3	1097.2	CA	1	0	0
18020163	Lower Sacramento	786286.3	3182.0	CA	1	0	0
18060006	Central Coastal	1231592.2	4984.1	CA	1	0	0
18060015	Monterey Bay	484626.6	1961.2	CA	1	0	0
05050008	Lower Kanawha	591554.2	2393.9	WV	3	0	0
18070103	Calleguas	280115.7	1133.6	CA	1	0	0
18070104	Santa Monica Bay	430957.7	1744.0	CA	1	0	0
18070105	Los Angeles	531817.9	2152.2	CA	1	0	0
18070106	San Gabriel	579966.3	2347.0	CA	5	0	0
18070203	Santa Ana	1084241.9	4387.8	CA	1	0	0
18070303	San Luis Rey-Escondido	531675.9	2151.6	CA	1	0	0
18070304	San Diego	993894.7	4022.2	CA,MX	1	0	0
01100006	Saugatuck	287476.3	1163.4	CT,NY	1	0	0
01100005	Housatonic	1248786.3	5053.7	CT,MA,NY	2	0	0
05030201	Little Muskingum-Middle Island	1161545.0	4700.6	OH,WV	2	0	0
05030202	Upper Ohio-Shade	906812.9	3669.7	OH,WV	1	0	0
05090101	Raccoon-Symmes	933778.8	3778.9	KY,OH,WV	1	0	0
05020003	Upper Monongahela	296728.7	1200.8	PA,WV	1	0	0
17110011	Snohomish	189946.6	768.7	WA	1	0	0
03070103	Upper Ocmulgee	1902869.0	7700.6	GA	1	0	0
03150101	Conasauga	465346.3	1883.2	GA,TN	1	0	0
07130001	Lower Illinois-Senachwine Lake	1254288.3	5075.9	IL	1	0	0
17050114	Lower Boise	850233.1	3440.8	ID	1	0	0
07120003	Chicago	419754.7	1698.7	IL,IN	1	0	0

HUC8	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
07140101	Cahokia-Joachim	1053340.7	4262.7	IL,MO	2	0	0
07120004	Des Plaines	931517.4	3769.7	IL,WI	3	0	0
04040001	Little Calumet-Galien	440799.0	1783.8	IL,IN,MI	1	0	0
17080003	Lower Columbia-Clatskanie	732479.8	2964.2	OR,WA	1	0	0
17020010	Upper Columbia-Entiat	958508.9	3878.9	WA	1	0	0
17030003	Lower Yakima	1860149.0	7527.8	WA	2	0	0
06040006	Lower Tennessee	446630.3	1807.5	KY,TN	1	0	0
05140202	Highland-Pigeon	663290.7	2684.2	IL,IN,KY	1	0	0
05090103	Little Scioto-Tygarts	644954.4	2610.0	KY,OH,W V	2	0	0
08070204	Lake Maurepas	456253.8	1846.4	LA	1	0	0
08070300	Lower Grand	508704.3	2058.7	LA	1	0	0
08080206	Lower Calcasieu	812177.5	3286.8	LA	2	0	0
02060003	Gunpowder-Patapsco	907202.4	3671.3	MD,PA	4	0	0
02060006	Patuxent	593323.7	2401.1	MD	1	0	0
04090004	Detroit	567874.0	2298.1	CN,MI	1	0	0
03160103	Buttahatchee	553396.1	2239.5	AL,MS	1	0	0
04120104	Niagara	871679.6	3527.6	CN,NY	2	0	0
04060102	Muskegon	1745075.3	7062.1	MI	1	0	0
04080201	Tittabawassee	926364.9	3748.9	MI	1	0	0
HUCs with Monitoring Sites Only (n = 42)							
03030003	Deep	928079.2	3755.8	NC	0	1	9
03030004	Upper Cape Fear	1043179.5	4221.6	NC	0	1	1
03030005	Lower Cape Fear	706736.1	2860.1	NC	0	3	14
03030006	Black	1007357.4	4076.6	NC	0	3	37
03030007	Northeast Cape Fear	1114550.1	4510.4	NC	0	4	28
03040101	Upper Yadkin	1571033.4	6357.8	NC,VA	0	2	21
03040103	Lower Yadkin	761498.9	3081.7	NC	0	1	9
03040105	Rocky	907088.6	3670.9	NC,SC	0	1	11
03050101	Upper Catawba	1508875.2	6106.2	NC,SC	0	4	47
06010105	Upper French Broad	1202906.3	4868.0	NC,SC,T N	0	3	33
06010108	Nolichucky	1125185.5	4553.5	NC,TN	0	1	12

HUC8	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
03010103	Upper Dan	1315517.1	5323.7	NC,VA	0	1	10
03010106	Roanoke Rapids	378781.5	1532.9	NC,VA	0	1	13
02040105	Middle Delaware-Musconetcong	869995.3	3520.8	NJ,PA	0	1	3
11080001	Canadian Headwaters	1104144.6	4468.3	CO,NM	0	12	13
11080002	Cimarron	671679.8	2718.2	NM	0	5	5
11080003	Upper Canadian	1314676.9	5320.3	NM	0	3	3
11080004	Mora	932568.3	3774.0	NM	0	6	6
11080006	Upper Canadian-Ute Reservoir	1432680.7	5797.9	NM,TX	0	5	6
11080008	Revuelto	515805.1	2087.4	NM	0	1	1
13020201	Rio Grande-Santa Fe	1197851.1	4847.5	NM	0	1	3
13020203	Rio Grande-Albuquerque	2057935.0	8328.2	NM	0	1	3
11040001	Cimarron Headwaters	1073779.5	4345.4	CO,NM,OK	0	1	1
11100101	Upper Beaver	1748464.8	7075.8	NM,OK,TX	0	1	1
03040202	Lynches	904417.1	3660.1	NC,SC	0	1	11
03040203	Lumber	1121797.1	4539.8	NC,SC	0	3	27
06030003	Upper Elk	821468.2	3324.4	AL,TN	0	4	8
12100303	Lower San Antonio	950344.1	3845.9	TX	0	1	1
03010107	Lower Roanoke	838200.5	3392.1	NC	0	1	2
03020202	Middle Neuse	681738.1	2758.9	NC	0	3	15
02070004	Conococheague-Opequon	1457399.0	5897.9	MD,PA,VA,WV	0	1	3
11030012	Little Arkansas	910452.3	3684.5	KS	0	5	14
07140102	Meramec	1375977.1	5568.4	MO	0	4	7
03020101	Upper Tar	835088.1	3379.5	NC	0	1	2
03020102	Fishing	572188.7	2315.6	NC	0	1	13
03020103	Lower Tar	614561.4	2487.0	NC	0	1	1
03020104	Pamlico	836270.2	3384.3	NC	0	1	2
03020201	Upper Neuse	1539933.1	6231.9	NC	0	1	13
03020204	Lower Neuse	1013224.6	4100.4	NC	0	2	14
03020302	New River	554324.3	2243.3	NC	0	1	2
03030002	Haw	1092854.1	4422.6	NC	0	2	21

HUC8	HUC Name	Area (Acres)	Area (km²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
09030008	Lower Rainy	982352.5	3975.4	CN,MN	0	1	2

Table_Apx E-2. Occurrence of Methylene Dichloride Releases (Facilities) and Monitoring Sites By HUC-12

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
HUCs with Methylene Dichloride Releases (Facilities) and Monitoring Sites (n = 1)							
150601060306	City of Phoenix-Salt River	87618.1	354.6	AZ	1	2	4
HUCs with Methylene Dichloride Releases (Facilities) Only (n = 86)							
031602040401	Gunnison Creek	28009.6	113.3	AL	1	0	0
060300020501	Upper Indian Creek	24626.8	99.7	AL	1	0	0
031601081005	Bodka Creek-Caney Creek	33649.7	136.2	AL,MS	1	0	0
031502010407	Lower Pintlala Creek	15550.7	62.9	AL	1	0	0
031502020202	Cahaba Valley Creek	17492.0	70.8	AL	1	0	0
031601030202	Cannon Mill Creek-Beaver Creek	28263.4	114.4	AL	1	0	0
080101000703	Loosahatchie Bar-Mississippi River	37253.2	150.8	AR,TN	2	0	0
150200160807	Janus Spring-Little Colorado River	27894.8	112.9	AZ	1	0	0
180201550405	Sevenmile Creek-Sacramento River	17275.5	69.9	CA	1	0	0
180701060606	Coyote Creek-San Gabriel River	37975.6	153.7	CA	2	0	0
180701060701	Long Beach Harbor	33394.5	135.1	CA	1	0	0
180702030804	East Etiwanda Creek-Santa Ana River	138518.8	560.6	CA	1	0	0
180703030504	Loma Alta Creek-Frontal Gulf of Santa Catalina	52326.8	211.8	CA	1	0	0
180201630403	Laguna Creek	30785.5	124.6	CA	1	0	0
150701020605	Lookout Mountain-Cave Creek	22632.2	91.6	AZ	2	0	0
150701020907	White Tank Number Three Wash	44741.3	181.1	AZ	1	0	0
180101020408	Mill Creek-Mad River	19798.6	80.1	CA	1	0	0
180600060106	Potrero Canyon-Carmel River	19786.8	80.1	CA	1	0	0
180703041300	Mission Beach-Frontal Pacific Ocean	107314.7	434.3	CA,MX	1	0	0
180600150305	Monterey Bay	224556.6	908.8	CA	1	0	0
180701030102	Lower Simi Arroyo	39214.2	158.7	CA	1	0	0
180701040500	Manhattan Beach-Frontal Santa Monica Bay	74377.4	301.0	CA	1	0	0
180701050401	Chavez Ravine-Los Angeles River	39431.4	159.6	CA	1	0	0
180701060102	Lower Dominguez Channel	36125.6	146.2	CA	2	0	0
030701031605	Stone Creek-Ocmulgee River	63787.5	258.1	GA	1	0	0

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
040400010603	Calumet River-Frontal Lake Michigan	34563.8	139.9	IL,IN	1	0	0
071200030104	North Shore Channel	14685.7	59.4	IL	1	0	0
071200040905	Des Plaines River	23822.3	96.4	IL	3	0	0
071401010401	Maline Creek-Mississippi River	60447.7	244.6	IL,MO	2	0	0
031501010504	Jobs Creek-Conasauga River	32865.9	133.0	GA	1	0	0
071300011004	Senachwine Lake-Illinois River	24040.8	97.3	IL	1	0	0
080702040103	Grand Goudine Bayou-New River	17644.3	71.4	LA	1	0	0
080703000207	Bayou Bourbeaux	16521.5	66.9	LA	1	0	0
051402020605	Beaverdam Creek-Ohio River	30633.3	124.0	IN,KY	1	0	0
080802060301	Maple Fork-Bayou d'Inde	22308.4	90.3	LA	1	0	0
080802060303	Prien Lake-Calcasieu River	29606.9	119.8	LA	1	0	0
020600030902	Dead Run-Gywnns Falls	31450.3	127.3	MD	4	0	0
060400060502	Guess Creek-Tennessee River	20398.5	82.5	KY	1	0	0
050901030105	Pond Run-Ohio River	28165.0	114.0	KY,OH	2	0	0
020600060202	Dorsey Run-Little Patuxent River	42440.5	171.8	MD	1	0	0
080102110302	Horn Lake-Horn Lake Pass	18306.6	74.1	MS,TN	1	0	0
041402011509	Onondaga Lake	26522.2	107.3	NY	1	0	0
020402060103	Whooping John Creek-Frontal Delaware River	10235.8	41.4	DE,NJ	1	0	0
020301040204	Morses Creek-Arthur Kill	18931.5	76.6	NJ,NY	1	0	0
050600020105	Oak Run	17133.2	69.3	OH	1	0	0
020200060402	Onesquethaw Creek	35841.4	145.1	NY	1	0	0
050800020106	Opossum Creek-Great Miami River	12167.1	49.2	OH	2	0	0
041201040603	Cayuga Creek	22754.1	92.1	NY	2	0	0
041300010501	Jeddo Creek	20039.9	81.1	NY	1	0	0
020200070504	Sandburg Creek	37947.4	153.6	NY	1	0	0
020301020203	East Creek-Frontal Long Island Sound	11252.5	45.5	NY	1	0	0
020301030801	Preakness Brook-Passaic River	14523.7	58.8	NJ	1	0	0
020301040203	Newark Bay	17761.8	71.9	NJ	1	0	0
020302020206	Reynolds Channel-East Rockaway Inlet	10571.6	42.8	NY	1	0	0
041300030502	Jaycox Creek-Genesee River	25635.1	103.7	NY	1	0	0

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
041300030704	Genesee River	14336.9	58.0	NY	1	0	0
050902021404	Duck Creek	9891.1	40.0	OH	1	0	0
020301050312	Lower Millstone River	31839.8	128.8	NJ	1	0	0
020302020406	Santapogue Creek-Great South Bay	17890.8	72.4	NY	1	0	0
050302011004	Haynes Run-Ohio River	19386.4	78.5	OH,W V	1	0	0
050302011006	Mill Creek-Ohio River	27702.4	112.1	OH,W V	1	0	0
050302020106	Sandy Creek-Ohio River	25650.1	103.8	OH,W V	1	0	0
050901010103	Long Run-Ohio River	16607.3	67.2	OH,W V	1	0	0
020301050501	Peters Brook-Raritan River	15666.0	63.4	NJ	1	0	0
020301050507	Mill Brook-Raritan River	17892.2	72.4	NJ	2	0	0
120701040505	Outlet Barzos River	35803.4	144.9	TX	1	0	0
120401040703	Vince Bayou-Buffalo Bayou	38130.8	154.3	TX	2	0	0
120401040705	Highlands Reservoir-San Jacinto River	18115.0	73.3	TX	1	0	0
120401040706	Goose Creek-Frontal Galveston Bay	37289.7	150.9	TX	1	0	0
120402030106	Cedar Point Lateral-Cedar Bayou	31473.7	127.4	TX	2	0	0
120402040400	Mustang Bayou	183973. 7	744.5	TX	1	0	0
050200030307	Cobun Creek-Monongahela River	21730.5	87.9	WV	1	0	0
050500080303	Tyler Creek-Kanawha River	21033.5	85.1	WV	2	0	0
050500080304	Scary Creek-Kanawha River	20472.1	82.8	WV	1	0	0
170200100307	Rainey Spring-Columbia River	21142.9	85.6	WA	1	0	0
170300030906	Sulphur Creek Wasteway	19187.2	77.7	WA	2	0	0
170501140403	Crane Creek-Boise River	18624.7	75.4	ID	1	0	0
171100110203	Snohomish River-Frontal Possession Sound	45483.4	184.1	WA	1	0	0
170800030602	City of Longview-Frontal Columbia River	25007.4	101.2	WA	1	0	0
040601021002	Mosquito Creek-Muskegon River	31043.0	125.6	MI	1	0	0
150503010906	Arroyo Chico-Santa Cruz River	43989.0	178.0	AZ	1	0	0
010700030101	Town Farm Brook-Contoocook River	27145.4	109.8	NH	1	0	0
040802010604	Prairie Creek-Tittabawassee River	25251.7	102.2	MI	1	0	0

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
011000051205	Long Meadow Pond Brook-Naugatuck River	18242.3	73.8	CT	2	0	0
011000060405	Horseneck Brook-Frontal Long Island Sound	23419.3	94.8	CT,NY	1	0	0
40900040503	Belle Isle-Detroit River	45973.7	186.1	CN,MI	1	0	0
HUCs with Monitoring Sites Only (n = 97)							
150601060202	Upper Indian Bend Wash	27058.2	109.5	AZ	0	1	3
150601060307	Town of Santa Maria-Salt River	34122.5	138.1	AZ	0	2	5
150701020606	Upper Arizona Canal Diversion Channel	15465.9	62.6	AZ	0	1	3
150701020607	Lower Arizona Canal Diversion Channel	19739.1	79.9	AZ	0	1	1
150701020806	Middle Skunk Creek	28304.4	114.5	AZ	0	1	3
150701020807	Lower Skunk Creek	24449.6	98.9	AZ	0	2	2
150701020809	City of Peoria-New River	38282.5	154.9	AZ	0	2	2
110400011005	Miller Canyon-Dry Cimarron River	36341.5	147.1	CO,N M	0	1	1
110800010101	Upper Chicorica Creek	36590.1	148.1	CO,N M	0	1	1
110800010104	Raton Creek	28802.5	116.6	CO,N M	0	1	1
110800010304	Bernal Creek-Vermejo River	17284.0	70.0	CO,N M	0	1	1
110300120303	110300120303-Little Arkansas River	23920.3	96.8	KS	0	1	4
110300120408	City of Sedgwick-Little Arkansas River	27404.6	110.9	KS	0	4	10
071401020703	Stater Creek-Meramec River	28521.9	115.4	MO	0	1	2
071401021001	Hamilton Creek-Meramec River	34956.9	141.5	MO	0	1	2
071401021002	Grand Glaize Creek-Meramec River	29896.0	121.0	MO	0	1	2
071401021004	Meramec River	27977.7	113.2	MO	0	1	1
030402030103	Naked Creek	25026.5	101.3	NC	0	1	12
030300020301	Upper Big Alamance Creek	23563.4	95.4	NC	0	1	11
030300020506	Marys Creek-Haw River	18499.4	74.9	NC	0	1	10
030300030104	Bull Run-Deep River	11364.4	46.0	NC	0	1	9
030402030402	Bear Swamp	18155.9	73.5	NC	0	1	13
030202011501	Headwaters Little River	27575.7	111.6	NC	0	1	13
030202020103	Seymour Johnson Air Force Base-Neuse River	10050.8	40.7	NC	0	1	1

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
030402031005	River Swamp-Lumber River	13009.7	52.6	NC	0	1	2
030202020303	Yadkin Branch-Neuse River	11135.9	45.1	NC	0	1	1
030300040706	City of Fayetteville-Cape Fear River	18506.3	74.9	NC	0	1	1
030300050206	White Lake-Cape Fear River	19631.2	79.4	NC	0	1	2
030300050302	Middle Livingston Creek	17637.8	71.4	NC	0	1	11
030202020404	Clayroot Swamp	31573.4	127.8	NC	0	1	13
030300050501	Indian Creek-Cape Fear River	18164.0	73.5	NC	0	1	1
030300060301	Caesar Swamp-Little Coharie Creek	30510.3	123.5	NC	0	1	12
030300060303	Bearskin Swamp	16148.0	65.3	NC	0	1	13
030300060805	Rowan Creek-Black River	26201.3	106.0	NC	0	1	12
030501010106	Toms Creek-Catawba River	17337.3	70.2	NC	0	1	11
030501010401	Upper Warrior Fork	23781.8	96.2	NC	0	1	12
030501010501	Upper Johns River	26796.4	108.4	NC	0	1	12
030501010504	Lower Wilson Creek	18305.8	74.1	NC	0	1	12
030201010903	Buck Swamp-Tar River	20652.5	83.6	NC	0	1	2
030201020204	Bear Swamp	28720.3	116.2	NC	0	1	13
030300070201	Lewis Branch-Northeast Cape Fear River	19845.8	80.3	NC	0	1	13
030202040204	Town of Trenton-Trent River	43012.8	174.1	NC	0	1	12
030202040401	City of New Bern-Neuse River	14210.7	57.5	NC	0	1	2
030101030109	Flat Shoals Creek-Dan River	28246.1	114.3	NC	0	1	10
030201030202	Town Creek-Tar River	19716.5	79.8	NC	0	1	1
060101050302	Clear Creek	28811.3	116.6	NC	0	1	10
060101050403	Mills River	20437.8	82.7	NC	0	1	11
060101050503	Lower Hominy Creek	15416.6	62.4	NC	0	1	12
030101070509	City of Williamston-Roanoke River	15369.3	62.2	NC	0	1	2
030201040103	Hills Creek-Pamlico River	20821.4	84.3	NC	0	1	2
030300070611	Lewis Creek-Northeast Cape Fear River	34873.9	141.1	NC	0	1	1
030300070802	Pike Creek-Northeast Cape Fear River	34936.3	141.4	NC	0	1	13
060101080206	Jacks Creek	13392.1	54.2	NC	0	1	12
030300070809	Ness Creek-Northeast Cape Fear River	17715.3	71.7	NC	0	1	1

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
030401010306	Mulberry Creek	31521.5	127.6	NC	0	1	10
030402020102	Headwaters Lynches River	32657.2	132.2	NC,SC	0	1	11
030401011005	Little Yadkin River	18870.5	76.4	NC	0	1	11
030203020103	Cowhorn Swamp-New River	18267.5	73.9	NC	0	1	2
030401030601	Lick Creek	21942.3	88.8	NC	0	1	9
030401050203	Irish Buffalo Creek	29616.8	119.8	NC	0	1	11
030101060205	Blue Mud Creek-Smith Creek	23151.8	93.7	NC,VA	0	1	13
020401050911	Buck Creek-Delaware River	15442.9	62.5	NJ,PA	0	1	3
110800010107	Outlet Una de Gato Creek	18883.6	76.4	NM	0	1	1
110800010305	York Canyon	19318.4	78.2	NM	0	1	1
110800010306	Griffin Canyon-Vermejo River	31314.3	126.7	NM	0	1	2
110800010309	Bracket Canyon-Vermejo River	27060.4	109.5	NM	0	1	1
110800010401	Rail Canyon-Vermejo River	28467.1	115.2	NM	0	2	2
110800010406	Stubblefield Arroyo-Vermejo River	28101.0	113.7	NM	0	1	1
110800010510	Maxwell National Wildlife Refuge	22719.1	91.9	NM	0	1	1
110800010606	110800010606-Canadian River	28344.2	114.7	NM	0	1	1
110800020104	Outlet Cieneguilla Creek	13369.9	54.1	NM	0	1	1
110800020105	Eagle Nest Lake	18531.5	75.0	NM	0	1	1
110800020109	Turkey Creek Canyon-Cimarron River	29455.4	119.2	NM	0	1	1
110800020401	Springer Lake	15355.0	62.1	NM	0	1	1
110800020404	Outlet Cimarron River	26894.7	108.8	NM	0	1	1
110800030107	Charette Lake-Ocate Creek	38051.9	154.0	NM	0	1	1
110800030505	Canon Vercere-Canadian River	17450.2	70.6	NM	0	1	1
130202010209	Canada de Cochiti-Rio Grande	20418.4	82.6	NM	0	1	3
130202030107	Town of Corrales-Rio Grande	26313.8	106.5	NM	0	1	3
110800030610	Canon Negro-Canadian River	25106.6	101.6	NM	0	1	1
110800040106	Lower Coyote Creek	29881.2	120.9	NM	0	1	1
110800040208	Phoenix Lake-Sapello River	14850.8	60.1	NM	0	1	1
110800040305	Encinal Creek-Mora River	15092.1	61.1	NM	0	1	1
110800040306	Santiago Creek	19713.5	79.8	NM	0	1	1
110800040308	Eagle Creek-Mora River	38784.0	156.9	NM	0	1	1
110800040605	Canon Vegocito-Mora River	29443.0	119.2	NM	0	1	1

HUC12	HUC Name	Area (Acres)	Area (km ²)	States	No. of Facilities	No. of Mon. Sites	No. of Samples
110800060909	Martin Draw-Canadian River	20893.7	84.5	NM,T X	0	1	1
110800060409	Carpenter Creek-Canadian River	36596.2	148.1	NM	0	1	2
110800060606	Outlet Pajarito Creek	34811.1	140.9	NM	0	1	1
110800060801	Hudson Lake-Ute Reservoir	32050.3	129.7	NM	0	1	1
110800060805	Town of Logan-Canadian River	25798.5	104.4	NM	0	1	1
110800080504	Lower Revuelto Creek	25500.0	103.2	NM	0	1	1
111001010204	Clayton Lake-Seneca Creek	21142.1	85.6	NM	0	1	1
020700040702	Dennis Creek-Back Creek	32533.8	131.7	PA	0	1	3
060300030201	Bradley Creek	30268.8	122.5	TN	0	4	8
121003030306	Salt Creek-Ecleto Creek	18817.5	76.2	TX	0	1	1
090300080501	City of International Falls-Rainy River	36508.3	147.7	CN,M N	0	1	2

Table_Apx E-3. Sample Information for WQX Surface Water Observations With Concentrations Above the Reported Detection Limit: 2013-2017^a

Monitoring Site Information					Sample Information		
Monitoring Site ID and Organization	Waterbody Type and Location	Lat/Long	HUC 8	Provider	Sample ID	Date and Time	Concentration (µg/L) ^b
USGS-11074000 USGS California Water Science Center	<i>Stream</i> SANTA ANA R BL PRADO DAM CA	33.8833488/ -117.6453296	18070203	NWIS	nwisca.01.01402259	2014-03-25 11:10:00 PDT	0.17
USGS-05537000 USGS Illinois Water Science Center	<i>Stream</i> CHICAGO SANITARY AND SHIP CANAL AT LOCKPORT, IL	41.5702778/ -88.0794444	7120004	NWIS	nwisil.01.01400214	2014-02-11 11:10:00 CST	0.13
					nwisil.01.01500412	2015-05-06 13:00:00 CST	0.04
					nwisil.01.01500568	2015-06-22 13:30:00 CST	0.07
USGS-05538020 USGS Illinois Water Science Center	<i>Stream</i> DES PLAINES RIVER IN LOCK CHANNEL AT ROCKDALE, IL	41.5/ -88.1069444	7120004	NWIS	nwisil.01.01500240	2015-05-06 18:00:00 CST	0.04
					nwisil.01.01500689	2015-06-22 16:30:00 CST	0.04
USGS-375348097262800 USGS Kansas Water Science Center	<i>Stream</i> DISCHARGE FROM L ARKANSAS R ASR NR SEDGWICK, KS	37.8967222/ -97.4410278	11030012	NWIS	nwisks.01.01401112	2014-06-09 10:30:00 CDT	0.8
USGS-405034073554501 USGS New York Water Science Center	<i>Estuary</i> Harlem River at Exterior Street, suite 2	40.8428611/ -73.9292222	2030101	NWIS	nwisny.01.01702060	2017-07-24 11:00:00 EST	0.61
21NC03WQ-B8484000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	<i>River/Stream</i> BEARSKIN SWAMP AT SR 1325 NR CLINTON	35.08754/ -78.43463	3030006	STORET	21NC03WQ-AMS20161206-B8484000-370870277	2016-12-06 11:40:00 EST	1.2
					21NC03WQ-AMS20161206-B8484000-381057619	2016-12-06 11:55:00 EST	1.2
21NC03WQ-E0380000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	<i>River/Stream</i> CHERRYFIELD CRK OFF STILL WATERS LN NR ROSMAN	35.18471/ -82.81184	6010105	STORET	21NC03WQ-RAMS2014-000245560	2014-08-04 15:45:00 EDT	1.2
21NC03WQ-E1485000	<i>River/Stream</i> North Mills River at SR 1343 (River Loop Rd) nr Mills River	35.39412/ -82.61646	6010105	STORET	21NC03WQ-AMS20160822-E1485000-381059366	2016-08-22 15:55:00 EST	29

Monitoring Site Information					Sample Information		
Monitoring Site ID and Organization	Waterbody Type and Location	Lat/Long	HUC 8	Provider	Sample ID	Date and Time	Concentration (µg/L) ^b
North Carolina Department of Environmental Resources NCDENR -DWQ WQX					21NC03WQ-AMS20160822-E1485000-381059612	2016-08-22 16:00:00 EST	29
21NC03WQ-E3475000 North Carolina Department of Environmental Resources NCDENR -DWQ WQX	<i>River/Stream</i> Hominy Creek at Pond Rd in Asheville ^c	35.54683/ -82.60264	6010105	STORET	21NC03WQ-RAMS20160817-E3475000-370533933	2016-08-17 17:05:00 EST	5
21NYDECA_WQX-01010001 New York State Dec Division Of Water	<i>River/Stream</i> NIAGARA R. IN FT.NIAGARA	43.2611111/ -79.0630556	4120104	STORET	21NYDECA_WQX-01010001_09172013_WS	2013-09-17 09:15:00 EDT	0.50
					21NYDECA_WQX-1010001_10072013_WS	2013-10-07 09:15:00 EDT	0.50
21NYDECA_WQX-01031002 New York State Dec Division Of Water	<i>River/Stream</i> Buffalo River	42.8616667/ -78.8677778	4120103	STORET	21NYDECA_WQX-01031002_09172013_WS	2013-09-17 01:30:00 EDT	0.50
					21NYDECA_WQX-01031002_10072013_WS	2013-10-07 11:30:00 EDT	0.50
21NYDECA_WQX-02010023 New York State Dec Division Of Water	<i>River/Stream</i> Allegheny River	42.1566667/ -78.7158333	5010001	STORET	21NYDECA_WQX-02010023_09172013_WS	2013-09-17 11:30:00 EDT	0.50
					21NYDECA_WQX-02010023_10072013_WS	2013-10-07 11:45:00 EDT	0.50
21NYDECA_WQX-04010003 New York State Dec Division Of Water	<i>River/Stream</i> Genesee River	43.2272222/ -77.6163889	4130003	STORET	21NYDECA_WQX-04010003_09182013_WS	2013-09-18 09:45:00 EDT	0.50
					21NYDECA_WQX-04010003_10082013_WS	2013-10-08 11:00:00 EDT	0.50
21NYDECA_WQX-05010005 New York State Dec Division Of Water	<i>River/Stream</i> Chemung River	42.0027778/ -76.6341667	2050105	STORET	21NYDECA_WQX-05010005_10212013_WS	2013-10-21 12:00:00 EDT	0.50
21NYDECA_WQX-06021001 New York State Dec Division Of Water	<i>River/Stream</i> Chenango River	42.1030556/ -75.915	2050102	STORET	21NYDECA_WQX-06021001_09182013_WS	2013-09-17 12:00:00 EDT	0.50
					21NYDECA_WQX-06021001_10092013_WS	2013-10-09 12:00:00 EDT	0.50
21NYDECA_WQX-06030006 New York State Dec Division Of Water	<i>River/Stream</i> Susquehanna River	42.0280556/ -76.3847222	2050103	STORET	21NYDECA_WQX-06030006_09182013_WS	2013-09-18 10:00:00 EDT	0.50
					21NYDECA_WQX-06030006_10092013_WS	2013-10-09 11:00:00 EDT	0.50

Monitoring Site Information					Sample Information		
Monitoring Site ID and Organization	Waterbody Type and Location	Lat/Long	HUC 8	Provider	Sample ID	Date and Time	Concentration (µg/L) ^b
21NYDECA_WQX-07010005 New York State Dec Division Of Water	<i>River/Stream</i> Oswego River	43.3980556/ -76.4708333	4140203	STORET	21NYDECA_WQX-07010005_09172013_WS	2013-09-17 10:00:00 EDT	0.50
					21NYDECA_WQX-07010005_10082013_WS	2013-10-08 10:00:00 EDT	0.50
21NYDECA_WQX-07011023 New York State Dec Division Of Water	<i>River/Stream</i> Seneca River	43.099/ -76.424	4140201	STORET	21NYDECA_WQX-07011023_09172013_WS	2013-09-17 11:00:00 EDT	0.50
					21NYDECA_WQX-07011023_10082013_WS	2013-10-08 11:00:00 EDT	0.50

- c. Data was downloaded from the WQP (www.waterqualitydata.us) on 10/3/2018. NWIS and STORET surface water data was obtained by selecting “Methylene chloride (NWIS, STORET)” for the Characteristic and selecting for surface water media and locations only. Results were reviewed and filtered to obtain a cleansed dataset (i.e., samples/sites were eliminated if identified as estimated, QC, media type other than surface water, Superfund, landfill, failed laboratory QC, etc.).
- d. Concentrations in bold exceed the lowest COC (8.2 µg/L).

Table_Apx E-4. E-FAST Modeling Results for Known Direct and Indirect Releasing Facilities for 2016

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in E-FAST ^c	E-FAST Waterbody Type ^d	Days of release ^e	Release (kg/day) ^f	7Q10 SWC (ppb) ^g	COC (ppb)	Days of Exceedance (days/yr) ^h
OES: Manufacturing								
COVESTRO LLC BAYTOWN, TX FRS: 110000463098	Surface Water	Active Releaser: NPDES TX0002798	Surface water	350	0.004	0.43	90.0	4
							151	4
							1800	4
				20	0.068	7.510	90.0	1
							151	1
							1800	0
EMERALD PERFORMANCE MATERIALS LLC HENRY, IL NPDES: IL0001392	Surface Water	Active Releaser: NPDES IL0001392	Still water	350	0.001	0.480	90.0	0
							151	0
							1800	0
				20	0.023	8.32	90.0	0
							151	0
							1800	0
FISHER SCIENTIFIC CO LL C FAIR LAWN, NJ NPDES: NJ0110281	POTW	Receiving Facility: PASSAIC VALLEY SEWER COMM; NPDES NJ0021016	Still water	350	0.01	0.000442	90.0	0
							151	0
							1800	0
FISHER SCIENTIFIC CO LLC BRIDGEWATER, NJ NPDES: NJ0119245	POTW	Receiving Facility: SOMERSET RARITIAN VALLEY SEWERAGE; NPDES NJ0024864	Surface water	350	0.01	0.07	90.0	0
							151	0
							1800	0
OLIN BLUE CUBE FREEPORT TX FREEPORT, TX TRI: 7754WBLCBP231NB	Non- POTW WWT	Receiving Facility: DOW CHEMICAL-FREEPORT, TX; NPDES TX0006483	Surface water	350	0.2	0.029	90.0	0
							151	0
							1800	0
REGIS TECHNOLOGIES INC MORTON GROVE, IL FRS: 110000429661	POTW	Receiving Facility: MWRDGC TERRENCE J O'BRIEN WTR RECLAMATION PLANT; NPDES IL0028088	Still water	350	0.01	0.00270	90.0	0
							151	0
							1800	0
SIGMA-ALDRICH MANUFACTURING LLC	POTW	Receiving Facility: BISSEL POINT WWTP ST LOUIS	Surface water	350	0.01	0.0000366	90.0	0
							151	0

SAINT LOUIS, MO FR: 110000743125		MSD; NPDES MO0025178					1800	0
VANDERBILT CHEMICALS LLC- MURRAY DIV MURRAY, KY NPDES: KY0003433	Non- POTW WWT	Receiving Facility: VALICOR ENVIRONMENTAL SERVICES; Organic Chemicals Manufacturing	Surface water	350	0.0013	0.110	90.0	0
							151	0
							1800	0
E I DUPONT DE NEMOURS - CHAMBERS WORKS DEEPWATER, NJ NPDES: NJ0005100	Surface Water	Active Releaser: NPDES NJ0005100	Surface water	350	0.2	0.0322	90.0	0
							151	0
							1800	0
				20	3.8	0.56	90.0	0
							151	0
							1800	0
BAYER MATERIALSCIENCE BAYTOWN , TX NPDES: TX0002798	Surface Water	Active Releaser: NPDES TX0002798	Surface water	350	0.03	3.15	90.0	11
							151	7
							1800	4
				20	0.50	55.08	90.0	3
							151	2
							1800	1
INSTITUTE PLANT INSTITUTE, WV NPDES: WV0000086	Surface Water	Active Releaser: NPDES WV0000086	Surface water	350	0.01	0.00282	90.0	0
							151	0
							1800	0
				20	0.16	0.0494	90.0	0
							151	0
							1800	0
MPM SILICONES LLC FRIENDLY, WV NPDES: WV0000094	Surface Water	Active Releaser: NPDES WV0000094	Surface water	350	0.005	0.000555	90.0	0
							151	0
							1800	0
				20	0.082	0.00972	90.0	0
							151	0
							1800	0
BASF CORPORATION WEST MEMPHIS, AR NPDES: AR0037770	Surface Water	Active Releaser: NPDES AR0037770	Surface water	350	0.003	0.0000134	90.0	0
							151	0
							1800	0
				20	0.059	0.000235	90.0	0
							151	0
							1800	0

							1800	0
ARKEMA INC PIFFARD, NY NPDES: NY0068225	Surface Water	Active Releaser: NPDES NY0068225	Surface water	350	0.001	0.00347	90.0	0
							151	0
							1800	0
				20	0.013	0.0608	90.0	0
							151	0
							1800	0
EAGLE US 2 LLC - LAKE CHARLES COMPLEX LAKE CHARLES, LA NPDES: LA0000761	Surface Water	Active Releaser: NPDES LA0000761	Surface water	350	0.001	0.00081	90.0	0
							151	0
							1800	0
				20	0.012	0.0141	90.0	0
							151	0
							1800	0
BAYER MATERIALSCIENCE NEW MARTINSVILLE, WV NPDES: WV0005169	Surface Water	Active Releaser: NPDES WV0005169	Surface water	350	0.001	0.000084	90.0	0
							151	0
							1800	0
				20	0.012	0.00148	90.0	0
							151	0
							1800	0
ICL-IP AMERICA INC GALLIPOLIS FERRY, WV NPDES: WV0002496	Surface Water	Active Releaser: NPDES WV0002496	Surface water	350	0.0004	0.0000262	90.0	0
							151	0
							1800	0
				20	0.0065	0.000458	90.0	0
							151	0
							1800	0
KEESHAN AND BOST CHEMICAL CO., INC. MANVEL, TX NPDES: TX0072168	Surface Water	Active Releaser: NPDES TX0072168	Still water	350	0.00005	4.73	90.0	0
							151	0
							1800	0
				20	0.00083	82.80	90.0	0
							151	0
							1800	0
INDORAMA VENTURES OLEFINS, LLC SULPHUR, LA NPDES: LA0069850	Surface Water	Active Releaser (Surrogate): NPDES LA0000761	Surface water	350	0.00003	0.0000301	90.0	0
							151	0
							1800	0
				20	0.00047	0.000527	90.0	0

							151	0
							1800	0
CHEMTURA NORTH AND SOUTH PLANTS MORGANTOWN, WV NPDES: WV0004740	Surface Water	Active Releaser: NPDES WV0004740	Surface water	350	0.00002	0.0000344	90.0	0
							151	0
							1800	0
				20	0.00041	0.000600	90.0	0
							151	0
							1800	0
OES: Import and Repackaging								
CHEMISPHERE CORP SAINT LOUIS, MO FRS: 110000852943	POTW	Receiving Facility: BISSEL POINT WWTP ST LOUIS MSD; NPDES MO0025178	Surface water	250	0.01	0.0000512	90.0	0
							151.0	0
							1800.0	0
HUBBARD-HALL INC WATERBURY, CT FRS: 110000317194	Non-POTW WWT	Receiving Facility: RECYCLE INC.; POTW (Ind.)	Surface water	250	0.58	34.38	90.0	8
							151.0	3
							1800.0	0
WEBB CHEMICAL SERVICE CORP MUSKEGON HEIGHTS, MI NPDES: MI0049719	POTW	Receiving Facility: MUSKEGON CO WWMS METRO WWTP; NPDES MI0027391	Surface water	250	0.4	0.1000	90.0	0
							151.0	0
							1800.0	0
RESEARCH SOLUTIONS GROUP INC PELHAM, AL NPDES: AL0074276	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0003	0.0442	90.0	0
							151.0	0
							1800.0	0
				20	0.0043	0.55	90.0	0
							151.0	0
							1800.0	0
EMD MILLIPORE CORP CINCINNATI, OH NPDES: OH0047759	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0001	0.0144	90.0	0
							151.0	0
							1800.0	0
				20	0.0014	0.18	90.0	0
							151.0	0
							1800.0	0
OES: Processing as a Reactant								
AMVAC CHEMICAL CO AXIS, AL FRS: 110015634866	Non-POTW WWT	Receiving Facility: DUPONT AGRICULTURAL PRODUCTS; NPDES AL0001597	Surface water	350	0.6	0.0151	90.0	0
							151.0	0
							1800.0	0

THE DOW CHEMICAL CO MIDLAND, MI NPDES: MI0000868	Surface Water	Active Releaser: NPDES MI0000868	Surface water	350	0.1	0.11	90.0	0
							151.0	0
							1800.0	0
				20	1.2	1.98	90.0	0
							151.0	0
							1800.0	0
FMC CORPORATION MIDDLEPORT, NY NPDES: NY0000345	Surface Water	Active Releaser: NPDES NY0000345	Surface water	350	0.0003	0.26	90.0	0
							151.0	0
							1800.0	0
				20	0.0057	4.55	90.0	0
							151.0	0
							1800.0	0
OES: Processing – Formulation								
ARKEMA INC CALVERT CITY, KY NPDES: KY0003603	Surface Water	Active Releaser: NPDES KY0003603	Surface water	300	0.1	0.00434	90.0	0
							151.0	0
							1800.0	0
				20	1.5	0.0668	90.0	0
							151.0	0
							1800.0	0
MCGEAN-ROHCO INC LIVONIA, MI FRS: 110000405801	POTW	Receiving Facility: DETROIT WWTP- CHLORINATION/DECHLO RINATION FACILITY; NPDES MI0022802	Surface water	300	0.4	0.00220	90.0	0
							151.0	0
							1800.0	0
WM BARR & CO INC MEMPHIS, TN FRS: 110000374265	POTW	Receiving Facility: MEMPHIS CITY MAXSON WASTEWATER TREATMENT; NPDES TN0020729	Surface water	300	0.002	0.00000277	90.0	0
							151.0	0
							1800.0	0
BUCKMAN LABORATORIES INC MEMPHIS, TN NPDES: TN0040606	POTW	Receiving Facility: MC STILES TREATMENT PLANT; NPDES TN0020711	Surface water	300	0.8	0.00156	90.0	0
							151.0	0
							1800.0	0
EUROFINS MWG OPERON LLC	POTW	Receiving Facility: VEOLIA ENVIRONMENTAL	Surface water	300	19	1659.44	90.0	221
							151.0	181

LOUISVILLE, KY TRI: 4029WRFNSM1271P		SERVICES TECH SOLUTIONS LLC; Inorganic Chemicals Manuf.					1800.0	21
SOLVAY - HOUSTON PLANT HOUSTON, TX NPDES: TX0007072	Surface Water	Active Releaser: NPDES TX0007072	Surface water	300	0.04	7.15	90.0	0
							151.0	0
							1800.0	0
				20	0.58	107.41	90.0	0
							151.0	0
							1800.0	0
HONEYWELL INTERNATIONAL INC - GEISMAR COMPLEX GEISMAR, LA NPDES: LA0006181	Surface Water	Active Releaser: NPDES LA0006181	Surface water	300	0.01	0.0000603	90.0	0
							151.0	0
							1800.0	0
				20	0.22	0.000890	90.0	0
							151.0	0
							1800.0	0
STEPAN CO MILLSDALE ROAD ELWOOD, IL NPDES: IL0002453	Surface Water	Active Releaser: NPDES IL0002453	Surface water	300	0.01	0.00324	90.0	0
							151.0	0
							1800.0	0
				20	0.12	0.0503	90.0	0
							151.0	0
							1800.0	0
ELEMENTIS SPECIALTIES, INC. CHARLESTON, WV NPDES: WV0051560	Surface Water	Active Releaser: NPDES WV0051560	Surface water	300	0.001	0.000474	90.0	0
							151.0	0
							1800.0	0
				20	0.011	0.00709	90.0	0
							151.0	0
							1800.0	0
OES: Polyurethane Foam								
PREGIS INNOVATIVE PACKAGING INC WURLAND, KY NPDES: KY0094005	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	0.01	1.13	90.0	0
							151.0	0
							1800.0	0
				20	0.11	14.09	90.0	0
							151.0	0
							1800.0	0
OES: Plastics Manufacturing								

SABIC INNOVATIVE PLASTICS US LLC BURKVILLE, AL NPDES: ALR16ECGK	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	0.03	4.08	90.0	0
							151.0	0
							1800.0	0
				20	0.41	51.12	90.0	1
							151.0	1
							1800.0	0
SABIC INNOVATIVE PLASTICS MT. VERNON, LLC MOUNT VERNON, IN NPDES: IN0002101	Surface Water	Active Releaser: NPDES IN0002101	Surface water	250	0.1	0.00491	90.0	0
							151.0	0
							1800.0	0
				20	1.40	0.0624	90.0	0
							151.0	0
							1800.0	0
SABIC INNOVATIVE PLASTICS US LLC SELKIRK, NY NPDES: NY0007072	Surface Water	Active Releaser: NPDES NY0007072	Surface water	250	0.03	0.00510	90.0	0
							151.0	0
							1800.0	0
				20	0.44	0.0641	90.0	0
							151.0	0
							1800.0	0
EQUISTAR CHEMICALS LP LA PORTE, TX NPDES: TX0119792	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	0.03	4.31	90.0	0
							151.0	0
							1800.0	0
				20	0.43	53.62	90.0	1
							151.0	1
							1800.0	0
CHEMOURS COMPANY FC LLC WASHINGTON, WV NPDES: WV0001279	Surface Water	Active Releaser: NPDES WV0001279	Surface water	250	0.03	0.00299	90.0	0
							151.0	0
							1800.0	0
				20	0.37	0.0371	90.0	0
							151.0	0
							1800.0	0
SHINTECH ADDIS PLANT A ADDIS, LA NPDES: LA0111023	Surface Water	Active Releaser: NPDES LA0055794	Surface water	250	0.01	0.0000417	90.0	0
							151.0	0
							1800.0	0
				20	0.13	0.000526	90.0	0
							151.0	0
							151.0	0

							1800.0	0
STYROLUTION AMERICA LLC CHANNAHON, IL NPDES: IL0001619	Surface Water	Active Releaser: NPDES IL0001619	Surface water	250	0.001	0.000230	90.0	0
							151.0	0
							1800.0	0
				20	0.01	0.00288	90.0	0
							151.0	0
							1800.0	0
DOW CHEMICAL CO DALTON PLANT DALTON, GA NPDES: GA0000426	Surface Water	Active Releaser: NPDES GA0000426	Surface water	250	0.001	0.00648	90.0	0
							151.0	0
							1800.0	0
				20	0.02	0.0811	90.0	0
							151.0	0
							1800.0	0
PREGIS INNOVATIVE PACKAGING INC WURTLAND, KY NPDES: KY0094005	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	0.0001	0.0116	90.0	0
							151.0	0
							1800.0	0
				20	0.0012	0.15	90.0	0
							151.0	0
							1800.0	0
OES: CTA Film Manufacturing								
KODAK PARK DIVISION ROCHESTER, NY NPDES: NY0001643	Surface Water	Active Releaser: NPDES NY0001643	Surface water	250	0.1	0.1100	90.0	0
							151.0	0
							1800.0	0
				20	1.4	1.36	90.0	0
							151.0	0
							1800.0	0
OES: Lithographic Printer								
FORMER REXON FACILITY AKA ENJEMS MILLWORKS WAYNE TWP, NJ NPDES: NJG218316	Surface Water	Active Releaser (Surrogate): Printing	Surface water	250	0.000004	0.0000540	90.0	0
							151.0	0
							1800.0	0
				20	0.000046	0.000677	90.0	0
							151.0	0
							1800.0	0
OES: Spot Cleaner								
				250	0.0002	0.00602	90.0	0

BOISE STATE UNIVERSITY BOISE, ID NPDES: IDG911006	Surface Water	Active Releaser (Surrogate): NPDES ID0020443	Surface water	20	0.0030	0.0753	151.0	0
							1800.0	0
							90.0	0
							151.0	0
							1800.0	0
OES: Recycling and Disposal								
JOHNSON MATTHEY WEST DEPTFORD, NJ NPDES: NJ0115843	Non- POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	250	2	147.01	90.0	68
							151.0	36
							1800.0	0
CLEAN HARBORS DEER PARK LLC LA PORTE, TX NPDES: TX0005941	Non- POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	250	2	123.89	90.0	56
							151.0	28
							1800.0	0
CLEAN HARBORS EL DORADO LLC EL DORADO, AR NPDES: AR0037800	Non- POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	250	0.5	26.68	90.0	5
							151.0	2
							1800.0	0
TRADEBE TREATMENT & RECYCLING LLC EAST CHICAGO, IN FRS: 110000397874	Non- POTW WWT	Receiving Facility: ADVANCED WASTE SERVICES OF INDIANA LLC and BEAVER OIL TREATMENT AND RECYCLING; POTW (Ind.)	Surface water	250	0.1	4.52	90.0	0
							151.0	0
							1800.0	0
VEOLIA ES TECHNICAL SOLUTIONS LLC WEST CARROLLTON, OH FRS: 110000394920	POTW	Receiving Facility: WESTERN REGIONAL WRF; NPDES OH0026638	Surface water	250	0.01	0.00785	90.0	0
							151.0	0
							1800.0	0
VEOLIA ES TECHNICAL SOLUTIONS LLC AZUSA, CA FRS: 110000477261	POTW	Receiving Facility: SAN JOSE CREEK WATER RECLAMATION PLANT; NPDES CA0053911	Surface water	250	0.002	0.00389	90.0	20
							151.0	20
							1800.0	20
VEOLIA ES TECHNICAL SOLUTIONS LLC MIDDLESEX, NJ NPDES: NJ0127477	Non- POTW WWT	Receiving Facility: MIDDLESEX COUNTY UTILITIES AUTHORITY; NPDES: NJ0020141	Still body	250	0.018	0.00504	90.0	0
							151.0	0
							1800.0	0
		Receiving Facility: Clean Harbors; POTW (Ind.)	Surface water	250	306	18100	90.0	250
							151.0	250
				250	0.01	1.84	1800.0	200
							90.0	0

CHEMICAL WASTE MANAGEMENT EMELLE, AL NPDES: AL0050580	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	20	0.18	23.20	151.0	0			
							1800.0	0			
							90.0	0			
							151.0	0			
							1800.0	0			
OILTANKING HOUSTON INC HOUSTON, TX NPDES: TX0091855	Surface Water	Active Releaser (Surrogate): NPDES TX0065943	Surface water	250	0.003	7.22	90.0	0			
							151.0	0			
							1800.0	0			
							90.0	0			
							20	0.041	90.00	151.0	0
1800.0	0										
HOWARD CO ALFA RIDGE LANDFILL MARRIOTTSVILLE, MD NPDES: MD0067865	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0002	0.0313	90.0	0			
							151.0	0			
							1800.0	0			
							20	0.0030	0.39	90.0	0
							151.0	0			
CLIFFORD G HIGGINS DISPOSAL SERVICE INC SLF KINGSTON, NJ NPDES: NJG160946	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0001	0.0124	90.0	0			
							151.0	0			
							1800.0	0			
							20	0.0012	0.16	90.0	0
							151.0	0			
CLEAN WATER OF NEW YORK INC STATEN ISLAND, NY NPDES: NY0200484	Surface Water	Active Releaser (Surrogate): NPDES NJ0000019	Still body	250	0.01	28.00	90.0	0			
							151.0	0			
							1800.0	0			
							20	0.12	352.94	90.0	20
							151.0	20			
FORMER CARBORUNDUM COMPLEX SANBORN, NY NPDES: NY0001988	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.001	0.13	90.0	0			
							151.0	0			
							1800.0	0			
							20	0.012	1.57	90.0	0
							151.0	0			
							1800.0	0			

OES: Other								
APPLIED BIOSYSTEMS LLC PLEASANTON, CA FRS: 110020517010	Non- POTW WWT	Receiving Facility: Evoqua Water Technologies; POTW (Ind.)	Surface water	250	0.2	10.02	90.0	1
							151.0	0
							1800.0	0
EMD MILLIPORE CORP JAFFREY, NH NPDES: NHR05C584	POTW	Receiving Facility: JAFFREY WASTEWATER TREATMENT FACILITY; NPDES NH0100595	Surface water	250	0.01	0.18	90.0	0
							151.0	0
							1800.0	0
GBC METALS LLC SOMERS THIN STRIP WATERBURY, CT NPDES: CT0021873	Surface Water	Active Releaser: NPDES CT0021873	Surface water	250	0.001	0.00491	90.0	0
							151.0	0
							1800.0	0
				20	0.009	0.0614	90.0	0
						151.0	0	
						1800.0	0	
HYSTER-YALE GROUP, INC SULLIGENT, AL NPDES: AL0069787	Surface Water	Active Releaser: Motor Vehicle Manuf.	Surface water	250	0.000001	0.000180	90.0	0
							151.0	0
							1800.0	0
				20	0.000012	0.00234	90.0	0
						151.0	0	
						1800.0	0	
AVNET INC (FORMER IMPERIAL SCHRADER) ELLENVILLE, NY NPDES: NY0008087	Surface Water	Active Releaser: Electronic Components Manuf.	Surface water	250	0.00002	0.0402	90.0	0
							151.0	0
							1800.0	0
				20	0.0002	0.50	90.0	0
						151.0	0	
						1800.0	0	
BARGE CLEANING AND REPAIR CHANNELVIEW, TX NPDES: TX0092282	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.0003	0.11	90.0	0
							151.0	0
							1800.0	0
				20	0.003	1.320	90.0	0
						151.0	0	
						1800.0	0	
AC & S INC NITRO, WV NPDES: WV0075621	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.00005	0.0188	90.0	0
							151.0	0
							1800.0	0

				20	0.001	0.24	90.0	0
							151.0	0
							1800.0	0
MOOG INC - MOOG IN-SPACE PROPULSION ISP NIAGARA FALLS, NY NPDES: NY0203700	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.00001	0.00485	90.0	0
							151.0	0
							1800.0	0
				20	0.0002	0.0602	90.0	0
							151.0	0
							1800.0	0
OILTANKING JOLIET CHANNAHON, IL NPDES: IL0079103	Surface Water	Active Releaser (Surrogate): NPDES IL0001619	Surface water	250	0.003	0.00088	90.0	0
							151.0	0
							1800.0	0
				20	0.032	0.0111	90.0	0
							151.0	0
							1800.0	0
NIPPON DYNAWAVE PACKAGING COMPANY LONGVIEW, WA NPDES: WA0000124	Surface Water	Active Releaser: NPDES WA0000124	Surface water	250	0.1	0.000703	90.0	0
							151.0	0
							1800.0	0
				20	1.090	0.00879	90.0	0
							151.0	0
							1800.0	0
TREE TOP INC WENATCHEE PLANT WENATCHEE, WA NPDES: WA0051527	Surface Water	Active Releaser (Surrogate): NPDES WA0023949	Surface water	250	0.00003	0.000000352	90.0	0
							151.0	0
							1800.0	0
				20	0.0004	0.00000440	90.0	0
							151.0	0
							1800.0	0
CAROUSEL CENTER SYRACUSE, NY NPDES: NY0232386	Surface Water	Active Releaser: POTW (Ind.)	Surface water	250	0.000002	0.000322	90.0	0
							151.0	0
							1800.0	0
				20	0.000031	0.00396	90.0	0
							151.0	0
							1800.0	0
OES: DoD								

US DOD USAF ROBINS AFB ROBINS AFB, GA NPDES: GA0002852	Surface Water	Active Releaser (Surrogate): NPDES GA0024538	Surface water	250	0.002	0.00182	90.0	0
							151.0	0
							1800.0	0
				20	0.023	0.0228	90.0	0
							151.0	0
							1800.0	0
OES: N/A (WWTP)								
EDWARD C. LITTLE WRP EL SEGUNDO, CA NPDES: CA0063401	Surface Water	Active Releaser (Surrogate): NPDES CA0000337	Still water	365	0.01	0.00601	90.0	0
							151.0	0
							1800.0	0
				20	0.19	0.11	90.0	0
							151.0	0
							1800.0	0
JUANITA MILLENDER- MCDONALD CARSON REGIONAL WRP CARSON, CA NPDES: CA0064246	Surface Water	Active Releaser (Surrogate): NPDES CA0000337	Still water	365	0.002	0.00127	90.0	0
							151.0	0
							1800.0	0
				20	0.04	0.0232	90.0	0
							151.0	0
							1800.0	0
LONDON WTP LONDON, OH NPDES: OH0041734	Surface Water	Active Releaser (Surrogate): NPDES OH0023779	Surface water	365	0.001	0.21	90.0	0
							151.0	0
							1800.0	0
				20	0.02	3.74	90.0	0
							151.0	0
							1800.0	0
LONG BEACH (C) WPCP LONG BEACH, NY NPDES: NY0020567	Surface Water	Active Releaser: NPDES NY0020567	Still water	365	7	322.14	90.0	365
							151.0	365
							1800.0	0
				20	136.49	5857.02	90.0	20
							151.0	20
							1800.0	20
MIDDLESEX COUNTY UTILITIES AUTHORITY	Surface Water	Active Releaser: NPDES NJ0020141	Still water	365	4	2.79	90.0	0
							151.0	0
							1800.0	0

SAYREVILLE, NJ NPDES: NJ0020141				20	81.68	50.90	90.0	0		
							151.0	0		
							1800.0	0		
JOINT WATER POLLUTION CONTROL PLANT CARSON, CA NPDES: CA0053813	Surface Water	Active Releaser: NPDES CA0053813	Still water	365	1.7	0.00665	90.0	0		
							151.0	0		
							1800.0	0		
						20	30.18	0.12	90.0	0
									151.0	0
									1800.0	0
HYPERION TREATMENT PLANT PLAYA DEL REY, CA NPDES: CA0109991	Surface Water	Active Releaser: NPDES CA0109991	Still water	365	0.5	0.00359	90.0	0		
							151.0	0		
							1800.0	0		
						20	8.22	0.0656	90.0	0
									151.0	0
									1800.0	0
SD CITY PT LOMA WASTEWATER TREATMENT SAN DIEGO, CA NPDES: CA0107409	Surface Water	Active Releaser: NPDES CA0107409	Still water	365	0.5	1.08	90.0	0		
							151.0	0		
							1800.0	0		
						20	8.22	19.74	90.0	0
									151.0	0
									1800.0	0
REGIONAL SANITATION DISTRICT ELK GROVE, CA NPDES: CA0077682	Surface Water	Active Releaser: NPDES CA0077682	Surface water	365	0.2	0.0151	90.0	0		
							151.0	0		
							1800.0	0		
						20	4.31	0.27	90.0	0
									151.0	0
									1800.0	0
BERGEN POINT STP & BERGEN AVE DOCK W BABYLON, NY NPDES: NY0104809	Surface Water	Active Releaser: NPDES NY0104809	Still water	365	0.2	3.65	90.0	0		
							151.0	0		
							1800.0	0		
						20	3.27	66.40	90.0	0
									151.0	0
									1800.0	0
Surface Water	Active Releaser: NPDES NY0026697	Still water	365	0.04	0.68	90.0	0			
						151.0	0			

NEW ROCHELLE STP NEW ROCHELLE, NY NPDES: NY0026697				20	0.77	12.47	1800.0	0
							90.0	0
							151.0	0
							1800.0	0
SIMI VLY CNTY SANITATION SIMI VALLEY, CA NPDES: CA0055221	Surface Water	Active Releaser: NPDES CA0055221	Surface water	365	0.02	0.82	90.0	142
							151.0	124
				20	0.330	14.88	1800.0	91
							90.0	10
OCEANSIDE OCEAN OUTFALL OCEANSIDE, CA NPDES: CA0107433	Surface Water	Active Releaser: NPDES CA0107433	Still water	365	0.01	0.66	151.0	0
							1800.0	0
				20	0.19	12.00	90.0	0
							151.0	0
SANTA CRUZ WASTEWATER TREATMENT PLANT SANTA CRUZ, CA NPDES: CA0048194	Surface Water	Active Releaser: NPDES CA0048194	Still water	365	0.01	0.11	1800.0	0
							90.0	0
				20	0.12	2.07	151.0	0
							1800.0	0
CORONA WWTP 1 CORONA, CA NPDES: CA8000383	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.005	0.61	90.0	0
							151.0	0
				20	0.09	11.10	1800.0	0
							90.0	0
BLIND BROOK SD WWTP RYE, NY NPDES: NY0026719	Surface Water	Active Releaser: NPDES NY0026719	Still water	365	0.003	0.17	151.0	0
							1800.0	0
				20	0.06	3.11	90.0	0
							151.0	0
				365	0.003	0.14	1800.0	0
							90.0	0

MCKINLEYVILLE CSD - WASTEWATER TREATMENT PLANT MCKINLEYVILLE, CA NPDES: CA0024490	Surface Water	Active Releaser: NPDES CA0024490	Surface water	20	0.05	2.47	151.0	0
							1800.0	0
							90.0	0
							151.0	0
							1800.0	0
SAN JOSE CREEK WATER RECLAMATION PLANT WHITTIER, CA NPDES: CA0053911	Surface Water	Active Releaser: NPDES CA0053911	Surface water	365	0.001	0.00556	90.0	29
							151.0	29
							1800.0	29
							90.0	2
							151.0	2
CARMEL AREA WASTEWATER DISTRICT TREATMENT FACILITY CARMEL, CA NPDES: CA0047996	Surface Water	Active Releaser: NPDES CA0047996	Still water	365	0.001	0.08	90.0	0
							151.0	0
							1800.0	0
							90.0	0
							151.0	0
CAMERON TRADING POST WWTP CAMERON, AZ NPDES: NN0021610	Surface Water	Active Releaser: POTW (Ind.)	Surface water	20	0.01	1.52	1800.0	0
							90.0	0
							151.0	0
							1800.0	0
							90.0	0
CITY OF RED BLUFF WASTEWATER RECLAMATION PLANT RED BLUFF, CA NPDES: CA0078891	Surface Water	Active Releaser: NPDES CA0078891	Surface water	365	0.001	0.000074	151.0	0
							1800.0	0
							90.0	0
							151.0	0
							1800.0	0
91ST AVE WASTEWATER TREATMENT PLANT TOLLESON, AZ NPDES: AZ0020524	Surface Water	Active Releaser: NPDES AZ0020524	Surface water	365	0.1	0.25	90.0	0
							151.0	0
							1800.0	0
							90.0	0
							151.0	0
				20	1.54	4.52	1800.0	0
							90.0	0
							151.0	0
							1800.0	0
							90.0	0

EVERETT WATER POLLUTION CONTROL FACILITY EVERETT, WA NPDES: WA0024490	Surface Water	Active Releaser: NPDES WA0024490	Surface water	365	0.1	0.85	90.0	0
							151.0	0
							1800.0	0
				20	1.50	15.54	90.0	0
							151.0	0
							1800.0	0
PIMA COUNTY - INA ROAD WWTP TUCSON, AZ NPDES: AZ0020001	Surface Water	Active Releaser: NPDES AZ0020001	Surface water	365	0.1	1.02	90.0	310
							151.0	310
							1800.0	303
				20	1.37	18.59	90.0	18
							151.0	18
							1800.0	17
23RD AVENUE WASTEWATER TREATMENT PLANT PHOENIX, AZ NPDES: AZ0020559	Surface Water	Active Releaser: NPDES AZ0020559	Surface water	365	0.1	0.14	90.0	0
							151.0	0
							1800.0	0
				20	0.95	2.49	90.0	0
							151.0	0
							1800.0	0
SUNNYSIDE STP SUNNYSIDE, WA NPDES: WA0020991	Surface Water	Active Releaser: NPDES WA0020991	Surface water	365	0.005	0.00611	90.0	0
							151.0	0
							1800.0	0
				20	0.08	0.11	90.0	0
							151.0	0
							1800.0	0
AGUA NUEVA WRF TUCSON, AZ NPDES: AZ0020923	Surface Water	Active Releaser: NPDES AZ0020923	Surface water	365	0.003	0.0292	90.0	303
							151.0	303
							1800.0	303
				20	0.06	0.53	90.0	17
							151.0	17
							1800.0	17
PORT OF SUNNYSIDE INDUSTRIAL WWTF SUNNYSIDE, WA NPDES: WA0052426	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.002	0.24	90.0	0
							151.0	0
							1800.0	0
				20	0.03	4.45	90.0	0
							151.0	0
							151.0	0

							1800.0	0
APACHE JUNCTION WWTP APACHE JUNCTION, AZ NPDES: AZ0023931	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.0003	0.04	90.0	0
							151.0	0
							1800.0	0
				20	0.0056	0.72	90.0	0
							151.0	0
							1800.0	0

- a. Facilities actively releasing dichloromethane were identified via DMR and TRI databases for the 2016 reporting year.
- b. Facilities actively releasing dichloromethane were identified via DMR and TRI databases for the 2016 reporting year.
- c. Release media are either direct (release from active facility directly to surface water) or indirect (transfer of wastewater from active facility to a receiving POTW or non-POTW WWTP facility). A wastewater treatment removal rate of 57% is applied to all indirect releases.
- d. If a valid NPDES of the direct or indirect releaser was not available in E-FAST, the release was modeled using either a surrogate representative facility in E-FAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.
- e. E-FAST uses either the “surface water” model, for rivers and streams, or the “still water” model, for lakes, bays, and oceans.
- f. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.
- g. The daily release amount was calculated from the reported annual release amount divided by the number of release days/yr.
- h. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.
- i. To determine the PDM days of exceedance for still bodies of water, the estimated number of release days should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.

Table Apx E-5. States with Monitoring Sites or Facilities in 2016

State Name	Methylene Dichloride Releasing Facility	Methylene Dichloride Monitoring Site	Methylene Dichloride Facility or Monitoring Site
Alabama	X		X
Arizona	X	X	X
California	X		X
Connecticut	X		X
Georgia	X		X
Idaho	X		X
Illinois	X		X
Indiana	X		X
Kansas		X	X
Kentucky	X		X
Louisiana	X		X
Maryland	X		X
Michigan	X		X
Minnesota		X	X
Missouri	X	X	X
New Hampshire	X		X
New Jersey	X	X	X
New Mexico		X	X
New York	X		X
North Carolina		X	X
Ohio	X		X
Pennsylvania		X	X
Tennessee	X	X	X
Texas	X	X	X
Washington	X		X
West Virginia	X		X
<i>Total</i>	21	10	26

Appendix F OCCUPATIONAL EXPOSURES

Appendix F.1 contains information gathered by EPA in support of understanding glove use for pure methylene chloride and for paint and coatings removal using methylene chloride formulations (<https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0231-0255>). This information may be generally useful for a broader range of uses of methylene chloride and is presented for illustrative purposes. Appendix F.2 contains a summary of information on gloves from Safety Data Sheets (SDS) for methylene chloride and formulations containing methylene chloride.

F.1 Information on Respirators and Gloves for Methylene Chloride including Paint and Coating Removal

Respirator Specifications

Table_Apx F-1 shows the specifications for respirators required to achieve the APFs shown in tables in Section 4.3 Human Health Risk. Assigned Protection Factors for Respirators in OSHA Standard 29 CFR 1910.134^a. Only respirators that meet OSHA requirements for routine exposures to methylene chloride are included in this table.

Table_Apx F-1. Respirator Specifications by APF for Use in Paint and Coating Removal Scenarios with Methylene Chloride Exposure

Assigned Protection Factor (APF)	Type of Respirator
10	No respirators with this APF meet OSHA requirements for routine exposures to methylene chloride. Any respirator listed in Table_Apx F-1 with APF greater than 10.
25	Any NIOSH-certified continuous flow supplied-air respirator equipped with a loose fitting facepiece, hood, or helmet. Any respirator listed in Table_Apx F-1 with APF greater than 25.
50	Any NIOSH-certified negative pressure (demand) supplied-air respirator equipped with a full facepiece. Any NIOSH-certified negative pressure (demand) self-contained breathing apparatus (SCBA) equipped with a hood, helmet, or a full facepiece. Any respirator listed in Table_Apx F-1 with APF greater than 50.
1,000	Any NIOSH-certified continuous flow supplied-air respirator equipped with a full facepiece.

Assigned Protection Factor (APF)	Type of Respirator
	<p>Any NIOSH-certified continuous flow supplied-air respirator equipped with a hood or helmet <i>with evidence demonstrating protection level of 1,000 or greater</i>. [See important note below].*</p> <p>Any NIOSH-certified pressure-demand or other positive pressure mode supplied-air respirator equipped with a full facepiece.</p> <p>Any respirator listed in Table Apx F-1 with APF greater than 1,000.</p>
10,000	Any NIOSH-certified pressure-demand or other positive-pressure mode (e.g., open/closed circuit) self-contained breathing apparatus (SCBA) equipped with a hood or helmet or a full facepiece.

Adapted from "OFFICE OF POLLUTION PREVENTION AND TOXIC'S (OPPT'S) DECISION LOGIC FOR SELECTION OF RESPIRATORS FOR PMN SUBSTANCES", May 2012.

OSHA has assigned APFs of 1000 for certain types of hoods and helmets with supplied air respirators (SARs) where the manufacturer can demonstrate adequate air flows to maintain positive pressure inside the hood or helmet in normal working conditions. However, the employer must have evidence provided by the respirator manufacturer that the testing of these respirators demonstrates performance at a level of protection of 1,000 or greater to receive an APF of 1,000. This level of performance can best be demonstrated by performing a Workplace Protection Factor or Simulated Workplace Protection Factor study or equivalent testing. **Without testing data that demonstrates a level of protection of 1,000 or greater, all SARs with helmets/hoods are to be treated as loose-fitting facepiece respirators and receive an APF of 25.**

Dermal Protection

OSHA indicates that dermal protection for workers exposed to methylene chloride is important. The information below provides information on glove protection when using pure methylene chloride or formulations containing methylene chloride.

Summary of Suitable Gloves for Pure Methylene Chloride and in Formulations

Several studies specified below indicate that gloves should be tested to determine whether they are protective against solvents when present in formulated products. According to these studies, the two best types of glove materials to protect against dermal exposure to pure methylene chloride are Silver Shield and Polyvinyl Alcohol (PVA), followed by Viton. Silver Shield gloves provide the best protection against methylene chloride whether it is in pure form or as part of a formulation. Detailed information on these and other glove types which were evaluated for their permeation characteristics against methylene chloride are provided below. The cited studies' results may be a good starting point for determining glove types to consider for glove testing.

Glove Information for Pure Methylene Chloride and for Methylene Chloride in Paint and Coating Removal Formulations

There are many factors that determine proper chemical-resistant glove selection. In addition to the specific chemical(s) used, the most important factors include duration, frequency, and severity of chemical exposure. The degree of dexterity required for the task and associated physical stress to the glove are also significant considerations. The manner in which employees are able to doff the various glove types to best prevent skin contamination is also important but sometimes overlooked.

Generally, dermal exposures to the solvents in paint and coating removal formulations may be assumed to be frequent or lengthy and may result in significant exposure. These assumptions affect the proper choice of glove type and also errs on the side of caution, which is advised for any personal protective equipment (PPE) decision since PPE is the last line of defense against exposure in an industrial hygienist's hierarchy of controls.

Table_Apx F-2 summarizes commonly used industrial hygiene literature (e.g., glove selection guides, manufacturer publications, etc.) and capture the highest rated glove types from each reference. Consideration of all factors (breakthrough time, qualitative indicator (QI), and other issues raised in the comments field) allow an overall determination of effectiveness.

Table_Apx F-2. Glove Types Evaluated for Pure Methylene Chloride

Reference	Glove type	Breakthrough Time	Qualitative Indicator	Comments
1	Polyvinyl Alcohol (PVA)	>360 mins	Very well suited	Degradation rate: Good Permeation rate: Excellent
	Viton/Butyl	29 mins	Suitable under careful control of use	Degradation rate: Excellent Permeation rate: Good
	Ansell Barrier (Laminate Film) Glove	20 mins	Suitable under careful control of use	Degradation rate: Excellent Permeation rate: Very Good
2	Viton	113 mins	Satisfactory	Change soon after exposure. Product is Best Viton 890
3	PVA	Not Provided	Recommended	Extended contact
	Viton	Not Provided	Recommended	Extended contact
	Nitrile	Not Provided	See Comment	Double-gloved 8-mil Nitrile gloves are only acceptable for "incidental contact". Change immediately

Reference	Glove type	Breakthrough Time	Qualitative Indicator	Comments
4	Silver Shield	>8 hrs	Good for total immersion	Degradation Rate: Excellent
	Viton	1 hr	Good for accidental splash protection and intermittent contact	Degradation Rate: Fair
5	PVA	Not Provided	Best protection	*Detailed comments provided in footnote
	Viton	Not Provided	Recommended	
	Nitrile	≤ 4 mins (thin)	Poor	
	Latex	Seconds	Very Poor	
6	Latex	Not Provided	NOT recommended	This source only evaluates latex and nitrile gloves
	Nitrile	Not Provided	NOT recommended	
7	Viton	“Generally greater than 4 hrs”	Good	Silver Shield and PVA are not evaluated by this source
	Nitrile	“Generally greater than 1 hr”	Fair	
8	Fluoroelastomer (Viton)	64 mins	Use for high chemical exposure	Specific glove evaluated is Fluonit 468
9	Silver Shield (North)	>6 hrs	Excellent	Degradation rate: Excellent
	PVA	>6 hrs	Good	Degradation rate: Good
10	Silver Shield (North)	Not Provided	Not Provided	Silver Shield and PVA gloves are the only two glove types recommended by this source
	PVA	Not Provided	Not Provided	

*Detailed comments from Cornell University Hand Protection and Glove Selection Guide: “Double glove with heavier weight (8 mil) nitrile gloves (incidental contact). Methylene chloride will permeate through thin (3-4 mil) nitrile gloves in four minutes or less. If you are double gloved, as recommended, and you splash or spill methylene chloride on your gloves, stop what you are doing and change the outer glove immediately. If you allow methylene chloride to remain on the outer nitrile glove for more than two to four minutes you must discard both sets of gloves and re-double glove. Methylene chloride permeates

disposable latex exam gloves in a matter of seconds and latex gloves should never be used to handle this material. For use of methylene chloride where contact with the glove is anticipated, such as stripping paint or gluing plastics, only polyvinyl acetate (PVA) or Viton gloves are recommended. These gloves come in .28-.33 mm thickness. PVA offers the best protection” (Cornell University).

Based on the information from Table_Apx F-2, the two best types of glove materials to protect against pure methylene chloride dermal exposure are Silver Shield and PVA (highlighted green above), followed by Viton. Silver Shield is a trade name and is generally regarded as the most protective glove type for the majority of chemicals. They are composed of laminate-layered polyethylene (PE)/ethylene vinyl alcohol (EVOH) materials. However, Silver Shield gloves do not provide much dexterity and because of this are commonly used in conjunction with a second tight-fitting glove of a different type over the top. Alternatively, PVA gloves could be worn and would provide significant protection. These conclusions are in agreement with OSHA’s recommendation from a Hazard Alert published in January of 2013 entitled “Methylene Chloride Hazards for Bathtub Refinishers,” where methylene chloride is used for paint/ coating removal ([OSHA; NIOSH, 2013](#)). The Hazard Alert states that “gloves made of PE)/ EVOH or other laminate materials that are resistant to methylene chloride are recommended to meet the requirements of the standard” (OSHA Hazard Alert).

Key Points and Examples for Paint and Coating Removal Formulations

The U.S. EPA’s Safety, Health and Environmental Management Division’s (SHEMD) Guideline 44 (Personal Protective Equipment) states that when working with mixtures and formulated products, the chemical component with the shortest break-through time must be considered when determining the appropriate glove type for protection against chemical hazards unless specific test data are available ([Enander et al., 2004](#)). Additionally, an industrial hygienist will consider the formulation’s chemical properties as a whole, the highest hazard component of the formulation, and whether individual components produce synergistic degradation effects. Typically, specific test data for formulations are not available and best judgment based on the aforementioned considerations provides the basis for glove type selection. However, in this case there are a few publications that specifically address glove types for use with methylene chloride and N-Methylpyrrolidone (NMP) as part of paint and coating removal formulations.

In early 2002, an article entitled “A Comparative Analysis of Glove Permeation Resistance to Paint Stripping Formulations” ([Stull et al., 2002](#)) specifically examined which glove types provide the best protection to users of commercial paint and coating removal products. Twenty different glove types were evaluated for degradation and resistance to permeation under continuous and/or intermittent contact with seven different paint and coating removal formulations in a multiple-phase experiment. Paint and coating removal formulations included some that were methylene chloride-based and others that were NMP-based. The study found that gloves made of Plastic Laminate (e.g., Silver Shield) resisted permeation by the majority of paint and coating removal while Butyl Rubber provided the next best level of permeation resistance against the majority of formulations. However, Butyl Rubber gloves did show rapid permeation for methylene chloride-based formulations and would not be recommended for methylene chloride. It should be noted that PVA gloves, shown to be effective against pure methylene chloride, were not evaluated. Interestingly, more glove types resisted permeation of NMP-based formulations than conventional solvent-based products such as methylene chloride. The results showed that relatively small-molecule, volatile, chemical-based solvents cause somewhat more

degradation and considerably more permeation of glove types as compared with NMP-based formulations against the same gloves. Key conclusions include the following: “However, paint stripper formulations represent varying multichemical mixtures and, ultimately, commercial paint strippers must be individually evaluated for permeation resistance against selected gloves” (Stull et al., 2002), and, “because of several potential synergistic effects well established in the literature and in this study for mixture permeation, it is highly recommended that glove selection decisions be based on testing of the commercial paint stripper against the specific glove in question”(Stull et al., 2002).

Another study from in 2007 entitled “Protective Glove Selection for Workers using NMP-Containing Products: Graffiti Removal” essentially came to the same conclusion; of the gloves studied Silver Shield gloves provide the best protection against NMP-based paint and coating removal formulations (HSL, 2007). The study states that “Butyl gloves, used with caution would be a second choice” (HSL, 2007). The increased dexterity and robustness of Butyl gloves were noted as an advantage of Butyl over Silver Shield. Key recommendations include that gloves should be “tested against all relevant chemical formulations as a matter of routine in order to inform glove selection” (HSL, 2007) and “assumptions of glove choice based on the use of model compounds or similar formulations should be made with extreme caution (HSL, 2007).” Additionally, Crook recommended that “The BS EN 374-3 continuous contact test and its successors should remain the benchmark for chemically protective glove type decisions” (HSL, 2007).

In summary, these studies indicate that glove permeation continuous contact testing of each formulation is necessary to provide proper protection. These studies’ results may be a good starting point for determining glove types to consider for permeation testing. The studies found that among gloves tested Silver Shield provide the best protection against both methylene chloride and NMP, whether they are in pure form or as part of a formulation. The best alternative for protection against methylene chloride would be PVA gloves, while the best alternative for NMP protection would be Butyl Rubber gloves. There are other glove type materials with varied effectiveness that could potentially be appropriate for use with incidental contact. However, these conclusions are based on lengthy, often, and significant exposure. A more task-specific decision on appropriate glove type selection could be made through employee interviews and observation of tasks using methylene chloride- or NMP-containing products.

References for Appendix F.1

All Safety Products: <http://www.allsafetyproducts.com/asp-glove-selection-chart-chemical-break-through-times.html>, accessed 3/14/15.

Ansell Healthcare, LLC:

http://www.ansellpro.com/download/Ansell_8thEditionChemicalResistanceGuide.pdf, accessed 3/14/15.

California Dept. of Public Health:

<http://www.cdph.ca.gov/programs/ohb/Documents/PPEChart.pdf>, accessed 3/14/15.

Cornell University Hand Protection and Glove Selection Guide:
http://collum.chem.cornell.edu/documents/Hand_Protection_and_Glove_Selection.pdf, accessed 3/14/15.

Cornell University Lab Safety Manual: <http://sp.ehs.cornell.edu/lab-research-safety/laboratory-safety-manual/Pages/Appendix-F.aspx>, accessed 3/14/15.

Crook V, Simpson A (2007). Protective Glove Selection for Workers using NMP-Containing Products: Graffiti Removal. Buxton: Health and Safety Laboratory.

Microflex Corporation:
http://www.microflex.com/Products/~~/media/Files/Literature/Domestic%20Reference%20Materials/DOM_Reference_Chemical%20Resistance.ashx, accessed 3/14/15.

MAPA Professional: <http://www.mapa-pro.com/hand-protection-selection-guide/protections/chemical-protection.html>, accessed 3/14/15.

North by Honeywell: Chemical Resistance Guide:
http://www.honeywellsafety.com/Products/Gloves/SilverShield_-_SSG29.aspx?site=/usa,%20Document%202948_pdf, accessed 3/14/15.

Northwestern University:
http://www.northwestern.edu/userservices/docs/labs/SafetyTrainer_gloveselection.pdf, accessed 3/14/15.

Occupational Health and Safety Administration (OSHA) Hazard Alert. Methylene Chloride Hazards for Bathtub Refinishers. January 2013.
https://www.osha.gov/dts/hazardalerts/methylene_chloride_hazard_alert.pdf

Showa Best Glove: <http://www.showabestglove.com/site/chemrest/default.aspx>, accessed 3/14/15.

Stull JO, Thomas RW, James LE (2002). A Comparative Analysis of Glove Permeation Resistance to Paint Stripping Formulations, AIHA Journal, 63:1, 62-71.

U.S. EPA Safety, Health and Environmental Management Division (SHEMD). Guideline 44, Personal Protective Equipment. October 2004.

F.2 Summary of Information on Gloves from SDS for Methylene Chloride and Formulations containing Methylene Chloride

EPA reviewed SDSs for neat methylene chloride and products containing methylene chloride for information on glove and respiratory protection. Specifically, EPA reviewed SDSs for each occupational scenario assessed in Section 2.4.1.2. EPA compiled the recommended glove

materials and respiratory protection for each scenario from the reviewed SDSs (total of 18 SDSs were reviewed) in Table_Apx F-2. For neat methylene chloride and methylene chloride-containing products, the SDSs recommend a variety of glove materials, including fluorinated rubbers (7 SDSs), PVA (6 SDSs), nitrile rubber (5 SDSs), neoprene (4 SDSs), polyvinyl chloride (3 SDSs), and various laminates. Note that many of the reviewed SDSs included multiple glove material recommendations.

Table Apx F-3. Recommended Glove Materials Methylene Chloride and Methylene Chloride-Containing Products from SDSs

Applicable OES	Methylene Chloride wt. %	Recommended Glove Material	Source
Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products), Cold Cleaning	30-40%	EVAL, neoprene, nitrile/Buna-N, PVC, or Viton	https://www.berrymanproducts.com/assets/2AA-E-0901-0905-0955-SDS-1.pdf
Manufacturing	99.9%	PVA, ethyl vinyl alcohol laminate, Viton, butyl rubber	http://208.112.58.204/pridesol/documents/sds/Methylene%20Chloride%20Tech%20-%20Dow%20-%202015-03-04.pdf
Batch Open-Top Vapor Degreasing; Conveyorized Vapor Degreasing; Manufacturing	99.5%	Chemical-resistant gloves	http://208.112.58.204/pridesol/documents/sds/Methylene%20Chloride%20VDG%20-%20Dow%20-%202015-04-01.pdf
Paints and Coatings; Flexible Polyurethane Foam Manufacturing; Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	99.97-100%	Chemical-resistant gloves	http://www.silverfermchemical.com/media/42759/SC-Methylene-Chloride-SDS-signed.pdf
Manufacturing; Laboratory Use	90-100%	Fluorinated rubber	https://www.nwmissouri.edu/naturalsciences/sds/d/Dichloromethane.pdf
Adhesives and Sealants; Processing - Incorporation into Formulation, Mixture, or Reaction Product	60-85%	Fluoroelastomer polymer laminate	https://multimedia.3m.com/mws/mediawebserver?mwsId=SSSSSuUn_zu8l00xM82SNY_Bnv70k17zHvu9lxtD7SSSSSS--
Adhesives and Sealants	80-90%	Chemical-resistant gloves	http://www.camie.com/sites/default/files/msds/camie-sds313B.pdf
Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products)	25-35%	Suitable gloves	https://www.dodgepackaging.net/msds/B-00002.PDF
Spot Cleaning	35-45%	Butyl rubber, chlorinated polyethylene, polyethylene, ethyl vinyl alcohol laminate, PVA, natural rubber, neoprene, nitrile/butadiene rubber, PVC, Viton	https://www.msdsdigital.com/sites/default/files/msds_record_database/1005.pdf

Applicable OES	Methylene Chloride wt.%	Recommended Glove Material	Source
Fabric Finishing; Spot Cleaning	70 - < 90%	PVA	https://www.davisint.com/Images/document/TS-VLR-Eng-US-SDS-GHS.pdf
Spot Cleaning	40-50%	Impervious gloves	http://www.allopar.com/wp-content/uploads/2015/05/spot-lifter-2.pdf
Paints and Coatings; Non-Aerosol Industrial and Commercial Uses	60-100%	Laminate film, nitrile rubber, neoprene, and PVC	https://gooffproducts.com/wp-content/uploads/2017/08/SprayableStripperMSDS.pdf
Laboratory Use	≥25 - ≤49%	Chemical-resistant gloves	https://www.agilent.com/cs/library/msds/5190-0487_NAEnglish.pdf
Paints and Coatings; Non-Aerosol Industrial and Commercial Uses	44-78%	Rubber or nitrile	https://www.antiseize.com/PDFs/m17052.pdf
Lithographic Printing Plate Cleaning	30-60%	PVA, Viton rubber (fluoro rubber)	http://www.lehmaninc.com/customer/leinco/pdf11/MSDS/Allied/msds-al-10034.pdf
Paints and Coatings; Flexible Polyurethane Foam Manufacturing; Commercial Aerosol Products (Aerosol Degreasing, Aerosol Lubricants, Automotive Care Products); Laboratory Use; Plastic Product Manufacturing; CTA Film Production	100%	Ansell laminate film (Barrier), or supported PVA	https://www.chemsupply.com.au/documents/MA0121CH2L.pdf
Adhesive and Caulk Removers	60-100%	Laminate film, nitrile rubber, neoprene, and PVC	http://www.kleanstrip.com/uploads/documents/GKAS94326_SDS-4015.34.pdf
Processing as a Reactant	0-0.5%	PVA, Viton	http://www.certifiedacpro.com/datasheets/msds/345_MSDS.pdf

Appendix G CONSUMER EXPOSURES

See the following supplemental documents:

- *Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Input Parameters* ([EPA, 2019i](#))
- *Risk Evaluation for Methylene Chloride, Supplemental Information on Consumer Exposure Assessment Model Outputs* ([EPA, 2019j](#))
- *Risk Evaluation for Methylene Chloride, Supplemental Information: Consumer Risk Calculator Dermal* ([EPA, 2020b](#))
- *Risk Evaluation for Methylene Chloride, Supplemental Information: Consumer Risk Calculator Inhalation* ([EPA, 2020c](#))

G.1 Consumer Exposure

Consumer exposure was evaluated utilizing a modeling approach because emissions and chemical specific personal monitoring data associated with consumer use of products containing methylene chloride were not identified during data gathering and literature searches performed as part of EPA's Systematic Review process. A detailed discussion of the approaches taken to evaluate consumer inhalation exposure is provided in Section 2.4.2.

G.2 Consumer Inhalation Exposure

To evaluate consumer inhalation exposures, EPA's Consumer Exposure Model (CEM) was used. EPA varied three key parameters when modeling consumer inhalation exposure to capture a range of potential exposure scenarios. The key parameters varied were duration of use per event (minutes/use), amount of chemical in the product (weight fraction), and mass of product used per event (gram(s)/use). These key parameters were varied because CEM is sensitive to all three parameters and they are representative of expected consumer behavior patterns for product use (based on survey data).

Modeling was conducted for all possible combinations of the three varied parameters. This results in a maximum of 27 different iterations for each consumer use as summarized in Table_Apx G-G-1.

Table_Apx G-G-1. Example Structure of CEM Cases Modeled for Each consumer Product Use Scenario.

CEM Set	Scenario Characterization (Duration-Weight Fraction-Product Mass)	Duration of Product Use Per Event (min/use) [not scalable]	Weight Fraction of Chemical in Product (unitless) [scalable]	Mass of Product Used (g/use) [scalable]
Set 1 (Low Intensity Use)	Case 1: Low-Low-Low	Low	Low	Low
	Case 2: Low-Low-Mid			Mid
	Case 3: Low-Low-High			High
	Case 4: Low-Mid-Low		Mid	Low

	Case 5: Low-Mid-Mid		High	Mid
	Case 6: Low-Mid-High			High
	Case 7: Low-High-Low			Low
	Case 8: Low-High-Mid			Mid
	Case 9: Low-High-High			High
Set 2 (Moderate Intensity Use)	Case 10: Mid-Low-Low	Mid	Low	Low
	Case 11: Mid-Low-Mid			Mid
	Case 12: Mid-Low-High			High
	Case 13: Mid-Mid-Low		Mid	Low
	Case 14: Mid-Mid-Mid			Mid
	Case 15: Mid-Mid-High			High
	Case 16: Mid-High-Low		High	Low
	Case 17: Mid-High-Mid			Mid
	Case 18: Mid-High-High			High
Set 3 (High Intensity Use)	Case 19: High-Low-Low	High	Low	Low
	Case 20: High-Low-Mid			Mid
	Case 21: High-Low-High			High
	Case 22: High-Mid-Low		Mid	Low
	Case 23: High-Mid-Mid			Mid
	Case 24: High-Mid-High			High
	Case 25: High-High-Low		High	Low
	Case 26: High-High-Mid			Mid
	Case 27: High-High-High			High

G.3 Consumer Dermal Exposure

Two models were used to evaluate consumer dermal exposures, the CEM (Fraction Absorbed) model and the CEM (Permeability) model. A brief comparison of these two dermal models through the calculation of acute dose rate (ADR) is provided below. This is followed by comparison of results from both models for all fifteen conditions of use evaluated for dermal exposure for the adult age group. Finally, a brief discussion on a sensitivity analysis of the overall model and for the two evaluated dermal models is provided along with explanations on selection and utilization for evaluated dermal exposure

G.3.1 Comparison of Two Dermal Model Methodologies to Calculate Acute Dose Rate (ADR)

CEM (Permeability) Model: The CEM (Permeability) model estimates acute dose rates based primarily on the permeability coefficient of the chemical of concern and duration of use. The CEM (Permeability) model assumes a constant supply of product on the skin throughout the exposure duration and does not consider evaporation from the skin. The CEM (Permeability) model estimates the acute dose rate (ADR) using the following equation:

Equation_Apx G-1. CEM Permeability Model, Acute Dose Rate

$$ADR = \frac{K_p \times D_{ac} \times Dil \times \rho \times \frac{SA}{BW} \times FQ_{ac} \times WF \times ED_{ac} \times CF_1}{AT_{ac} \times CF_2}$$

Where:

ADR = Potential Acute Dose Rate (mg/kg-day)

K_p = Permeability coefficient (cm/hr)

D_{ac} = Duration of use (min/event), acute

Dil = Product dilution fraction (unitless)

ρ = Density of formulation (g/cm³)

$\frac{SA}{BW}$ = Surface area to body weight ratio (cm²/kg)

FQ_{ac} = Frequency of use, acute (events/day)

WF = Weight fraction of chemical in product (unitless)

ED_{ac} = Exposure Duration, acute (days)

CF_1 = Conversion factor (1000 mg/g)

AT_{ac} = Averaging time, acute (days)

CF_2 = Conversion factor (60 min/hr)

The key inputs driving this calculation are the permeability coefficient (K_p), duration of use, product density (ρ), and weight fraction (WF). The K_p is particularly important in this calculation because its values can vary widely for a single chemical depending on the literature or estimation source. The CEM (Permeability) model the permeability coefficient is estimated as a function of the permeation coefficients of the lipid medium, protein fraction of the stratum corneum, and the water epidermal layer utilizing the following equation:

Equation_Apx G-2. CEM Permeability Model, Permeability Coefficient K_p

$$K_p = \frac{1}{\left(\frac{1}{K_{lip} + K_{pol}}\right) + \left(\frac{1}{K_{aq}}\right)}$$

Where:

K_p = Permeability coefficient for chemical transport through the SC from an aqueous vehicle (cm/hr)

K_{lip} = Permeation coefficient of the lipid medium

K_{pol} = Permeation coefficient of the protein fraction of the SC

K_{aq} = Permeation coefficient of water (epi)dermal layer

CEM (Fraction Absorbed) Model: The CEM (Fraction Absorbed) model estimates dermal exposure for products that are applied on the skin in a thin film and partially absorbed. This partial absorption is modeled by an absorption fraction which accounts for the amount of substance that penetrates across the absorption barriers of an organism. The CEM (Fraction Absorbed) model requires an assumption that the entire mass of the chemical of concern within the thin film enters the skin surface (stratum corneum) to correctly apply the absorption fraction. Utilizing this assumption, the CEM (Fraction Absorbed) model estimates the (ADR) using the following equation:

Equation_Apx G-3. CEM Absorption Fraction Model, Acute Dose Rate

$$ADR = \frac{AR \times \frac{SA}{BW} \times FQ_{ac} \times FR_{abs} \times Dil \times WF \times ED_{ac} \times CF_1}{AT_{ac}}$$

Where:

ADR = Potential Acute Dose Rate (mg/kg-day)

AR = Amount retained on the skin (g/cm²-event)

$\frac{SA}{BW}$ = Surface area to body weight ratio (cm²/kg)

FQ_{ac} = Frequency of use, acute (events/day)

FR_{abs} = Absorption fraction (unitless)

Dil = Product dilution fraction (unitless)

WF = Weight fraction of chemical in product (unitless)

ED_{ac} = Exposure duration, acute (days)

CF_1 = Conversion factor (1000 mg/g)

AT_{ac} = Averaging time, acute (days)

All terms listed in the above equation are singular inputs except AR , the amount retained on skin, and FR_{abs} , the absorption fraction (or fraction absorbed). The amount retained on skin (AR) represents the amount of product remaining on the skin after use, and is in the units of grams of product per square centimeter of skin area.

Equation_Apx G-4, shows the AR variable can be calculated as a product of the film thickness of the liquid on the skin's surface (FT) and the density of the product (ρ), subtracting any removal that may occur through washing or other removal methods.

Equation_Apx G-4. CEM Absorption Fraction Model, Amount Retained on Skin

$$AR = FT \times \rho \times (1 - \text{FracRemove})$$

The absorption fraction (FR_{abs}) represents how much of the available material can be absorbed into the skin and can be estimated through an exponential function defined primarily by D , the duration of use, and χ , the ratio of the evaporation rate from the stratum corneum surface to the dermal absorption rate through the stratum corneum. The equation for FR_{abs} , Equation_Apx G-5, is a simplification of the equation used by Frasch ([Frasch and Bunge, 2015](#))

Equation_Apx G-5. CEM Absorption Fraction Model, Fraction Absorbed

$$FR_{abs} = \frac{3 + \chi \left[1 - \exp \left(-\alpha \frac{D_{cr}}{t_{lag} \times CF_1} \right) \right]}{3(1 + \chi)}$$

Where:

χ = Ratio of the evaporation rate from the SC surface to the dermal absorption rate through the SC (unitless)

α = Constant (2.906)

D_{cr} = Duration of use (min)

t_{lag} = Lag time for chemical transport through the SC (hr)

CF_1 = Conversion factor (60 min/hr)

The equation for χ , Equation_Apx G-6, relies on chemical properties like molecular weight and vapor pressure, making χ values chemical-specific.

Equation_Apx G-6. CEM Absorption Fraction Model, χ

$$\chi = \frac{h \times P_{vap} \times MW \times CF_1}{K_p \times S_w \times R \times T}$$

Where:

h = Gas phase mass transfer coefficient (m/hr)

P_{vap} = Vapor Pressure (Torr)

MW = Molecular weight (mg/mmol)

K_p = Permeability coefficient for chemical transport through the SC from an aqueous vehicle (cm/hr)

S_w = Water solubility (mg/mL)

R = Real gas constant (62.37 mL-Torr/K-mmol)

T = Temperature (Kelvin)

CF_1 = Conversion factor (100 cm/m)

After simplifying the acute dose rate equation and substituting in for constants, the CEM Absorption Fraction acute dose rate becomes a function of the product density, film thickness,

G.3.2 Comparison of Estimated ADRs Across the Two Dermal Models

The three dermal models described in Section **Comparison of Two Dermal Model Methodologies to Calculate Acute Dose Rate (ADR)** G.3.1 were each run for all eight conditions of use for which consumer dermal exposure was evaluated. The purpose was to allow a comparison between the two results while recognizing each model is unique in its approach to estimating dermal exposure and may not be directly comparable. Keeping these limitations in mind, 2.4.2.4 shows the results from all three dermal models for each condition of use evaluated for dermal exposure.

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

Condition of Use	Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Fraction Absorbed Acute ADR (mg/kg/day) ²	Permeability Acute ADR (mg/kg/day) ²
Adhesives	High Intensity User	95% (60)	Max (90)	Adult (≥21 years)	2.55E+00	1.33E+01
				Youth (16-20 years)	2.38E+00	1.24E+01
				Youth (11-15 years)	2.60E+00	1.36E+01
		50%	Mid	Adult (≥21 years)	6.02E-01	6.27E-01

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

	Moderate Intensity User	(4.25)	(60)	Youth (16-20 years)	5.63E-01	5.87E-01
				Youth (11-15 years)	6.16E-01	6.41E-01
	Low Intensity User	10% (0.33) ¹	Min (30)	Adult (≥21 years)	4.30E-02	3.69E-02
				Youth (16-20 years)	4.02E-02	3.45E-02
			Youth (11-15 years)	4.40E-02	3.77E-02	
Adhesive Remover	High Intensity User	95% (480)	Max (75)	Adult (≥21 years)	8.63E+00	1.79E+02
				Youth (16-20 years)	8.07E+00	1.68E+02
				Youth (11-15 years)	8.83E+00	1.83E+02
	Moderate Intensity User	50% (60)	Max (75)	Adult (≥21 years)	8.61E+00	2.24E+01
				Youth (16-20 years)	8.06E+00	2.10E+01
				Youth (11-15 years)	8.81E+00	2.29E+01
	Low Intensity User	10% (3)	Min (50)	Adult (≥21 years)	1.53E+00	7.47E-01
				Youth (16-20 years)	1.43E+00	6.99E-01
				Youth (11-15 years)	1.56E+00	7.64E-01
	Auto Leak Sealer	High Intensity User	95% (120)	Single Value (1)	Adult (≥21 years)	4.11E-02
Youth (16-20 years)					3.84E-02	2.00E-01
Youth (11-15 years)					4.20E-02	2.18E-01
Moderate Intensity User		50% (15)	Single Value (1)	Adult (≥21 years)	3.23E-02	2.67E-02
				Youth (16-20 years)	3.02E-02	2.49E-02
				Youth (11-15 years)	3.30E-02	2.73E-02
Low Intensity User		10% (5)	Single Value (1)	Adult (≥21 years)	1.65E-02	8.89E-03
				Youth (16-20 years)	1.54E-02	8.32E-03
				Youth (11-15 years)	1.69E-02	9.09E-03
Auto AC Refrigerant	High Intensity User	95% (120)	Max (3)	Adult (≥21 years)	1.50E-01	7.78E-01
				Youth (16-20 years)	1.40E-01	7.28E-01
				Youth (11-15 years)	1.53E-01	7.96E-01
	Moderate Intensity User	50% (15)	Max (3)	Adult (≥21 years)	1.18E-01	9.72E-02
				Youth (16-20 years)	1.10E-01	9.10E-02
				Youth (11-15 years)	1.20E-01	9.95E-02
	Low Intensity User	10% (5)	Min (1)	Adult (≥21 years)	2.01E-02	1.08E-02
				Youth (16-20 years)	1.88E-02	1.01E-02
				Youth (11-15 years)	2.05E-02	1.11E-02
Brake Cleaner		95%	Max	Adult (≥21 years)	9.49E+00	4.93E+01

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

	High Intensity User	(120)	(65)	Youth (16-20 years)	8.88E+00	4.61E+01
				Youth (11-15 years)	9.71E+00	5.05E+01
	Moderate Intensity User	50% (15)	Mid (35)	Adult (\geq 21 years)	4.35E+00	3.60E+00
				Youth (16-20 years)	4.07E+00	3.36E+00
				Youth (11-15 years)	4.45E+00	3.68E+00
	Low Intensity User	10% (1)	Min (10)	Adult (\geq 21 years)	1.55E-01	6.85E-02
				Youth (16-20 years)	1.45E-01	6.41E-02
				Youth (11-15 years)	1.59E-01	7.01E-02
	Brush Cleaner	High Intensity User	95% (420)	Single Value (1)	Adult (\geq 21 years)	3.51E-02
Youth (16-20 years)					3.28E-02	3.17E+00
Youth (11-15 years)					3.59E-02	3.47E+00
Moderate Intensity User		50% (60)	Single Value (1)	Adult (\geq 21 years)	3.50E-02	4.84E-01
				Youth (16-20 years)	3.27E-02	4.53E-01
				Youth (11-15 years)	3.58E-02	4.96E-01
Low Intensity User		10% (5)	Single Value (1)	Adult (\geq 21 years)	1.41E-02	4.04E-02
				Youth (16-20 years)	1.32E-02	3.78E-02
				Youth (11-15 years)	1.44E-02	4.13E-02
Carbon Remover	High Intensity User	95% (120)	Max (70)	Adult (\geq 21 years)	8.46E+00	4.39E+01
				Youth (16-20 years)	7.91E+00	4.11E+01
				Youth (11-15 years)	8.65E+00	4.50E+01
	Moderate Intensity User	50% (15)	Max (70)	Adult (\geq 21 years)	6.65E+00	5.49E+00
				Youth (16-20 years)	6.22E+00	5.14E+00
				Youth (11-15 years)	6.80E+00	5.62E+00
	Low Intensity User	10% (2)	Min (40)	Adult (\geq 21 years)	8.99E-01	4.18E-01
				Youth (16-20 years)	8.42E-01	3.92E-01
				Youth (11-15 years)	9.20E-01	4.28E-01
Carburetor Cleaner	High Intensity User	95% (45)	Max (70)	Adult (\geq 21 years)	8.09E+00	1.59E+01
				Youth (16-20 years)	7.57E+00	1.49E+01
				Youth (11-15 years)	8.28E+00	1.63E+01
	Moderate Intensity User	50% (7)	Mid (45)	Adult (\geq 21 years)	2.69E+00	1.59E+00
				Youth (16-20 years)	2.52E+00	1.49E+00
				Youth (11-15 years)	2.76E+00	1.63E+00
			10%	Min	Adult (\geq 21 years)	2.29E-01

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

	Low Intensity User	(1)	(20)	Youth (16-20 years)	2.14E-01	9.45E-02
				Youth (11-15 years)	2.34E-01	1.03E-01
Coil Cleaner	High Intensity User	95% (120)	Max (100)	Adult (\geq 21 years)	1.38E+01	7.19E+01
				Youth (16-20 years)	1.29E+01	6.73E+01
				Youth (11-15 years)	1.42E+01	7.35E+01
	Moderate Intensity User	50% (15)	Max (100)	Adult (\geq 21 years)	1.09E+01	8.98E+00
				Youth (16-20 years)	1.02E+01	8.41E+00
				Youth (11-15 years)	1.11E+01	9.19E+00
	Low Intensity User	10% (2)	Min (60)	Adult (\geq 21 years)	1.55E+00	7.19E-01
				Youth (16-20 years)	1.45E+00	6.73E-01
				Youth (11-15 years)	1.58E+00	7.35E-01
Cold Pipe Insulation	High Intensity User	95% (60)	Max (60)	Adult (\geq 21 years)	2.97E+00	7.72E+00
				Youth (16-20 years)	2.78E+00	7.23E+00
				Youth (11-15 years)	3.04E+00	7.90E+00
	Moderate Intensity User	50% (5)	Max (60)	Adult (\geq 21 years)	1.20E+00	6.44E-01
				Youth (16-20 years)	1.12E+00	6.02E-01
				Youth (11-15 years)	1.22E+00	6.59E-01
	Low Intensity User	10% (0.25) ¹	Min (30)	Adult (\geq 21 years)	7.52E-02	3.22E-02
				Youth (16-20 years)	7.03E-02	3.01E-02
				Youth (11-15 years)	7.69E-02	3.29E-02
Electronics Cleaner	High Intensity User	95% (30)	Single Value (5)	Adult (\geq 21 years)	2.50E-01	3.41E-01
				Youth (16-20 years)	2.34E-01	3.19E-01
				Youth (11-15 years)	2.56E-01	3.49E-01
	Moderate Intensity User	50% (2)	Single Value (5)	Adult (\geq 21 years)	4.88E-02	2.27E-02
				Youth (16-20 years)	4.57E-02	2.12E-02
				Youth (11-15 years)	5.00E-02	2.32E-02
	Low Intensity User	10% (0.17) ¹	Single Value (5)	Adult (\geq 21 years)	1.33E-02	5.68E-03
				Youth (16-20 years)	1.24E-02	5.31E-03
				Youth (11-15 years)	1.36E-02	5.81E-03
Engine Cleaner	High Intensity User	95% (120)	Max (70)	Adult (\geq 21 years)	8.17E+00	4.24E+01
				Youth (16-20 years)	7.64E+00	3.97E+01
				Youth (11-15 years)	8.36E+00	4.34E+01
		50%	Mid	Adult (\geq 21 years)	4.13E+00	3.41E+00

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

	Moderate Intensity User	(15)	(45)	Youth (16-20 years)	3.86E+00	3.19E+00	
				Youth (11-15 years)	4.22E+00	3.49E+00	
	Low Intensity User	10% (5)	Min (20)	Adult (\geq 21 years)	9.38E-01	5.05E-01	
				Youth (16-20 years)	8.78E-01	4.73E-01	
Gasket Remover	High Intensity User	95% (60)	Max (80)	Youth (11-15 years)	9.60E-01	5.17E-01	
				Adult (\geq 21 years)	8.56E+00	2.23E+01	
				Youth (16-20 years)	8.01E+00	2.08E+01	
	Moderate Intensity User	50% (15)	Max (80)	Youth (11-15 years)	8.76E+00	2.28E+01	
				Adult (\geq 21 years)	6.74E+00	5.57E+00	
				Youth (16-20 years)	6.31E+00	5.21E+00	
	Low Intensity User	10% (2)	Min (60)	Youth (16-20 years)	6.90E+00	5.70E+00	
				Adult (\geq 21 years)	1.20E+00	5.57E-01	
				Youth (11-15 years)	1.12E+00	5.21E-01	
	Sealants	High Intensity User	95% (60)	Max (30)	Youth (11-15 years)	1.22E+00	5.70E-01
					Adult (\geq 21 years)	1.30E+00	3.38E+00
					Youth (16-20 years)	1.22E+00	3.16E+00
Moderate Intensity User		50% (15)	Max (30)	Youth (16-20 years)	1.33E+00	3.46E+00	
				Adult (\geq 21 years)	1.02E+00	8.45E-01	
				Youth (11-15 years)	9.57E-01	7.91E-01	
Low Intensity User		10% (2)	Min (10)	Youth (11-15 years)	1.05E+00	8.64E-01	
				Adult (\geq 21 years)	8.07E-02	3.75E-02	
				Youth (16-20 years)	7.55E-02	3.51E-02	
Weld Spatter Protectant	High Intensity User	95% (60)	Single Value (90)	Youth (11-15 years)	8.26E-02	3.84E-02	
				Adult (\geq 21 years)	4.86E+00	1.26E+01	
				Youth (16-20 years)	4.55E+00	1.18E+01	
	Moderate Intensity User	50% (5)	Single Value (90)	Youth (16-20 years)	4.97E+00	1.29E+01	
				Adult (\geq 21 years)	1.96E+00	1.05E+00	
				Youth (11-15 years)	1.83E+00	9.86E-01	
	Low Intensity User	10% (0.25) ¹	Single Value (90)	Youth (16-20 years)	2.00E+00	1.08E+00	
				Adult (\geq 21 years)	2.46E-01	1.05E-01	
				Youth (11-15 years)	2.30E-01	9.86E-02	
				Youth (16-20 years)	2.52E-01	1.08E-01	

¹Low-end durations reported by U.S. EPA (1987) that are less than 0.5 minutes (30 seconds) are modeled as being equal to 0.5 minutes due to that being the minimum timestep available within the model used.

Table_Apx G-1. Dermal Results for each Condition of Use using the Fraction Absorbed (PDER-2a) and Permeability (PDER-2b) Submodels within Consumer Exposure Model (CEM)

²Bolded numbers represent the selected CEM submodel results presented within Section 2.4.2.4 for each condition of use (either Fraction Absorbed or Permeability)

Generally, the estimated exposure concentrations for methylene chloride are highest utilizing the CEM (Permeability) model for high intensity use scenarios with youths (11-15 years) having the highest estimated exposures.

Estimated exposure concentrations for methylene chloride at moderate and low intensity uses tend to be higher, but within the same order of magnitude, for the CEM (Absorption Fraction) model as compared to the CEM (Permeability) model. The only exception is the brush cleaner scenario where the CEM (Permeability) model was higher across all user scenarios.

Selection of the models used to evaluate dermal exposure considered the sensitivity of the two models as well as the representativeness of the model estimates to the expected consumer exposure scenarios for each condition of use. The sensitivity and impacts of several parameters within the two dermal models considered are discussed below.

G.4 Sensitivity Analysis

G.4.1 Sensitivity Analysis of Overall CEM Model

The CEM developers conducted a detailed sensitivity analysis for CEM version 1.5, as described in Appendix C of the CEM User Guide ([EPA, 2017](#)).

In brief, the analysis was conducted on non-linear, continuous variables and categorical variables that were used in CEM models. A base run of different models using various product or article categories along with CEM defaults was used. Individual variables were modified, one at a time, and the resulting Chronic Average Daily Dose (CADD) and Acute Dose Rate (ADR) were then compared to the corresponding results for the base run. Two chemicals were used in the analysis: bis(2-ethylhexyl) phthalate was chosen for the SVOC Article model (emission model E6) and benzyl alcohol for other models. These chemicals were selected because bis(2-ethylhexyl) phthalate is a SVOC, better modeled by the Article model, and benzyl alcohol is a VOC, better modeled by other equations.

All model parameters were increased by 10% except those in the SVOC Article model (increased by 900% because a 10% change in model parameters resulted in very small differences). The measure of sensitivity for continuous variables was elasticity, defined as the ratio of percent change in each result to the corresponding percent change in model input. A positive elasticity means that an increase in the model parameter resulted in an increase in the model output whereas a negative elasticity had an associated decrease in the model output. For categorical variables such as receptor and room type, the percent difference in model outputs for different

category pairs was used as the measure of sensitivity. The results are summarized below for inhalation vs. dermal exposure models and for categorical vs. continuous user-defined variables.

Exposure Models

For the first five inhalation models (E1-E5) a negative elasticity was observed when increasing the use environment, building size, air zone exchange rate, and interzone ventilation rate. All of these factors decrease the chemical concentration, either by increasing the volume or by replacing the indoor air with cleaner (outdoor) air. Increasing the weight fraction or amount of product used had a positive elasticity because this change increases the amount of chemical added to the air, resulting in higher exposure. Vapor pressure and molecular weight also tended to have positive elasticities.

For most inhalation models, the saturation concentration did not have a notable effect on the ADR or the CADD. Mass of product used and weight fraction both had a positive linear relationship with dose. All negative parameters had elasticities less than 0.4, indicating that some terms (e.g., air exchange rates, building volume) mitigated the full effect of dilution. That is, even though the concentration is lowered, the effect of removal/dilution is not stronger than that of the chemical emission rate. Most models had an increase in dose with increasing duration of use. Increasing this parameter typically increases the peak concentration of the product, thus giving a higher overall exposure.

The results for the dermal model were different from the inhalation models, in that the elasticities for CADD and ADR were nearly the same. This outcome is consistent with the model structure, in that the chemical is placed on the skin so there is no time factor for a peak concentration to occur. The modeled exposure is based on the ability of a chemical to penetrate the skin layer once contact occurs. Dermal permeability had a near linear elasticity whereas $\log K_{OW}$ and molecular weight had zero elasticities.

User-defined Variables

These variables were separated into categorical vs. continuous. For categorical variables there were multiple parameters that affected other model inputs. For example, varying the room type changed the ventilation rates, volume size and the amount of time per day that a person spent in the room. Thus, each modeling result was calculated as the percent difference from the base run. For continuous variables, each modeling result was calculated as elasticity.

Among the categorical variables, both inhalation and dermal model results had a positive change when comparing an adult to a child and to a youth, with dermal having a smaller change between receptors than inhalation and the largest difference occurring between an adult and a child for both models. The time of day when the product was used and the duration of use occurred while the person was at home; thus, there was no effect on the ADR because the acute exposure period was too short to be affected by work schedule. Most rooms had a negative percent difference for inhalation, with the single exception of the bedroom where the receptor spent a large amount of time with a smaller volume than the living room. For dermal, the only room that resulted in a large percent difference was office/school, due to the fact that the person spent only $\frac{1}{2}$ hour at that location when the stay-at-home activity pattern was selected. For inhalation, changing from a far field to a near field base resulted in a higher ADR and CADD, likely because the near field has a smaller volume than that of the total room.

There are three input parameters for the near-field, far-field option for CEM product inhalation models. To determine the sensitivity of model results to these inputs, CEM first was run in base scenario with the near-field option, after which separate runs were performed whereby the near-field volume was increased by 10%, the far-field volume was increased by 10%, and the air exchange rate was increased by 10%. For inhalation, both the air exchange rate and volume had negative elasticities, but the air exchange rate had a much higher elasticity (near one) than the volume (0.11).

G.4.2 Sensitivity of Dermal Modeling

G.4.2.1 Duration of Use

The duration of use for this evaluation was assumed equal to the exposure time for both models. The basic relationship between the duration of use or exposure time to the acute dose rate is quite distinct for each of the three models. The CEM (Permeability) model maintains a strong positive correlation between duration of use and ADR, with ADR increasing by the same factor of the duration of use. The exact slopes of these lines are influenced differently by other factors, such as weight fraction, which will be discussed later. The CEM (Fraction Absorbed) model maintains a logarithmic relationship between duration of use and ADR, hitting a horizontal asymptote limit of 3.33E-01 after a certain duration (that duration varies by chemical). This limit will be discussed in the next section as it relates to the fraction absorbed term.

G.4.2.2 Fraction Absorbed

The fraction absorbed is essentially the factor that determines what mass of chemical is absorbed into the body. It is intended to be the mass absorbed from the stratum corneum as presented by Frasch ([Frasch and Bunge, 2015](#)), but the CEM (Fraction Absorbed) model calculates and utilizes this factor differently. In terms of the equations utilizing fraction absorbed, the CEM (Fraction Absorbed) model identifies this factor as FR_{abs} .

For the CEM (Fraction Absorbed) model, the fraction absorbed factor relies on χ (the ratio of evaporation rate to steady-state dermal permeation rate), the exposure time, and certain physical-chemical properties (e.g., molecular weight, vapor pressure). As the χ value increases, at least $2/3$ of the chemical in the skin will evaporate at the end of the exposure. Therefore, for highly volatile chemicals with large χ values (e.g., methylene chloride) the fraction absorbed factor will quickly reach a maximum ($1/3$) with increasing duration (represented by taking the limit at infinity of the absorption fraction equations). After a certain duration, the fraction that will evaporate, and the fraction that will be absorbed remains constant.

The lag time (calculated based on the chemical molecular weight) used in the two fraction absorbed equations influences how quickly the fraction absorbed limit of 3.33E-01 is reached. Chemicals with shorter lag times will reach the limit of FR_{abs} at shorter durations of use. For methylene chloride, the calculated lag time is about 0.47 hours with an estimated χ value of about 5735. This results in the FR_{abs} for methylene chloride reaching the limit of 3.33E-01 at an exposure time of about 64 minutes (based on a K_p of 8.66E-03). Linking this to the calculation of the ADR in the CEM (Fraction Absorbed) model, while duration of use influences the fraction absorbed term, and the fraction absorbed term influences the ADR, the influence of the fraction

absorbed on the ADR calculation peaks as the fraction absorbed approaches the 3.33E-01 limit. Therefore, for methylene chloride, while the fraction absorbed term increases quickly as exposure time increases, after about 64 minutes, the exposure time has little influence on the fraction absorbed or the ADR.

G.4.2.3 Mass Terms

Ultimately, the ADRs for both models are driven by how much product is available and absorbed into the skin, but the mass terms are calculated quite differently. To help distinguish the models, the mass terms were investigated primarily as they relate to the exposure time (assumed to be the duration of product use obtained from survey data in this evaluation).

The CEM (Permeability) model calculates the mass absorbed term within the ADR equation (equation Apx_G-1) based on the permeability coefficient, dilution factor, duration of exposure, density, surface area of skin, and weight fraction. The dilution factor is assumed to be 1 in all modeling scenarios (no dilution). The product of these terms gives the mass of the chemical of concern absorbed by the body from exposure to the modeled product(s). The CEM (Permeability) model assumes an unlimited supply of the product is present against the skin for the entire duration period and does not consider losses due to evaporation or rinsing.

The CEM (Fraction Absorbed) model calculates the mass available for absorption within the ADR equation (equation Apx_G-3) utilizing the following terms: amount retained on skin (the mathematical product of film thickness and product density), the surface area of skin, and weight fraction. The product of these terms multiplied by the absorption fraction gives the total absorbed mass. This assumes that the product or chemical is applied once to the skin's surface in a thin film and then absorbed based on the absorption fraction. What this model doesn't consider is the mass of the product or chemical that may enter the skin continuously during the use of the product or chemical.

Because neither the CEM (Permeability) model nor the CEM (Fraction Absorbed) model considers the mass of chemical in the ADR equations, both models have the potential to overestimate the dermal absorption by modeling a mass which is larger than the mass used in a scenario. Therefore, when utilizing either of the CEM models for dermal exposure estimations, a mass check is necessary outside of the CEM model to make sure the mass absorbed does not exceed the mass used in a given scenario.

Weight Fraction

Both the CEM (Permeability) model and the CEM (Fraction Absorbed) model calculate mass values considering a weight fraction multiplier. This gives the weight fraction a potential to have considerable influence over the final ADR.

The weight fraction term in both the CEM (Permeability) model and the CEM (Fraction Absorbed) model influences the mass over time component of the models. A higher weight fraction results in a higher mass term within the models. The influence of weight fraction on the relationship between duration of use and acute dose rate (ADR) is similar to that between

duration of use and the modeled mass terms for the two CEM models. As noted previously, the weight fraction influences the slope of the curves associated with the duration of use and ADR.

G.4.2.4 Permeability Coefficients

The permeability coefficient (K_p) is a term used in both dermal models considered for this evaluation. This value represents the rate of transfer of a compound across a membrane (cm/hr). The K_p value is used directly in the ADR calculation within the CEM (Permeability) model and therefore has a direct influence on the ADR estimates. The K_p value indirectly influences the ADR estimates within the CEM (Fraction Absorbed) model through the fraction absorbed term (via χ).

Experimental K_p values may be found in the literature or can be estimated utilizing various methods. Experimental K_p values can be directly entered into both CEM dermal models or can be estimated within CEM as described in the CEM Users Guide ([U.S. EPA, 2019a](#)) and associated User Guide appendices ([U.S. EPA, 2019b](#)).

The sensitivity of both models to changing K_p values on the ADR estimates shows the CEM (Permeability) model has a very strong response to changing K_p values in relation to the slope of the curve. Larger K_p values increase the slope of the curve showing the ADR estimates resulting in a much more rapid increase in ADR estimates over a shorter duration of use. The CEM (Fraction Absorbed) model is only very slightly influenced by changing K_p values.

G.4.2.5 Other Parameters

While the parameters discussed in previous sections have the potential to significantly impact ADR estimates from the three models, other parameters can still influence the model outputs or provide insight into differences between model outputs.

Product Density: Product density is a factor in both the CEM (Permeability) model and the CEM (Fraction Absorbed) models. Product density is directly utilized within the CEM (Permeability) model ADR calculation and indirectly utilized within the CEM (Fraction Absorbed) model ADR calculation (through amount retained on skin).

Both of the CEM model ADR estimates change proportionately to changes in the product density. While the general behavior and curve shapes for the ADR do not appear to change much for either of the CEM models in response to product density, the ADR estimates decrease with lower densities. Though the influence of product density does not explain or describe much difference between the CEM (Permeability) model and the CEM (Fraction Absorbed) model

Film Thickness on Skin: Film thickness is only an input to the CEM (Fraction Absorbed) model ADR calculations (as an input to the amount retained on skin term). Similar to the product density influence, the ADR estimates from the CEM (Fraction Absorbed) model change proportionately to changes in the film thickness. A larger film thickness results in a larger ADR estimate with the CEM (Fraction Absorbed) model.

G.4.2.6 Selection of Dermal Models

Two general exposure scenarios were applied to select conditions of use.

- 1) Evaporation is inhibited/prohibited or full immersion of a body part occurs during product use.
- 2) Evaporation is uninhibited and full immersion of a body part does not occur during product use.

When applying the general constructs outlined above, the CEM (Permeability) model has a component which is applicable to conditions of use where evaporation is inhibited/prohibited or full immersion of a body part occurs during use. Additionally, the CEM (Permeability) model directly considers product density (rather than solubility) within components of the ADR equation. Since most of the products utilized for these conditions of use are solvent based (rather than aqueous), utilization of the CEM (Permeability) model along with a neat permeability coefficient (K_p) is expected to provide a more representative ADR estimate for this evaluation. When applying the general constructs outlined above, the CEM (Fraction Absorbed) model has a component which is applicable to conditions of use where evaporation is uninhibited and full immersion of a body part does not occur during use. Similar to the discussion above, the products utilized for these conditions of use are solvent based (rather than aqueous) based. Since the CEM (Fraction Absorbed) model considers product density (indirectly through the amount retained on skin), utilization of the CEM (Fraction Absorbed) model is expected to provide a more representative ADR estimate for this evaluation.

Appendix H ENVIRONMENTAL HAZARDS

H.1 Aquatic Toxicity Data Extraction Table for Methylene Chloride

Table Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
<i>Fish</i>								
Rainbow trout (<i>Oncorhynchus mykiss</i>)	Fresh	23-day	LC ₅₀ = 13.51	0, 0.008, 0.042, 0.41, 5.55, 23.1, 36.5	Flow-through, Measured	Mortality	(Black et al., 1982)	High
Rainbow trout (<i>Oncorhynchus mykiss</i>)	Fresh	27-day	LC ₅₀ = 13.16	0, 0.008, 0.042, 0.41, 5.55, 23.1, 36.5	Flow-through, Measured	Mortality	(Black et al., 1982)	High
Rainbow trout (<i>Oncorhynchus mykiss</i>)	Fresh	27-day	NOEC = 0.41 LOEC = 5.55	0, 0.008, 0.042, 0.41, 5.55, 23.1, 36.5	Flow-through, Measured	Teratic larvae	(Black et al., 1982)	High
Bluegill (<i>Lepomis macrochirus</i>)	Fresh	24-hr	LC ₅₀ = 230	Not reported	Static, Nominal	Mortality	(Buccafuso et al., 1981)	Unacceptable
Bluegill (<i>Lepomis macrochirus</i>)	Fresh	96-hr	LC ₅₀ = 220	Not reported	Static, Nominal	Mortality	(Buccafuso et al., 1981)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₉₀ = 722.1	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₅₀ = 193	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₁₀ = 51.2	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	LC ₉₀ = 802	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	LC ₅₀ = 232.4	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	LC ₁₀ = 67.3	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	LC ₉₀ = 746.3	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	LC ₅₀ = 265	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	LC ₁₀ = 94 mg AI/L	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	LC ₉₀ = 589	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	LC ₅₀ = 268	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	LC ₁₀ = 122	Not reported	Flow-through, Measured	Mortality	(Alexander et al., 1978)	Medium

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₅₀ = 310	Not reported	Static, Nominal	Mortality	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	EC ₉₀ = 220.1	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	EC ₅₀ = 112.8	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	EC ₁₀ = 68.5 L	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	EC ₉₀ = 147.6	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	EC ₅₀ = 99	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	48-hr	EC ₁₀ = 66.3	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	EC ₉₀ = 147.6	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	EC ₅₀ = 99	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	72-hr	EC ₁₀ = 66.3	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	EC ₉₀ = 147.6	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	EC ₅₀ = 99	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	EC ₁₀ = 66.3	Not reported	Flow-through, Measured	Immobilization	(Alexander et al., 1978)	Medium
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	5-day	LC ₅₀ >34	0, 0.003, 0.11, 0.80, 6.77, 21.3, 34.3	Flow-through, Nominal	Mortality	(Black et al., 1982)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	9-day	LC ₅₀ = ~34	0, 0.003, 0.11, 0.80, 6.77, 21.3, 34.3	Flow-through, Nominal	Mortality	(Black et al., 1982)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	24-hr	EC ₅₀ = 49,400	0, 21, 42, 63, 84, 105	In vitro, Nominal	Inhibition of total protein content	(Dierickx, 1993)	Unacceptable
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₅₀ = 502	79, 135, 207, 357, 527, 855	Flow-through, Measured	Mortality	(Dill et al., 1987)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	192-hr	LC ₅₀ = 471	79, 135, 207, 357, 527, 855	Flow-through, Measured	Mortality	(Dill et al., 1987)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	32-day	MATC = 108 NOEC = 82.5 LOEC = 142	29, 55, 82, 142, 209, 321	Flow-through, Measured	Growth: body weight	(Dill et al., 1987)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	32-day	NOEC = 142 LOEC = 209	29, 55, 82, 142, 209, 321	Flow-through, Measured	Mortality	(Dill et al., 1987)	High
Rainbow trout (<i>Oncorhynchus mykiss</i>) cited as <i>Salmo gairdneri</i>	Fresh	96-hr	LC ₅₀ = 108	29, 39, 78, 111, 146, 240	Flow-through, Measured	Mortality	(E I Dupont Denemours & Co Inc, 1987b)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	LC ₅₀ = 330	6.42, 78.4, 169, 212, 288, 485	Flow-through, Measured	Mortality	(Geiger et al., 1986)	High
Fathead minnow (<i>Pimephales promelas</i>)	Fresh	96-hr	EC ₅₀ = 330	6.42, 78.4, 169, 212, 288, 485	Flow-through, Measured	Hypo- and hyperactivity	(Geiger et al., 1986)	High

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Sheepshead minnow (<i>Cyprinodon variegatus</i>)	Salt	24-hr	LC ₅₀ = 370	Not reported	Static, Nominal	Mortality	(Heitmuller et al., 1981)	Unacceptable
Sheepshead minnow (<i>Cyprinodon variegatus</i>)	Salt	48-hr	LC ₅₀ = 360	Not reported	Static, Nominal	Mortality	(Heitmuller et al., 1981)	Unacceptable
Sheepshead minnow (<i>Cyprinodon variegatus</i>)	Salt	72hr	LC ₅₀ = 360	Not reported	Static, Nominal	Mortality	(Heitmuller et al., 1981)	Unacceptable
Sheepshead minnow (<i>Cyprinodon variegatus</i>)	Salt	96-hr	LC ₅₀ = 330	Not reported	Static, Nominal	Mortality	(Heitmuller et al., 1981)	Unacceptable
Sheepshead minnow (<i>Cyprinodon variegatus</i>)	Salt	96-hr	NOEC = 130	Not reported	Static, Nominal	Mortality	(Heitmuller et al., 1981)	Unacceptable
<i>Aquatic Invertebrates</i>								
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	EC ₅₀ = 135.8077071	Not reported	Static, Nominal	Immobilization	(Abernethy et al., 1986)	Medium
Water flea (<i>Daphnia magna</i>)	Fresh	24-hr	EC ₀ = 1,447	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	24-hr	EC ₅₀ = 1,959	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	24-hr	EC ₁₀₀ = 2,500	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	EC ₀ = 1,005	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	EC ₅₀ = 1,682	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	EC ₁₀₀ = 2,500	Not reported	Static, Nominal	Immobilization	(Kuhn et al., 1989)	Low
Water flea (<i>Daphnia magna</i>)	Fresh	24-hr	LC ₅₀ = 310	Not reported	Static, Nominal	Mortality	(Leblanc, 1980)	High
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	LC ₅₀ = 220	Not reported	Static, Nominal	Mortality	(Leblanc, 1980)	High

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	NOEC = 68	Not reported	Static, Nominal	Mortality	(Leblanc, 1980)	High
Water flea (<i>Daphnia magna</i>)	Fresh	12-15-day	BCF = < 1	0.11890606-0.7559028	Static, Measured	Residue, whole body	(Thiébaud et al., 1994)	Unacceptable
Water flea (<i>Daphnia magna</i>)	Fresh	48-hr	EC ₅₀ = 177	23, 34, 60, 106, 180, 253	Static, Measured	Immobilization	(E I Dupont Denemours & Co Inc, 1987a)	High
Bladder snail (<i>Physa fontinalis</i>)	Fresh	12-15-day	BCF = 5 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, whole body	(Thiébaud et al., 1994)	Unacceptable
Bladder snail (<i>Physa fontinalis</i>)	Fresh	12-15-day	BCF = 7 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, whole body	(Thiébaud et al., 1994)	Unacceptable
Bladder snail (<i>Physa fontinalis</i>)	Fresh	12-15-day	BCF = 8 (Expt. 3)	0.11890606-0.7559028	Static, Measured	Residue, whole body	(Thiébaud et al., 1994)	Unacceptable
Bladder snail (<i>Physa fontinalis</i>)	Fresh	12-15-day	BCF = < 1 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, egg	(Thiébaud et al., 1994)	Unacceptable
Bladder snail (<i>Physa fontinalis</i>)	Fresh	12-15-day	BCF = < 1 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, egg	(Thiébaud et al., 1994)	Unacceptable
Brine shrimp (<i>Artemia salina</i>)	Salt	24-hr	LC ₅₀ = 122.303376	Not reported	Static, Nominal	Mortality, 24-hr age class	(Sanchez-Fortun et al., 1997)	Unacceptable
Brine shrimp (<i>Artemia salina</i>)	Salt	24-hr	LC ₅₀ = 96.823506	Not reported	Static, Nominal	Mortality, 48-hr age class	(Sanchez-Fortun et al., 1997)	Unacceptable
Brine shrimp (<i>Artemia salina</i>)	Salt	24-hr	LC ₅₀ = 87.480887	Not reported	Static, Nominal	Mortality, 72-hr age class	(Sanchez-Fortun et al., 1997)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₅₀ = 1170 (Expt. 1)	Not reported	Static, Nominal	Mortality	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₅₀ = 758 (Expt. 2)	Not reported	Static, Not reported	Mortality	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₅₀ = 891 (Expt. 3)	Not reported	Static, Nominal	Mortality	(Rayburn and Fisher, 1999)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	12-day	LC ₅₀ = 319 (Expt. 1)	Not reported	Static, Nominal	Mortality	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	12-day	LC ₅₀ = 452 (Expt. 2)	Not reported	Static, Nominal	Mortality	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	12-day	LC ₅₀ = 479 (Expt. 3)	Not reported	Static, Nominal	Mortality	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	7-day	NOAEL = 930 (Expt. 1)	0, 130, 400, 670, 930	Static, Nominal	Growth: Length	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	7-day	NOAEL = 930 (Expt. 2)	0, 130, 400, 670, 930	Static, Nominal	Growth: Length	(Rayburn and Fisher, 1999)	Unacceptable
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₁₀₀ = 0.5% v/v (if 100% purity = 6,700)	0, 0.01, 0.05, 0.1, 0.5, 1% v/v (if 100% purity = 0, 130, 670, 1,300, 6,700, 13,000)	Static, Nominal, Embryonic stage 3	Mortality	(Wilson, 1998)	High
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₁₀₀ = 1% v/v (if 100% purity = 13,000)	0, 0.01, 0.05, 0.1, 0.5, 1% v/v (if 100% purity = 0, 130, 670, 1,300, 6,700, 13,000)	Static, Nominal, Embryonic stage 4	Mortality	(Wilson, 1998)	High
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	LC ₁₀₀ = 0.5% v/v (if 100% purity = 6,700)	0, 0.01, 0.05, 0.1, 0.5, 1% v/v (if 100% purity = 0, 130, 670, 1,300, 6,700, 13,000)	Static, Nominal, Embryonic stage 6	Mortality	(Wilson, 1998)	High
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	NOEC = 0.05% v/v (if 100% purity = 670) LOEC = 0.1% v/v (if 100% purity = 1,300)	0, 0.01, 0.05, 0.1, 0.5, 1% v/v (if 100% purity = 0, 130, 670, 1,300, 6,700, 13,000)	Static, Nominal	Developmental delay	(Wilson, 1998)	High
Daggerblade grass shrimp (<i>Palaemonetes pugio</i>)	Salt	4-day	NOEC = 670 LOEC = 1,300	0, 0.01, 0.05, 0.1, 0.5, 1% v/v (if 100% purity = 0, 130, 670, 1,300, 6,700, 13,000)	Static, Nominal	Mortality	(Wilson, 1998)	High

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
<i>Algae</i>								
Green algae (<i>Chlamydomonas reinhardtii</i>)	Fresh	72-hr	EC ₁₀ = 115	Not reported	Static, Measured	Biomass	(Brack and Rottler, 1994)	High
Green algae (<i>Chlamydomonas reinhardtii</i>)	Fresh	72-hr	EC ₅₀ = 242	Not reported	Static, Measured	Biomass	(Brack and Rottler, 1994)	High
Green algae (<i>Chlorella vulgaris</i>)	Fresh	10-day	NOAEL = 2	0, 0.002, 0.02, 0.2, 2	Static, Nominal	Growth (chlorophyll A concentration)	(Ando et al., 2003)	Medium
Green algae (<i>Pseudokirchneriella subcapitata</i>)	Fresh	10-day	NOAEL = 2	0, 0.002, 0.02, 0.2, 2	Static, Nominal	Growth (chlorophyll A concentration)	(Ando et al., 2003)	Medium
Green algae (<i>Volvox steinii</i>)	Fresh	10-day	LOAEL = 0.002	0, 0.002, 0.02, 0.2,	Static, Nominal	Growth (chlorophyll A concentration)	(Ando et al., 2003)	Medium
Green algae (<i>Pseudokirchneriella subcapitata</i>)	Fresh	48-hr	EC ₅₀ = 33.09	Not reported	Static, Nominal	Cell density	(Tsai and Chen, 2007)	High
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	EC ₅₀ = 0.98	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Growth	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	LOAEL = 221	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Catalase activity	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	LOAEL = 221	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Malondialdehyde content	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	NOAEL = 221 LOAEL = 299	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Superoxide dismutase (SOD) enzyme activity	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	NOAEL = 221 LOAEL = 299	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Cell density	(Wu et al., 2014)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	NOAEL = 299 LOAEL = 403	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Total protein content	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	96-hr	LOAEL = 221	0, 221, 299, 403, 550, 735, 992	Static, Nominal	Chlorophyll A concentration	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	6-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of photosystem I reaction center protein subunit B gene	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	12-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of photosystem I reaction center protein subunit B gene	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	48-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of photosystem I reaction center protein subunit B gene	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	64-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of photosystem I reaction center protein subunit B gene	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	64-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of gene for photosystem II membrane protein component	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	48-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of gene for photosystem II membrane protein component	(Wu et al., 2014)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Green algae (<i>Chlorella vulgaris</i>)	Fresh	24-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of gene for photosystem II membrane protein component	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	12-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of gene for photosystem II membrane protein component	(Wu et al., 2014)	Unacceptable
Green algae (<i>Chlorella vulgaris</i>)	Fresh	6-hr	LOAEL = 0.98	0, 0.98	Static, Nominal	Transcription of gene for photosystem II membrane protein component	(Wu et al., 2014)	Unacceptable
<i>Aquatic Plants</i>								
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 39 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, colonies	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 4 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, colonies	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 54 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, young fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = <1 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, young fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 15 (Expt. 3)	0.11890606-0.7559028	Static, Measured	Residue, young fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 13 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, old fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 4 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, old fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 7 (Expt. 3)	0.11890606-0.7559028	Static, Measured	Residue, old fronds	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 112 (Expt. 1)	0.11890606-0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = <1 (Expt. 2)	0.11890606-0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/ Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Duckweed (<i>Lemna minor</i>)	Fresh	12-15-day	BCF = 28 (Expt. 3)	0.11890606- 0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 74 (Expt. 1)	0.11890606- 0.7559028	Static, Measured	Residue, leaves	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 9 (Expt. 2)	0.11890606- 0.7559028	Static, Measured	Residue, leaves	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 5 (Expt. 3)	0.11890606- 0.7559028	Static, Measured	Residue, leaves	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 34 (Expt. 1)	0.11890606- 0.7559028	Static, Measured	Residue, stems	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 5 (Expt. 2)	0.11890606- 0.7559028	Static, Measured	Residue, stems	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 10 (Expt. 3)	0.11890606- 0.7559028	Static, Measured	Residue, stems	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 10 (Expt. 1)	0.11890606- 0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 1 (Expt. 2)	0.11890606- 0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable
Pondweed (<i>Groenlandia densa</i>)	Fresh	12-15-day	BCF = 15 (Expt. 3)	0.11890606- 0.7559028	Static, Measured	Residue, roots	(Thiébaud et al., 1994)	Unacceptable
Waterweed (<i>Elodea canadensis</i>)	Fresh	12-15-day	BCF = 5	0.11890606- 0.7559028	Static, Measured	Residue, leaves	(Thiébaud et al., 1994)	Unacceptable
Waterweed (<i>Elodea canadensis</i>)	Fresh	12-15-day	BCF = 3	0.11890606- 0.7559028	Static, Measured	Residue, stems	(Thiébaud et al., 1994)	Unacceptable
Moss (<i>Fontinalis antipyretica</i>)	Fresh	12-15-day	BCF = 577 (Expt. 1)	0.11890606- 0.7559028	Static, Measured	Residue, whole plant	(Thiébaud et al., 1994)	Unacceptable
Moss (<i>Fontinalis antipyretica</i>)	Fresh	12-15-day	BCF = 9 (Expt. 2)	0.11890606- 0.7559028	Static, Measured	Residue, whole plant	(Thiébaud et al., 1994)	Unacceptable
Moss (<i>Fontinalis antipyretica</i>)	Fresh	12-15-day	BCF = 41 (Expt. 3)	0.11890606- 0.7559028	Static, Measured	Residue, whole plant	(Thiébaud et al., 1994)	Unacceptable
<i>Amphibians</i>								
Bullfrog (<i>Rana catesbeiana</i>)	Fresh	4-day	LC ₅₀ = 30.61	0, 0.017, 0.071, 0.66, 6.73, 46.8	Flow-through, Measured	Teratogenesis and Mortality	(Birge et al., 1980)	High

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Bullfrog (<i>Rana catesbeiana</i>)	Fresh	8-day	LC ₅₀ = 17.78	0, 0.017, 0.071, 0.66, 6.73, 46.8	Flow-through, Measured	Teratogenesis and Mortality	(Birge et al., 1980)	High
Fowler's toad (<i>Anaxyrus woodhousei</i> ssp.) cited as <i>Bufo fowleri</i>	Fresh	3-day	LC ₅₀ >32	0, 0.022, 0.13, 1.42, 10.1, 32.1	Flow-through, Measured	Teratogenesis and Mortality	(Birge et al., 1980)	High
Fowler's toad (<i>Anaxyrus woodhousei</i> ssp.) cited as <i>Bufo fowleri</i>	Fresh	7-day	LC ₅₀ >32	0, 0.022, 0.13, 1.42, 10.1, 32.1	Flow-through, Measured	Teratogenesis and Mortality	(Birge et al., 1980)	High
Pickerel frog (<i>Lithobates palustris</i>) cited as <i>Rana palustris</i>	Fresh	4-day	LC ₅₀ >32	0, 0.022, 0.13, 1.42, 10.1, 32.1	Flow-through, Measured	Teratogenesis and Mortality	(Birge et al., 1980)	High
Pickerel frog (<i>Lithobates palustris</i>) cited as <i>Rana palustris</i>	Fresh	8-day	LC ₅₀ >32	0, 0.022, 0.13, 1.42, 10.1, 32.1	Flow-through, Measured	Mortality	(Birge et al., 1980)	High
Bullfrog (<i>Rana catesbeiana</i>)	Fresh	8-day	LC ₁₀ = 0.981	0, 0.017, 0.071, 0.66, 6.73, 46.8	Flow-through, Measured	Mortality	(Birge et al., 1980)	High
Bullfrog (<i>Rana catesbeiana</i>)	Fresh	8-day	LC ₀₁ = 0.0925	0, 0.017, 0.071, 0.66, 6.73, 46.8	Flow-through, Measured	Mortality	(Birge et al., 1980)	High
Bullfrog (<i>Rana catesbeiana</i>)	Fresh	8-day	LC ₀ = 0.017	0, 0.017, 0.071, 0.66, 6.73, 46.8	Flow-through, Measured	Mortality	(Birge et al., 1980)	High
European Common Frog (<i>Rana temporaria</i>)	Fresh	5-day	LC ₅₀ = 23.03	0, 0.004, 0.18, 0.65, 8.05, 18.9, 30.8	Flow-through, Measured	Mortality	(Birge et al., 1980)	High
European Common Frog (<i>Rana temporaria</i>)	Fresh	9-day	LC ₅₀ = 16.93	0, 0.004, 0.18, 0.65, 8.05, 18.9, 30.8	Flow-through, Measured	Mortality	(Black et al., 1982)	High
European Common Frog (<i>Rana temporaria</i>)	Fresh	9-day	LC ₁₀ = 0.8224	0, 0.004, 0.18, 0.65, 8.05, 18.9, 30.8	Flow-through, Measured	Mortality	(Black et al., 1982)	High
European Common Frog (<i>Rana temporaria</i>)	Fresh	9-day	LC ₀₁ = 0.0699	0, 0.004, 0.18, 0.65, 8.05, 18.9, 30.8	Flow-through, Measured	Mortality	(Black et al., 1982)	High

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	End-point (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Northwestern salamander (<i>Ambystoma gracile</i>)	Fresh	5.5-day	LC ₅₀ = 23.86	0, 0.004, 0.18, 0.65, 7.83, 18.6, 29.4	Flow-through, Measured	Mortality	(Black et al., 1982)	High
Northwestern salamander (<i>Ambystoma gracile</i>)	Fresh	9.5-day	LC ₅₀ = 17.82	0, 0.004, 0.18, 0.65, 7.83, 18.6, 29.4	Flow-through, Measured	Mortality	(Black et al., 1982)	High
African clawed frog (<i>Xenopus laevis</i>)	Fresh	2-day	LC ₅₀ >29	0, 0.003, 0.18, 0.65, 7.61, 18.6, 29.3	Flow-through, Measured	Mortality	(Black et al., 1982)	High
African clawed frog (<i>Xenopus laevis</i>)	Fresh	6-day	LC ₅₀ >29	0, 0.003, 0.18, 0.65, 7.61, 18.6, 29.3 mg/L	Flow-through, Nominal	Mortality	(Black et al., 1982)	High
Leopard frog (<i>Lithobates pipiens</i>)	Fresh	5-day	LC ₅₀ >48	0, 0.010, 0.077, 1.17, 28.7, 47.8 mg/L	Flow-through, Nominal	Mortality	(Black et al., 1982)	High
Leopard frog (<i>Lithobates pipiens</i>)	Fresh	9-day	LC ₅₀ >48	0, 0.010, 0.077, 1.17, 28.7, 47.8 mg/L	Flow-through, Nominal	Mortality	(Black et al., 1982)	High
European Common Frog (<i>Rana temporaria</i>)	Fresh	48-hr	NOAEL = 0.1 mL/L	0, 0.001, 0.1 mL/L	Static, Nominal, Eggs without jelly coat	Mortality	(Marquis et al., 2006)	Unacceptable
European Common Frog (<i>Rana temporaria</i>)	Fresh	48-hr	LOAEL = 0.1 mL/L	0, 0.1 mL/L	Static, Nominal, Eggs with jelly coat	Mortality	(Marquis et al., 2006)	Unacceptable
European Common Frog (<i>Rana temporaria</i>)	Fresh	48-hr	NOAEL = 0.1 mL/L	0, 0.1 mL/L	Static, Nominal, Tadpoles	Mortality	(Marquis et al., 2006)	Unacceptable
<i>Fungi</i>								
Fungus (<i>Aspergillus versicolor</i>)	Vapor exposure	32-hr	LT ₅₀ = 11.5 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
Fungus (<i>Aspergillus cejpai</i> , formerly <i>Dichotomomyces cejpai</i>)	Vapor exposure	32-hr	LT ₅₀ = ~30 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
Fungus (<i>Coniothrium sp.</i>)	Vapor exposure	32-hr	LT ₅₀ = ~5 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable

Table_Apx H-1. Aquatic Toxicity Data Extraction Table for Methylene Chloride

Test Species	Fresh/Saltwater	Duration	Endpoint (mg/L)	Concentration(s) (mg/L)	Test Analysis	Effect(s)	References	Data Quality Evaluation
Fungus (<i>Acremonium tubakii</i>)	Vapor exposure	32-hr	LT ₅₀ = ~4 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
Fungus (<i>Phoma putaminum</i>)	Vapor exposure	32-hr	LT ₅₀ = 2.8 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
Fungus (Unidentified <i>Basidiomycetes</i>)	Vapor exposure	32-hr	LT ₅₀ = 1.9 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
Fungus (Unidentified <i>Basidiomycetes</i>)	Vapor exposure	32-hr	LT ₅₀ = 1.4 hours	0, 2,400 mg AI/L air	Static, Not reported	Mortality	(Steiman et al., 1995)	Unacceptable
<i>Insects</i>								
Yellow fever mosquito (<i>Aedes aegypti</i>)	Fresh	4-hr	LC ₅₀ = 6,920	Not reported	Static, Nominal	Mortality	(Kramer et al., 1983)	Unacceptable
<i>Terrestrial Invertebrates</i>								
Beer nematode (<i>Panagrellus redivivus</i>)	Culture medium	96-hr	LOAEL = 0.00085	0, 0.00085, 0.0085, 0.085, 0.85, 8.5, 85	Static, Nominal	Growth: slowed, retarded, delayed, or non-developmental delay	(Samoiloff et al., 1980)	Unacceptable

H.2 Risk Quotients for All Facilities Modeled in E-FAST

Table_Apx H-2. Risk Quotients for All Facilities Modeled in E-FAST

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
OES: Manufacturing									
COVESTRO LLC BAYTOWN, TX FRS: 110000463098	Surface Water	Active Releaser: NPDES TX0002798	Surface water	350	0.43	1.63E-04	4.78E-03	2.85E-03	2.39E-04
				20	7.510	2.86E-03	8.34E-02	4.97E-02	4.17E-03
EMERALD PERFORMANCE MATERIALS LLC HENRY, IL NPDES: IL0001392	Surface Water	Active Releaser: NPDES IL0001392	Still water	350	0.480	1.83E-04	5.33E-03	3.18E-03	2.67E-04
				20	8.32	3.16E-03	9.24E-02	5.51E-02	4.62E-03

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
FISHER SCIENTIFIC CO LLC FAIR LAWN, NJ NPDES: NJ0110281	POTW	Receiving Facility: PASSAIC VALLEY SEWER COMM; NPDES NJ0021016	Still water	350	0.000442	1.68E-07	4.91E-06	2.93E-06	2.46E-07
FISHER SCIENTIFIC CO LLC BRIDGEWATER, NJ NPDES: NJ0119245	POTW	Receiving Facility: SOMERSET RARITIAN VALLEY SEWERAGE; NPDES NJ0024864	Surface water	350	0.07	2.65E-05	7.73E-04	4.61E-04	3.87E-05
OLIN BLUE CUBE FREEPORT TX FREEPORT, TX TRI: 7754WBLCBP231NB	Non-POTW WWT	Receiving Facility: DOW CHEMICAL-FREEPORT, TX; NPDES TX0006483	Surface water	350	0.029	1.11E-05	3.26E-04	1.94E-04	1.63E-05
REGIS TECHNOLOGIES INC MORTON GROVE, IL FRS: 110000429661	POTW	Receiving Facility: MWRDGC TERRENCE J O'BRIEN WTR RECLAMATION PLANT; NPDES IL0028088	Still water	350	0.00270	1.03E-06	3.00E-05	1.79E-05	1.50E-06
SIGMA-ALDRICH MANUFACTURING LLC SAINT LOUIS, MO FRS: 110000743125	POTW	Receiving Facility: BISSEL POINT WWTP ST LOUIS MSD; NPDES MO0025178	Surface water	350	0.0000366	1.39E-08	4.07E-07	2.42E-07	2.03E-08

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
VANDERBILT CHEMICALS LLC-MURRAY DIV MURRAY, KY NPDES: KY0003433	Non-POTW WWT	Receiving Facility: VALICOR ENVIRONMENTAL SERVICES; Organic Chemicals Manufacturing	Surface water	350	0.110	4.18E-05	1.22E-03	7.28E-04	6.11E-05
E I DUPONT DE NEMOURS - CHAMBERS WORKS DEEPWATER, NJ NPDES: NJ0005100	Surface Water	Active Releaser: NPDES NJ0005100	Surface water	350	0.0322	1.22E-05	3.58E-04	2.13E-04	1.79E-05
				20	0.56	2.13E-04	6.22E-03	3.71E-03	3.11E-04
BAYER MATERIALSCIENCE BAYTOWN, TX NPDES: TX0002798	Surface Water	Active Releaser: NPDES TX0002798	Surface water	350	3.15	1.20E-03	3.50E-02	2.09E-02	1.75E-03
				20	55.08	2.09E-02	6.12E-01	3.65E-01	3.06E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
INSTITUTE PLANT INSTITUTE, WV NPDES: WV0000086	Surface Water	Active Releaser: NPDES WV0000086	Surface water	350	0.00282	1.07E-06	3.13E-05	1.87E-05	1.57E-06
				20	0.0494	1.88E-05	5.49E-04	3.27E-04	2.74E-05
MPM SILICONES LLC FRIENDLY, WV NPDES: WV0000094	Surface Water	Active Releaser: NPDES WV0000094	Surface water	350	0.000555	2.11E-07	6.17E-06	3.68E-06	3.08E-07
				20	0.00972	3.70E-06	1.08E-04	6.44E-05	5.40E-06
BASF CORPORATION WEST MEMPHIS, AR NPDES: AR0037770	Surface Water	Active Releaser: NPDES AR0037770	Surface water	350	0.0000134	5.10E-09	1.49E-07	8.87E-08	7.44E-09
				20	0.000235	8.94E-08	2.61E-06	1.56E-06	1.31E-07

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
ARKEMA INC PIFFARD, NY NPDES: NY0068225	Surface Water	Active Releaser: NPDES NY0068225	Surface water	350	0.00347	1.32E-06	3.86E-05	2.30E-05	1.93E-06
				20	0.0608	2.31E-05	6.76E-04	4.03E-04	3.38E-05
EAGLE US 2 LLC - LAKE CHARLES COMPLEX LAKE CHARLES, LA NPDES: LA0000761	Surface Water	Active Releaser: NPDES LA0000761	Surface water	350	0.00081	3.06E-07	8.96E-06	5.34E-06	4.48E-07
				20	0.0141	5.36E-06	1.57E-04	9.34E-05	7.83E-06
BAYER MATERIALSCIENCE NEW	Surface Water	Active Releaser: NPDES WV0005169	Surface water	350	0.000084	3.21E-08	9.38E-07	5.59E-07	4.69E-08

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
MARTINSVILLE, WV NPDES: WV0005169									
				20	0.00148	5.63E-07	1.64E-05	9.80E-06	8.22E-07
ICL-IP AMERICA INC GALLIPOLIS FERRY, WV NPDES: WV0002496	Surface Water	Active Releaser: NPDES WV0002496	Surface water	350	0.0000262	9.96E-09	2.91E-07	1.74E-07	1.46E-08
				20	0.000458	1.74E-07	5.09E-06	3.03E-06	2.54E-07
KEESHAN AND BOST CHEMICAL CO., INC. MANVEL, TX NPDES: TX0072168	Surface Water	Active Releaser: NPDES TX0072168	Still water	350	4.73	1.80E-03	5.26E-02	3.13E-02	2.63E-03
				20	82.80	3.15E-02	9.20E-01	5.48E-01	4.60E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
INDORAMA VENTURES OLEFINS, LLC SULPHUR, LA NPDES: LA0069850	Surface Water	Active Releaser (Surrogate): NPDES LA0000761	Surface water	350	0.0000301	1.14E-08	3.34E-07	1.99E-07	1.67E-08
				20	0.000527	2.00E-07	5.86E-06	3.49E-06	2.93E-07
CHEMTURA NORTH AND SOUTH PLANTS MORGANTOWN, WV NPDES: WV0004740	Surface Water	Active Releaser: NPDES WV0004740	Surface water	350	0.0000344	1.31E-08	3.82E-07	2.28E-07	1.91E-08
				20	0.0006	2.28E-07	6.67E-06	3.97E-06	3.33E-07
OES: Import and Repackaging									
CHEMISPHERE CORP SAINT LOUIS, MO FRS: 110000852943	POTW	Receiving Facility: BISSEL POINT WWTP ST LOUIS MSD; NPDES MO0025178	Surface water	250	0.0000512	1.95E-08	5.69E-07	3.39E-07	2.84E-08
				250	34.38	1.31E-02	3.82E-01	2.28E-01	1.91E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
HUBBARD-HALL INC WATERBURY, CT FRS: 110000317194	Non-POTW WWT	Receiving Facility: RECYCLE INC.; POTW (Ind.)	Surface water						
WEBB CHEMICAL SERVICE CORP MUSKEGON HEIGHTS, MI NPDES: MI0049719	POTW	Receiving Facility: MUSKEGON CO WWMS METRO WWTP; NPDES MI0027391	Surface water	250	0.1000	3.80E-05	1.11E-03	6.62E-04	5.56E-05
RESEARCH SOLUTIONS GROUP INC PELHAM, AL NPDES: AL0074276	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0442	1.68E-05	4.91E-04	2.93E-04	2.46E-05
				20	0.55	2.09E-04	6.11E-03	3.64E-03	3.06E-04
EMD MILLIPORE CORP CINCINNATI, OH NPDES: OH0047759	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0144	5.48E-06	1.60E-04	9.54E-05	8.00E-06
				20	0.18	6.84E-05	2.00E-03	1.19E-03	1.00E-04

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
OES: Processing as a Reactant									
AMVAC CHEMICAL CO AXIS, AL FRS: 110015634866	Non-POTW WWT	Receiving Facility: DUPONT AGRICULTURAL PRODUCTS; NPDES AL0001597	Surface water	350	0.0151	5.74E-06	1.68E-04	1.00E-04	8.39E-06
THE DOW CHEMICAL CO MIDLAND, MI NPDES: MI0000868	Surface Water	Active Releaser: NPDES MI0000868	Surface water	350	0.11	4.18E-05	1.22E-03	7.28E-04	6.11E-05
				20	1.98	7.53E-04	2.20E-02	1.31E-02	1.10E-03
FMC CORPORATION MIDDLEPORT, NY NPDES: NY0000345	Surface Water	Active Releaser: NPDES NY0000345	Surface water	350	0.26	9.89E-05	2.89E-03	1.72E-03	1.44E-04
				20	4.55	1.73E-03	5.06E-02	3.01E-02	2.53E-03

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
OES: Processing – Formulation									
ARKEMA INC CALVERT CITY, KY NPDES: KY0003603	Surface Water	Active Releaser: NPDES KY0003603	Surface water	300	0.00434	1.65E-06	4.82E-05	2.87E-05	2.41E-06
				20	0.0668	2.54E-05	7.42E-04	4.42E-04	3.71E-05
MCGEAN-ROHCO INC LIVONIA, MI FRS: 110000405801	POTW	Receiving Facility: DETROIT WWTP- CHLORINATION/DECHLORINATION FACILITY; NPDES MI0022802	Surface water	300	0.00220	8.37E-07	2.44E-05	1.46E-05	1.22E-06
WM BARR & CO INC MEMPHIS, TN FRS: 110000374265	POTW	Receiving Facility: MEMPHIS CITY MAXSON WASTEWATER TREATMENT; NPDES TN0020729	Surface water	300	0.00000277	1.05E-09	3.08E-08	1.83E-08	1.54E-09
BUCKMAN LABORATORIES INC MEMPHIS, TN NPDES: TN0040606	POTW	Receiving Facility: MC STILES TREATMENT PLANT; NPDES TN0020711	Surface water	300	0.00156	5.93E-07	1.73E-05	1.03E-05	8.67E-07
	POTW			300	1659.44	6.31E-01	1.84E+01	1.10E+01	9.22E-01

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
EUROFINS MWG OPERON LLC LOUISVILLE, KY TRI: 4029WRFNSM1271P		Receiving Facility: VEOLIA ENVIRONMENTAL SERVICES TECH SOLUTIONS LLC; Inorganic Chemicals Manuf.	Surface water						
SOLVAY - HOUSTON PLANT HOUSTON, TX NPDES: TX0007072	Surface Water	Active Releaser: NPDES TX0007072	Surface water	300	7.15	2.72E-03	7.94E-02	4.74E-02	3.97E-03
				20	107.41	4.08E-02	1.19E+00	7.11E-01	5.97E-02
HONEYWELL INTERNATIONAL INC - GEISMAR COMPLEX GEISMAR, LA NPDES: LA0006181	Surface Water	Active Releaser: NPDES LA0006181	Surface water	300	0.0000603	2.29E-08	6.70E-07	3.99E-07	3.35E-08
				20	0.000890	3.38E-07	9.89E-06	5.89E-06	4.94E-07
STEPAN CO MILLSDALE ROAD ELWOOD, IL NPDES: IL0002453	Surface Water	Active Releaser: NPDES IL0002453	Surface water	300	0.00324	1.23E-06	3.60E-05	2.15E-05	1.80E-06

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				20	0.0503	1.91E-05	5.59E-04	3.33E-04	2.79E-05
ELEMENTIS SPECIALTIES, INC. CHARLESTON, WV NPDES: WV0051560	Surface Water	Active Releaser: NPDES WV0051560	Surface water	300	0.000474	1.80E-07	5.27E-06	3.14E-06	2.63E-07
				20	0.00709	2.70E-06	7.88E-05	4.70E-05	3.94E-06
OES: Polyurethane Foam									
PREGIS INNOVATIVE PACKAGING INC WURLAND, KY NPDES: KY0094005	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	1.13	4.30E-04	1.26E-02	7.48E-03	6.28E-04
				20	14.09	5.36E-03	1.57E-01	9.33E-02	7.83E-03

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OES: Plastics Manufacturing									
SABIC INNOVATIVE PLASTICS US LLC BURKVILLE, AL NPDES: ALR16ECGK	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	4.08	1.55E-03	4.53E-02	2.70E-02	2.27E-03
				20	51.12	1.94E-02	5.68E-01	3.39E-01	2.84E-02
SABIC INNOVATIVE PLASTICS MT. VERNON, LLC MOUNT VERNON, IN NPDES: IN0002101	Surface Water	Active Releaser: NPDES IN0002101	Surface water	250	0.00491	1.87E-06	5.46E-05	3.25E-05	2.73E-06
				20	0.0624	2.37E-05	6.93E-04	4.13E-04	3.47E-05
SABIC INNOVATIVE PLASTICS US LLC SELKIRK, NY NPDES: NY0007072	Surface Water	Active Releaser: NPDES NY0007072	Surface water	250	0.00510	1.94E-06	5.67E-05	3.38E-05	2.83E-06
				20	0.0641	2.44E-05	7.12E-04	4.25E-04	3.56E-05

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EQUISTAR CHEMICALS LP LA PORTE, TX NPDES: TX0119792	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	4.31	1.64E-03	4.79E-02	2.85E-02	2.39E-03
				20	53.62	2.04E-02	5.96E-01	3.55E-01	2.98E-02
CHEMOURS COMPANY FC LLC WASHINGTON, WV NPDES: WV0001279	Surface Water	Active Releaser: NPDES WV0001279	Surface water	250	0.00299	1.14E-06	3.32E-05	1.98E-05	1.66E-06
				20	0.0371	1.41E-05	4.12E-04	2.46E-04	2.06E-05
SHINTECH ADDIS PLANT A ADDIS, LA NPDES: LA0111023	Surface Water	Active Releaser: NPDES LA0055794	Surface water	250	0.0000417	1.59E-08	4.63E-07	2.76E-07	2.32E-08

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				20	0.000526	2.00E-07	5.84E-06	3.48E-06	2.92E-07
STYROLUTION AMERICA LLC CHANNAHON, IL NPDES: IL0001619	Surface Water	Active Releaser: NPDES IL0001619	Surface water	250	0.000230	8.75E-08	2.56E-06	1.52E-06	1.28E-07
				20	0.00288	1.10E-06	3.20E-05	1.91E-05	1.60E-06
DOW CHEMICAL CO DALTON PLANT DALTON, GA NPDES: GA0000426	Surface Water	Active Releaser: NPDES GA0000426	Surface water	250	0.00648	2.46E-06	7.20E-05	4.29E-05	3.60E-06
				20	0.0811	3.08E-05	9.01E-04	5.37E-04	4.51E-05

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PREGIS INNOVATIVE PACKAGING INC WURLAND, KY NPDES: KY0094005	Surface Water	Active Releaser (Surrogate): Plastic Resins and Synthetic Fiber Manuf.	Surface water	250	0.0116	4.41E-06	1.29E-04	7.68E-05	6.44E-06
				20	0.15	5.70E-05	1.67E-03	9.93E-04	8.33E-05
OES: CTA Film Manufacturing									
KODAK PARK DIVISION ROCHESTER, NY NPDES: NY0001643	Surface Water	Active Releaser: NPDES NY0001643	Surface water	250	0.1100	4.18E-05	1.22E-03	7.28E-04	6.11E-05
				20	1.36	5.17E-04	1.51E-02	9.01E-03	7.56E-04
OES: Lithographic Printer									
	Surface Water	Active Releaser (Surrogate): Printing		250	0.0000540	2.05E-08	6.00E-07	3.58E-07	3.00E-08

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FORMER REXON FACILITY AKA ENJEMS MILLWORKS WAYNE TWP, NJ NPDES: NJG218316			Surface water						
				20	0.000677	2.57E-07	7.52E-06	4.48E-06	3.76E-07
OES: Spot Cleaner									
BOISE STATE UNIVERSITY BOISE, ID NPDES: IDG911006	Surface Water	Active Releaser (Surrogate): NPDES ID0020443	Surface water	250	0.00602	2.29E-06	6.69E-05	3.99E-05	3.34E-06
				20	0.0753	2.86E-05	8.37E-04	4.99E-04	4.18E-05
OES: Recycling and Disposal									
JOHNSON MATTHEY WEST DEPTFORD, NJ NPDES: NJ0115843	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	250	147.01	5.59E-02	1.63E+00	9.74E-01	8.17E-02
				250	123.89	4.71E-02	1.38E+00	8.20E-01	6.88E-02

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CLEAN HARBORS DEER PARK LLC LA PORTE, TX NPDES: TX0005941	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water						
CLEAN HARBORS EL DORADO LLC EL DORADO, AR NPDES: AR0037800	Non-POTW WWT	Receiving Facility: Clean Harbors of Baltimore, Inc; POTW (Ind.)	Surface water	250	26.68	1.01E-02	2.96E-01	1.77E-01	1.48E-02
TRADEBE TREATMENT & RECYCLING LLC EAST CHICAGO, IN FRS: 110000397874	Non-POTW WWT	Receiving Facility: ADVANCED WASTE SERVICES OF INDIANA LLC and BEAVER OIL TREATMENT AND RECYCLING; POTW (Ind.)	Surface water	250	4.52	1.72E-03	5.02E-02	2.99E-02	2.51E-03
VEOLIA ES TECHNICAL SOLUTIONS LLC WEST CARROLLTON, OH FRS: 110000394920	POTW	Receiving Facility: WESTERN REGIONAL WRF; NPDES OH0026638	Surface water	250	0.00785	2.98E-06	8.72E-05	5.20E-05	4.36E-06
VEOLIA ES TECHNICAL SOLUTIONS LLC AZUSA, CA FRS: 110000477261	POTW	Receiving Facility: SAN JOSE CREEK WATER RECLAMATION PLANT; NPDES CA0053911	Surface water	250	0.00389	1.48E-06	4.32E-05	2.58E-05	2.16E-06
VEOLIA ES TECHNICAL SOLUTIONS LLC	Non-POTW WWT	Receiving Facility: MIDDLESEX COUNTY UTILITIES AUTHORITY; NPDES: NJ0020141	Still body	250	0.00504	1.92E-06	5.60E-05	3.34E-05	2.80E-06

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MIDDLESEX, NJ NPDES: NJ0127477									
		Receiving Facility: Clean Harbors; POTW (Ind.)	Surface water	250	18100	6.88E+00	2.01E+02	1.20E+02	1.01E+01
CHEMICAL WASTE MANAGEMENT EMELLE, AL NPDES: AL0050580	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	1.84	7.00E-04	2.04E-02	1.22E-02	1.02E-03
				20	23.20	8.82E-03	2.58E-01	1.54E-01	1.29E-02
OILTANKING HOUSTON INC HOUSTON, TX NPDES: TX0091855	Surface Water	Active Releaser (Surrogate): NPDES TX0065943	Surface water	250	7.22	2.75E-03	8.02E-02	4.78E-02	4.01E-03
				20	90.00	3.42E-02	1.00E+00	5.96E-01	5.00E-02

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HOWARD CO ALFA RIDGE LANDFILL MARRIOTTSVILLE, MD NPDES: MD0067865	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0313	1.19E-05	3.48E-04	2.07E-04	1.74E-05
				20	0.39	1.48E-04	4.33E-03	2.58E-03	2.17E-04
CLIFFORD G HIGGINS DISPOSAL SERVICE INC SLF KINGSTON, NJ NPDES: NJG160946	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.0124	4.71E-06	1.38E-04	8.21E-05	6.89E-06
				20	0.16	6.08E-05	1.78E-03	1.06E-03	8.89E-05
CLEAN WATER OF NEW YORK INC STATEN ISLAND, NY NPDES: NY0200484	Surface Water	Active Releaser (Surrogate): NPDES NJ0000019	Still body	250	28.00	1.06E-02	3.11E-01	1.85E-01	1.56E-02

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				20	352.94	1.34E-01	3.92E+00	2.34E+00	1.96E-01
FORMER CARBORUNDUM COMPLEX SANBORN, NY NPDES: NY0001988	Surface Water	Active Releaser (Surrogate): POTW (Ind.)	Surface water	250	0.13	4.94E-05	1.44E-03	8.61E-04	7.22E-05
				20	1.57	5.97E-04	1.74E-02	1.04E-02	8.72E-04
OES: Other									
APPLIED BIOSYSTEMS LLC PLEASANTON, CA FRS: 110020517010	Non-POTW WWT	Receiving Facility: Evoqua Water Technologies; POTW (Ind.)	Surface water	250	10.02	3.81E-03	1.11E-01	6.64E-02	5.57E-03
EMD MILLIPORE CORP JAFFREY, NH NPDES: NHR05C584	POTW	Receiving Facility: JAFFREY WASTEWATER TREATMENT FACILITY; NPDES NH0100595	Surface water	250	0.18	6.84E-05	2.00E-03	1.19E-03	1.00E-04

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GBC METALS LLC SOMERS THIN STRIP WATERBURY, CT NPDES: CT0021873	Surface Water	Active Releaser: NPDES CT0021873	Surface water	250	0.00491	1.87E-06	5.46E-05	3.25E-05	2.73E-06
				20	0.0614	2.33E-05	6.82E-04	4.07E-04	3.41E-05
HYSTER-YALE GROUP, INC SULLIGENT, AL NPDES: AL0069787	Surface Water	Active Releaser: Motor Vehicle Manuf.	Surface water	250	0.000180	6.84E-08	2.00E-06	1.19E-06	1.00E-07
				20	0.00234	8.90E-07	2.60E-05	1.55E-05	1.30E-06
AVNET INC (FORMER IMPERIAL SCHRADE) ELLENVILLE, NY NPDES: NY0008087	Surface Water	Active Releaser: Electronic Components Manuf.	Surface water	250	0.0402	1.53E-05	4.47E-04	2.66E-04	2.23E-05
				20	0.50	1.90E-04	5.56E-03	3.31E-03	2.78E-04

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BARGE CLEANING AND REPAIR CHANNELVIEW, TX NPDES: TX0092282	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.11	4.18E-05	1.22E-03	7.28E-04	6.11E-05
				20	1.320	5.02E-04	1.47E-02	8.74E-03	7.33E-04
AC & S INC NITRO, WV NPDES: WV0075621	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.0188	7.15E-06	2.09E-04	1.25E-04	1.04E-05
				20	0.24	9.13E-05	2.67E-03	1.59E-03	1.33E-04
MOOG INC - MOOG IN-SPACE PROPULSION ISP NIAGARA FALLS,	Surface Water	Active Releaser: Metal Finishing	Surface water	250	0.00485	1.84E-06	5.39E-05	3.21E-05	2.69E-06

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
NY NPDES: NY0203700									
				20	0.0602	2.29E-05	6.69E-04	3.99E-04	3.34E-05
OILTANKING JOLIET CHANNAHON, IL NPDES: IL0079103	Surface Water	Active Releaser (Surrogate): NPDES IL0001619	Surface water	250	0.00088	3.36E-07	9.81E-06	5.85E-06	4.91E-07
				20	0.0111	4.22E-06	1.23E-04	7.35E-05	6.17E-06
NIPPON DYNAWAVE PACKAGING COMPANY LONGVIEW, WA NPDES: WA0000124	Surface Water	Active Releaser: NPDES WA0000124	Surface water	250	0.000703	2.67E-07	7.81E-06	4.66E-06	3.91E-07
				20	0.00879	3.34E-06	9.77E-05	5.82E-05	4.88E-06

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
TREE TOP INC WENATCHEE PLANT WENATCHEE, WA NPDES: WA0051527	Surface Water	Active Releaser (Surrogate): NPDES WA0023949	Surface water	250	0.000000352	1.34E-10	3.91E-09	2.33E-09	1.96E-10
				20	0.00000440	1.67E-09	4.89E-08	2.91E-08	2.44E-09
CAROUSEL CENTER SYRACUSE, NY NPDES: NY0232386	Surface Water	Active Releaser: POTW (Ind.)	Surface water	250	0.000322	1.22E-07	3.58E-06	2.13E-06	1.79E-07
				20	0.00396	1.51E-06	4.40E-05	2.62E-05	2.20E-06
OES: DoD									
US DOD USAF ROBINS AFB ROBINS AFB, GA NPDES: GA0002852	Surface Water	Active Releaser (Surrogate): NPDES GA0024538	Surface water	250	0.00182	6.92E-07	2.02E-05	1.21E-05	1.01E-06
				20	0.0228	8.67E-06	2.53E-04	1.51E-04	1.27E-05

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
OES: N/A (WWTP)									
EDWARD C. LITTLE WRP EL SEGUNDO, CA NPDES: CA0063401	Surface Water	Active Releaser (Surrogate): NPDES CA0000337	Still water	365	0.00601	2.29E-06	6.68E-05	3.98E-05	3.34E-06
				20	0.11	4.18E-05	1.22E-03	7.28E-04	6.11E-05
JUANITA MILLENDER-MCDONALD CARSON REGIONAL WRP CARSON, CA NPDES: CA0064246	Surface Water	Active Releaser (Surrogate): NPDES CA0000337	Still water	365	0.00127	4.83E-07	1.41E-05	8.41E-06	7.06E-07
				20	0.0232	8.82E-06	2.58E-04	1.54E-04	1.29E-05
LONDON WTP LONDON, OH NPDES: OH0041734	Surface Water	Active Releaser (Surrogate): NPDES OH0023779	Surface water	365	0.21	7.98E-05	2.33E-03	1.39E-03	1.17E-04

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
				20	3.74	1.42E-03	4.16E-02	2.48E-02	2.08E-03
LONG BEACH (C) WPCP LONG BEACH, NY NPDES: NY0020567	Surface Water	Active Releaser: NPDES NY0020567	Still water	365	322.14	1.22E-01	3.58E+00	2.13E+00	1.79E-01
				20	5857.02	2.23E+00	6.51E+01	3.88E+01	3.25E+00
MIDDLESEX COUNTY UTILITIES AUTHORITY SAYREVILLE, NJ NPDES: NJ0020141	Surface Water	Active Releaser: NPDES NJ0020141	Still water	365	2.79	1.06E-03	3.10E-02	1.85E-02	1.55E-03
				20	50.90	1.94E-02	5.66E-01	3.37E-01	2.83E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
JOINT WATER POLLUTION CONTROL PLANT CARSON, CA NPDES: CA0053813	Surface Water	Active Releaser: NPDES CA0053813	Still water	365	0.00665	2.53E-06	7.39E-05	4.40E-05	3.69E-06
				20	0.12	4.56E-05	1.33E-03	7.95E-04	6.67E-05
HYPERION TREATMENT PLANT PLAYA DEL REY, CA NPDES: CA0109991	Surface Water	Active Releaser: NPDES CA0109991	Still water	365	0.00359	1.37E-06	3.99E-05	2.38E-05	1.99E-06
				20	0.0656	2.49E-05	7.29E-04	4.34E-04	3.64E-05
SD CITY PT LOMA WASTEWATER TREATMENT SAN DIEGO, CA NPDES: CA0107409	Surface Water	Active Releaser: NPDES CA0107409	Still water	365	1.08	4.11E-04	1.20E-02	7.15E-03	6.00E-04
				20	19.74	7.51E-03	2.19E-01	1.31E-01	1.10E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
REGIONAL SANITATION DISTRICT ELK GROVE, CA NPDES: CA0077682	Surface Water	Active Releaser: NPDES CA0077682	Surface water	365	0.0151	5.74E-06	1.68E-04	1.00E-04	8.39E-06
				20	0.27	1.03E-04	3.00E-03	1.79E-03	1.50E-04
BERGEN POINT STP & BERGEN AVE DOCK W BABYLON, NY NPDES: NY0104809	Surface Water	Active Releaser: NPDES NY0104809	Still water	365	3.65	1.39E-03	4.06E-02	2.42E-02	2.03E-03
				20	66.40	2.52E-02	7.38E-01	4.40E-01	3.69E-02
NEW ROCHELLE STP NEW ROCHELLE, NY NPDES: NY0026697	Surface Water	Active Releaser: NPDES NY0026697	Still water	365	0.68	2.59E-04	7.56E-03	4.50E-03	3.78E-04

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
				20	12.47	4.74E-03	1.39E-01	8.26E-02	6.93E-03
SIMI VLY CNTY SANITATION SIMI VALLEY, CA NPDES: CA0055221	Surface Water	Active Releaser: NPDES CA0055221	Surface water	365	0.82	3.12E-04	9.11E-03	5.43E-03	4.56E-04
				20	14.88	5.66E-03	1.65E-01	9.85E-02	8.27E-03
OCEANSIDE OCEAN OUTFALL OCEANSIDE, CA NPDES: CA0107433	Surface Water	Active Releaser: NPDES CA0107433	Still water	365	0.66	2.51E-04	7.33E-03	4.37E-03	3.67E-04
				20	12.00	4.56E-03	1.33E-01	7.95E-02	6.67E-03

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
SANTA CRUZ WASTEWATER TREATMENT PLANT SANTA CRUZ, CA NPDES: CA0048194	Surface Water	Active Releaser: NPDES CA0048194	Still water	365	0.11	4.18E-05	1.22E-03	7.28E-04	6.11E-05
				20	2.07	7.87E-04	2.30E-02	1.37E-02	1.15E-03
CORONA WWTP 1 CORONA, CA NPDES: CA8000383	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.61	2.32E-04	6.78E-03	4.04E-03	3.39E-04
				20	11.10	4.22E-03	1.23E-01	7.35E-02	6.17E-03
BLIND BROOK SD WWTP RYE, NY NPDES: NY0026719	Surface Water	Active Releaser: NPDES NY0026719	Still water	365	0.17	6.46E-05	1.89E-03	1.13E-03	9.44E-05
				20	3.11	1.18E-03	3.46E-02	2.06E-02	1.73E-03

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
MCKINLEYVILLE CSD - WASTEWATER TREATMENT PLANT MCKINLEYVILLE, CA NPDES: CA0024490	Surface Water	Active Releaser: NPDES CA0024490	Surface water	365	0.14	5.32E-05	1.56E-03	9.27E-04	7.78E-05
				20	2.47	9.39E-04	2.74E-02	1.64E-02	1.37E-03
SAN JOSE CREEK WATER RECLAMATION PLANT WHITTIER, CA NPDES: CA0053911	Surface Water	Active Releaser: NPDES CA0053911	Surface water	365	0.00556	2.11E-06	6.18E-05	3.68E-05	3.09E-06
				20	0.1000	3.80E-05	1.11E-03	6.62E-04	5.56E-05
CARMEL AREA WASTEWATER DISTRICT TREATMENT	Surface Water	Active Releaser: NPDES CA0047996	Still water	365	0.08	3.16E-05	9.23E-04	5.50E-04	4.62E-05

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
FACILITY CARMEL, CA NPDES: CA0047996									
				20	1.52	5.78E-04	1.69E-02	1.01E-02	8.44E-04
CAMERON TRADING POST WWTP CAMERON, AZ NPDES: NN0021610	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.08	3.17E-05	9.28E-04	5.53E-04	4.64E-05
				20	1.52	5.78E-04	1.69E-02	1.01E-02	8.44E-04
CITY OF RED BLUFF WASTEWATER RECLAMATION PLANT RED BLUFF, CA NPDES: CA0078891	Surface Water	Active Releaser: NPDES CA0078891	Surface water	365	0.000074	2.82E-08	8.24E-07	4.91E-07	4.12E-08
				20	0.00135	5.13E-07	1.50E-05	8.94E-06	7.50E-07

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
91ST AVE WASTEWATER TREATMENT PLANT TOLLESON, AZ NPDES: AZ0020524	Surface Water	Active Releaser: NPDES AZ0020524	Surface water	365	0.25	9.51E-05	2.78E-03	1.66E-03	1.39E-04
				20	4.52	1.72E-03	5.02E-02	2.99E-02	2.51E-03
EVERETT WATER POLLUTION CONTROL FACILITY EVERETT, WA NPDES: WA0024490	Surface Water	Active Releaser: NPDES WA0024490	Surface water	365	0.85	3.23E-04	9.44E-03	5.63E-03	4.72E-04
				20	15.54	5.91E-03	1.73E-01	1.03E-01	8.63E-03
PIMA COUNTY - INA ROAD WWTP TUCSON, AZ NPDES: AZ0020001	Surface Water	Active Releaser: NPDES AZ0020001	Surface water	365	1.02	3.88E-04	1.13E-02	6.75E-03	5.67E-04
				20	18.59	7.07E-03	2.07E-01	1.23E-01	1.03E-02

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
23RD AVENUE WASTEWATER TREATMENT PLANT PHOENIX, AZ NPDES: AZ0020559	Surface Water	Active Releaser: NPDES AZ0020559	Surface water	365	0.14	5.32E-05	1.56E-03	9.27E-04	7.78E-05
				20	2.49	9.47E-04	2.77E-02	1.65E-02	1.38E-03
SUNNYSIDE STP SUNNYSIDE, WA NPDES: WA0020991	Surface Water	Active Releaser: NPDES WA0020991	Surface water	365	0.00611	2.32E-06	6.79E-05	4.05E-05	3.39E-06
				20	0.11	4.18E-05	1.22E-03	7.28E-04	6.11E-05
AGUA NUEVA WRF TUCSON, AZ NPDES: AZ0020923	Surface Water	Active Releaser: NPDES AZ0020923	Surface water	365	0.0292	1.11E-05	3.24E-04	1.93E-04	1.62E-05

Name, Location, and ID of Active Releaser Facility ^a	Release Media ^b	Modeled Facility or Industry Sector in EFAST ^c	EFAST Waterbody Type ^d	Days of release ^e	7Q10 SWC (ppb) ^g	Acute Risk Quotients (using COC of 2,630 ppb)	Chronic Risk Quotients (using amphibian COC of 90)	Chronic Risk Quotients (using fish COC of 151)	Chronic Risk Quotients (using invertebrate COC of 1,800)
				20	0.53	2.02E-04	5.89E-03	3.51E-03	2.94E-04
PORT OF SUNNYSIDE INDUSTRIAL WWTF SUNNYSIDE, WA NPDES: WA0052426	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.24	9.13E-05	2.67E-03	1.59E-03	1.33E-04
				20	4.45	1.69E-03	4.94E-02	2.95E-02	2.47E-03
APACHE JUNCTION WWTP APACHE JUNCTION, AZ NPDES: AZ0023931	Surface Water	Active Releaser: POTW (Ind.)	Surface water	365	0.04	1.51E-05	4.40E-04	2.62E-04	2.20E-05
				20	0.72	2.74E-04	8.00E-03	4.77E-03	4.00E-04

a. Facilities actively releasing methylene chloride were identified via DMR and TRI databases for the 2016 reporting year.

- b. Release media are either direct (release from active facility directly to surface water) or indirect (transfer of wastewater from active facility to a receiving POTW or non-POTW WWTP facility). A wastewater treatment removal rate of 57% is applied to all indirect releases, as well as direct releases from WWTPs.
- c. If a valid NPDES of the direct or indirect releaser was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.
- d. EFAST uses either the “surface water” model, for rivers and streams, or the “still water” model, for lakes, bays, and oceans.
- e. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.
- f. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.
- g. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.
- h. To determine the PDM days of exceedance for still bodies of water, the estimated number of release days should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.

Appendix I DERIVATION OF IUR AND NON-CANCER HUMAN EQUIVALENT CONCENTRATION FOR CHRONIC EXPOSURES

The reader is referred to *Risk Evaluation for Methylene Chloride, Supplemental File – Methylene Chloride Benchmark Dose and PBPK Modeling Report* ([EPA, 2019h](#)) for additional details on dose metrics, models used to derive the IUR as well as individual model outputs.

I.1 Cancer Inhalation Unit Risk

Methylene chloride's cancer IUR of 1.38×10^{-6} per mg/m^3 ⁽²⁹⁾ was derived from mouse liver and lung tumor incidence data ([Mennear et al., 1988](#); [NTP, 1986](#)). Figure_Apx I-1 describes the steps used to derive the methylene chloride IUR using PBPK modeling. Because this modeling is updated from the model used for the methylene chloride IRIS assessment, additional details on aspects of IUR derivation are included in the IRIS assessment ([U.S. EPA, 2011](#)).

The derivation steps are the following:

1. **Dose conversion:** A deterministic mouse PBPK model ([Marino et al., 2006](#)) was used to convert the mouse inhalation exposures to long-term daily average internal doses in the liver or lung. The selected internal dose-metric was long-term average daily mass of methylene chloride metabolized *via* the GST pathway per unit volume of liver or lung tissue. The choice of the dose metric was based on evidence related to the involvement of the GST metabolites in methylene chloride-induced carcinogenicity ([U.S. EPA, 2011](#)).
2. **Dose-response modeling and extrapolation:** All dichotomous models that use likelihood optimization and profile likelihood-base CIs from BMDS version 3.1 were used to fit the mouse liver and lung tumor incidence and PBPK-derived internal doses and derive a mouse internal BMD_{10} and BMDL_{10} ³⁰ associated with 10% ER ([U.S. EPA, 2011](#)). Several tumors using multiple models were evaluated. The chosen model was the multi-tumor (MS_Comb) model, which uses individual Multistage models fit to the individual (liver and lung) tumors to estimate the risk of getting one or more of the tumors being analyzed ([EPA, 2019h](#)).

Standard and non-standard forms of these models were run separately in BMDS 3.1 so that auto-generated model selection recommendations accurately reflect current EPA model selection procedures ([EPA, 2012](#), [EPA, 2014](#)). BMDS 3.1 models that use Bayesian fitting procedures and Bayesian model averaging were not applied in this work.

²⁹ The inhalation unit risk for methylene chloride should not be used with exposures exceeding the point of departure ($\text{BMDL}_{10} = 7,700 \text{ mg}/\text{m}^3$ or 2,200 ppm), because above this level the fitted dose-response model better characterizes what is known about the carcinogenicity of methylene chloride.

³⁰ The benchmark dose (BMD) is a dose or concentration that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background ([U.S. EPA, 2011](#)).

BMD_{10} = benchmark dose at the 10% response

BMDL_{10} =lower confidence limit of the benchmark dose at the 10% response

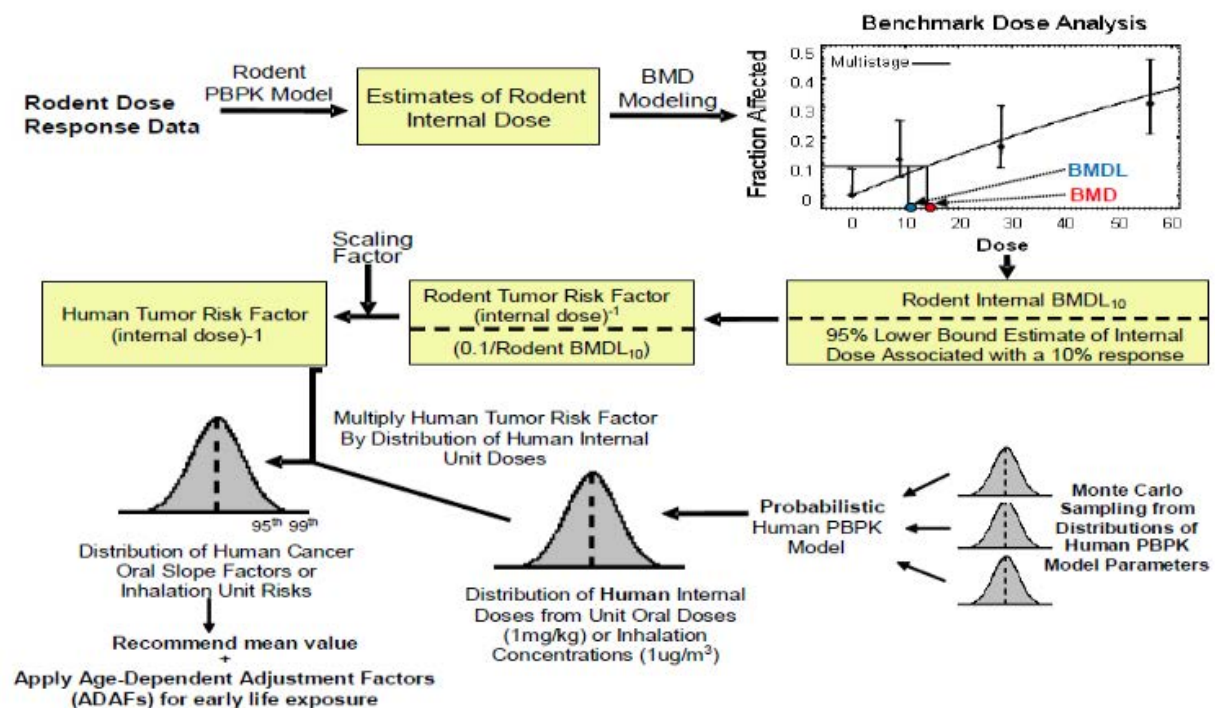
The mouse internal BMDL₁₀ (0.1/BMDL₁₀) were used to derive inhalation risk factors for lung and liver tumors by linear extrapolation. Consistent with EPA *Guidelines for Carcinogen Risk Assessment*, a linear low-dose extrapolation approach is used for chemicals with DNA-reactive and mutagenic properties ([EPA, 2005b](#)).

3. **Application of allometric scaling factor:** The chosen dose metric is a rate of metabolism rather than the concentration of putative toxic metabolites. Currently, there are no data pertaining to the reactivity or clearance rate of the relevant metabolite(s). A scaling factor was used to address the possibility that the rate of clearance for the metabolite is limited by processes that are known to scale allometrically. The human BMDL₁₀ was derived by applying a mouse:human dose-rate scaling factor of 7 [i.e., (Body Weight human/Body Weight mouse)^{0.25} = 7] to adjust the mouse-based BMDL₁₀ values downward based on the potential slower clearance per volume tissue in the human compared with the mouse ([EPA, 2019h](#); [U.S. EPA, 2011](#)).
4. **Linear extrapolation:** A linear extrapolation approach using the internal human BMDL₁₀ for liver and lung tumors was used to calculate human tumor risk factors by dividing the BMR of 0.1 by the human BMDL for each tumor type for adults aged 18-65. Currently, there are no data from chronic inhalation cancer bioassays in mice or rats providing support for a nonlinear dose-response relationship at low doses. ; ([EPA, 2019h](#); [U.S. EPA, 2011](#)).
5. **Calculation of the IUR:** A probabilistic human PBPK model (adapted from David ([2006](#))) with Monte Carlo sampling was used to determine a distribution of human internal doses - lung, liver, or blood - associated with chronic unit inhalation (1 µg/m³) exposures. The distribution of IURs was derived by multiplying the human inhalation tumor risk factors by the respective distributions of human average daily internal doses resulting from chronic, unit inhalation exposures of one µg/m³ methylene chloride.

Sampling of the full distribution of GSTT1 genotypes in the human population (GSTT1^{+/+}, GSTT1^{+/-} and GSTT1^{-/-}) was done to derive the IUR for liver and lung tumors. To model the distribution of GST-T1-mediated metabolism characterized by the rate coefficient, k_{FC} , David et al. ([2006](#)) used the known distribution of GST-T1 genotypes in the U.S. population ([Haber et al., 2002](#)) and the genotype-specific activity distributions from Warholm et al. ([1994](#)) scaled to have the same mean value as the overall mean estimate of the population mean obtained by David et al. ([2006](#)): 0.852 kg^{0.3}/hour. However, because David et al. ([2006](#)) did not incorporate the uncertainty of the population mean, EPA used a two-dimensional sampling technique for k_{FC} . First, EPA sampled $k_{FC,mean}$ from a log-normal distribution with GM = 0.6944 kg^{0.3}/hour and GSD = 1.896 kg^{0.3}/hour (converted from the linear-space mean of 0.852 kg^{0.3}/hour and CV of 0.711 from David et al. ([2006](#))). Then EPA sampled an individual's genotype from the discrete incidence distribution, which was 32% chance of GST-T1 ^{+/+}, 48% chance of ^{+/-}, and 20% chance of ^{-/-} ([Haber et al., 2002](#)). Given those genotype frequencies, the interindividual variability was then characterized by rescaling the activity distributions from Warholm et al. ([1994](#)), using upper and lower bounds

of zero and mean + 5 SDs, respectively instead of zero and mean + 3 SDs used by David et al. (2006).

The slope of the linear extrapolation from the lower 95 percent bound estimate $BMDL_{10}$ is 1.38×10^{-6} per mg/m^3 , which represents an upper-bound estimate for exposure for adult workers 18-65 years old, 8 hrs/day, 5 days/week without consideration of increased early-life susceptibility due to methylene chloride's mutagenic MOA because the IUR is used for scenarios in occupational settings where only adults are expected to be exposed. Use of the upper-bound estimate for the full population distribution of the GSTT1 genotypes is considered sufficiently protective of sensitive sub-populations.



Figure_Apx I-1. Process of Deriving the Cancer Inhalation Unit Risk for Methylene Chloride

Source: U.S. EPA (2011)

I.2 Non-Cancer Hazard Value

The non-cancer hazard value for methylene chloride is based on liver effects. These effects were reported in female rats exposed to methylene chloride for 6 hrs/day, 5 days/week for 2 years (Nitschke et al., 1988a). The rat data were suitable for non-cancer dose-response analysis.

Because the study was suitable for dose-response analysis, EPA used a PBPK model ([Andersen et al., 1991](#)) to estimate rat internal doses from the Nitschke ([1988a](#)) study. BMD modeling used the rat internal doses and their corresponding incidence data (i.e., hepatic vacuolation) to estimate the rat internal BMDL₁₀ for hepatic effects. In other words, the BMDL₁₀ is the lower 95% confidence limit of the BMD at the 10% BMR ([EPA, 2012a](#)). A BMR of 10% was selected because, in the absence of information regarding the magnitude of change in a response that is thought to be minimally biologically significant, a BMR of 10% is generally recommended since it provides a consistent basis of comparison across assessments. Moreover, there were no additional data to suggest that the severity of the critical effect or the power of the study would warrant a lower BMR ([U.S. EPA, 2011](#)).

The rat internal BMDL₁₀ was allometrically adjusted because the dose-metric is a rate of metabolism and the clearance of these metabolites may be slower per volume tissue in the human compared with the rat. This adjustment consisted of dividing the rat internal BMDL₁₀ by $4.09 [(BW_{\text{human}})/(BW_{\text{rat}})^{0.25} \approx 4.09]^{31}$ to obtain a human equivalent internal BMDL₁₀ of 130.03 mg methylene dichloride metabolized via CYP³² pathway/litter liver tissue/day ([EPA, 2019h](#)).

A probabilistic PBPK model for methylene chloride in humans (adapted from David ([2006](#))) was then used with Monte Carlo sampling to calculate distributions of chronic hHEC (in units of mg/m³) associated with the internal BMDL₁₀ based on the responses in female Sprague-Dawley rats. Estimated HECs corresponding to the mean, 1st, and 5th percentiles of the distribution were 48.5, 17.2 and 21.3 mg/m³, respectively. The 1st percentile of the distribution of HECs i.e., the HEC₉₉ the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard, 17.2 mg/m³, was chosen as the POD³³ for the non-cancer hazard value because it would protect toxicokinetically sensitive individuals. EPA's use of the human toxicokinetics data distribution is similar to using data-derived extrapolation factors (DDEFs) because it uses information more specific to methylene chloride hazard. DDEFs are suggested by agency guidance as preferable to default UFs ([EPA, 2014b](#)).

³¹ BW=body weight

³² CYP=cytochrome P450

³³ A POD is a dose or concentration that can be considered to be in the range of observed responses, without significant extrapolation. A POD is used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures ([U.S. EPA, 2011](#)).

Appendix J CASE REPORTS OF FATALITIES ASSOCIATED WITH METHYLENE CHLORIDE EXPOSURE

The main cause of death from high levels of inhalation of methylene chloride is related to CNS effects. This includes loss of consciousness and respiratory depression leading to irreversible coma, hypoxia and death ([Nac/Aegl, 2008b](#)). The organ most often affected in fatal accidents is the brain, followed by the lungs and heart. Changes in these organs include congestion and edema. The lung and heart also showed petechiae in a few cases. Cardiotoxic effects are observed in a few cases ([Nac/Aegl, 2008b](#)). Only one case, a 66-year old who was stripping furniture, had chest pains when using an 80% methylene chloride varnish without CNS depression; he died of myocardial infarction after the third use (Steward and Hake, 1976) cited in NAC/AEGL ([2008b](#)).

The Program on Reproductive Health and the Environment from University of California, San Francisco gathered fatality information from 10 sources that included PubMed, AAPCC, OSHA, CPSC, Lexis Nexis, News Bank, NIOSH, CPI and EASCR. A total of 85 fatalities were reported from the year 1980 to 2018, most in occupational users (> 80%) versus consumers. Of the reported product types, paint strippers were most often the cause (69%). Deaths occurred most in the bathroom (31%) and then in industrial settings (21%). Ages of the individuals ranged from 14 to 80, and most were white males. This information updates a previous similar analysis by Safer Chemicals, Health Families done in March 2018 that used CPSC and AAPCC information ([Schf, 2020](#)).

CDC ([2012](#)) provided some details regarding 13 deaths from bathtub refinishing using methylene chloride between 2000 to 2011, which are also likely to be included in the count above. The percent of methylene chloride in the paint strippers was 60-100%. Methylene chloride blood concentrations for six decedents ranged from 18 to 223 mg/L. Among 5 decedents with COHb measurements, levels ranged from undetected to 5%, indicating CO was unlikely to be the primary cause of death.

Although very few details of the exposures associated with deaths have been reported, Table Apx_J-1 identifies cases where air concentrations have been measured or estimated and/or blood concentrations were measured.

NIOSH lists a value of 2300 ppm (7981 mg/m³) as IDLH ([NIOSH, 1994](#)). Individuals should not be exposed to methylene chloride at this level for any length of time. The IDLH is based on acute inhalation toxicity data in humans. The AEGL-3 value for death ranges from 12,000 ppm (42,000 mg/m³) to 2100 ppm (7400 mg/m³) for a 10-min to 8-hr value, respectively. The value is based on mortality from CNS effects in rats and COHb formation in humans ([Nac/Aegl, 2008b](#)).

Table Apx J-1. Fatalities That Have Associated Exposure Concentrations

Subject (s)	Use	Circumstances of exposure	Cause of death, symptoms, autopsy	Possible methylene chloride air concentration (mixture identification)	Reference
27-year old male	Paint stripping (occupational)	Found dead 20-30 min after being alive; slumped over tank with paint stripper; head and trunk in tank, arms in solvent	<p>Cause of death: asphyxia secondary to inhalation of fumes Transported to hospital in cardio-respiratory arrest;</p> <p>Lungs: congestion/edema; micro-hemorrhagic changes; significant \uparrow in pigmented macrophages in alveoli/bronchioles;</p> <p>Liver: \uparrow consistency/size, mild portal inflammation, dilated centrilobular veins, acute congestion</p> <p>Methylene chloride: 0.14 mg/mL (blood), 0.54 mg/mL (pulmonary exudate) COHb: 3%</p>	<p>Samples taken after the accident: >140,000 mg/m³ (>39,200 ppm) (5-10 cm from solvent) 89,474 mg/m³ (25,053 ppm) (25 cm above solvent) 4789 mg/m³ (1341 ppm) (75 cm from solvent) 243 mg/m³ (68 ppm) and 390 mg/m³ (109 ppm) at level of upper airways of standing worker (resting/stirring) [colleagues suggest the worker had been very close to the solvent surface with his head]</p> <p>(77% methylene chloride; 18% methanol)</p>	Zarrabeitia et al. (2001) cited in NAC/AEGL (2008b)
19-year old male	Paint stripping of furniture (occupational)	Found slumped over immersion tank; arms and forehead submerged	<p>Cause of death: suffocation due to inhalation of toxic solvents</p> <p>Methylene chloride: 0.4 mg/mL (blood) Methanol: 2.4 mg/mL (blood) COHb: none found</p>	<p>Air concentrations: n/a (methylene chloride; methanol)</p>	Novak and Hain (1990) cited in NAC/AEGL (2008b)
21-year old male	Paint stripping of furniture (occupational)	Found unconscious with head and shoulders submerged in solvent; man was resuscitated, remained comatose and died 7 days later	<p>Methylene chloride: n/a Methanol: 0.2 mg/mL COHb: 3.6%</p>	<p>Re-enactment air samples: 1711, 89, and \geq 771 ppm of methylene chloride, toluene and methanol, respectively at 10 cm above surface. 64, 6, and \geq 44 ppm, respectively at top of tank (76 cm above surface)</p>	Novak and Hain (1990) cited in NAC/AEGL (2008b)

Subject (s)	Use	Circumstances of exposure	Cause of death, symptoms, autopsy	Possible methylene chloride air concentration (mixture identification)	Reference
				<p>100, 3, and ≥ 124 ppm (55-min samples) and 313, 13 ppm and NA (10-min samples) (76 cm away from tank at breathing zone)</p> <p>(65-85% methylene chloride, 6-12% methanol, 6-12% toluene, monoethanolamine)</p>	
50 and 55-year old men	Burying waste barrels (occupational)	Burying barrels of mixed solvent and solid waste from nearby plant for a few hours (in well 2 meters below ground level in a building); found dead in evening; death estimated as early afternoon	<p>Cause of death: narcosis, loss of consciousness, respiratory depression and irreversible coma, hypoxia and death</p> <p>Besides respiratory depression, levels of formaldehyde, formic acid and carbon dioxide may have led to hypoxia, cardio-respiratory failure, and death.</p> <p>Methylene chloride: 0.572 and 0.601 mg/mL (blood) COHb: 30%</p>	<p>Air concentrations:</p> <p>Near well, soon after discovery of bodies: 1,800 and 10,700 mg/m³ (504 and 2996 ppm) -</p> <p>Bottom of well, next day: 582,500 mg/m³ (163,100 ppm)</p> <p>Near bodies, next day: 72,900 mg/m³ (20,412 ppm)</p> <p>Concentrations of other solvents (1,2-dichloroethane, 1,1,1-trichloroethane, and styrene) were much lower</p>	Manno et al. (1989, 1992) cited in NAC/AEGL (2008b)
20- and 40-year olds	Paint stripping (occupational)	Removing original surface of squash court, found dead at 2 hrs and 20 min after starting; not known whether they stayed in the room or left and returned	N/A	<p>Air concentrations: 53,000 ppm (estimated from amount of stripper used, room size, etc.) ($> 80\%$ methylene chloride)</p>	Fairfax (1996) cited in NAC/AEGL (2008b)

Subject (s)	Use	Circumstances of exposure	Cause of death, symptoms, autopsy	Possible methylene chloride air concentration (mixture identification)	Reference
N/A	Paint stripping (occupational)	Occupational poisoning in a plant where the employee was using a paint stripper	N/A	Air concentration: ≤ 100,000 ppm (estimated) (75% methylene chloride)	Tay et al. (1995) cited in NAC/AEGL (2008b)
13-year old male	Paint stripping (consumer)	N/A	Cause of death: Narcosis Methylene chloride: 0.510 mg/mL (blood) 0.248 mg/g (brain) COHb: 3.0	Air concentrations: n/a (methylene chloride, toluene, methanol, ethanol, mineral spirit, methyl ethyl ketone, and n-methylpyrimidol tetraethylammonium phosphate)	Bonventre et al. (1977) cited in NAC/AEGL (2008b)
37-yr old female	Bathtub refinishing (occupational)	Found unresponsive; slumped over the bathtub; No respiratory protection or ventilation controls	Cause of death: Inhalation exposure of paint remover pulmonary edema and congestion; congestion of the conjunctivae; hyperemia of the small bowel and gastric mucosa; and dilated right ventricle. Methylene chloride: 0.12 mg/mL (blood) Methanol: 7 mg/dL (blood)	Air concentrations: 23,000 ppm (estimate based on volume removed from can) (80-90% methylene chloride, 5-10% methanol)	Iowa FACE (2012b)
24-yr old male, no known health problems	Paint stripping (occupational)	Stripping baptismal font in small enclosed room; found unresponsive 6.5 hrs later	Cause of death: Intoxication by methylene chloride resulting in hypoxia, dysrhythmia, death. Autopsy: identified underlying cardiopulmonary disease (found cardiomegaly with 4-chamber dilation, arteriosclerosis – 50% in left anterior descending artery) Methylene chloride:	Air concentrations: n/a (70-85% methylene chloride, smaller amounts of methanol, isopropyl alcohol, 2-butoxy-ethanol, and ethanol)	MacIsaac et al. (2013); CaFACE (2012a)

Subject (s)	Use	Circumstances of exposure	Cause of death, symptoms, autopsy	Possible methylene chloride air concentration (mixture identification)	Reference
			37.8 mg/dL (blood) Other chems (methanol, ethanol, isopropyl alcohol) undetectable in blood COHb: 10%		
65-yr old male, history of diabetes and chronic neuropathic pain; medications metformin and gabapentin	Paint stripping (occupational)	Entered empty paint-mixing tank through small opening in top; applied paint stripper to inside walls to remove paint; wore organic vapor cartridge respirator; fan and hose used for exhaust but positioned only halfway between tank opening and tank floor; found unconscious 2.5 hrs after entering tank	Cause of death: asphyxia due to inhalation of methylene chloride Found in state of asystole; congestion in lungs and myocardium Methylene chloride: 220 mg/dL (blood) COHb: < 5%	Air concentrations: n/a (60-100% methylene chloride, 10-30% methanol, 1-5% Stoddard solvent)	MacIsaac et al. (2013)
52-yr old male, no history of heart attack or asthma; medication for cholesterol	Bathtub stripping (occupational)	Found slumped over bathtub with face on bottom of tub; found ~2 hrs later	Cause of death: Sudden cardio-respiratory arrest due to inhalation of toxic fumes; Autopsy: mild atherosclerotic cardiovascular disease; heavy congested lungs with mucous plugging Methylene chloride: 50 mg/L COHb: negative	Air concentrations: 637-1062 ppm in room (estimated 1-hr TWA from volume used – 6 oz. – and room size) 11,618-19,364 ppm in tub (estimated 1-hr TWA) But average (assuming 80% mc) in tub estimated to be 123,933 ppm in tub (60-100% methylene chloride, 3-7% ethyl alcohol, smaller percent of other chemicals)	NIOSH (2011a) Also cited in NIOSH (2011a) ^a

^aSame as CDC (2012).

Appendix K SUMMARY OF METHYLENE CHLORIDE GENOTOXICITY DATA

This appendix provides a high-level summary of genotoxicity studies available for methylene chloride. Table Apx K-1 summarizes recent studies and one study not identified in EPA's 2011 IRIS assessment. The appendix also includes a summary of the conclusions from EPA's 2011 IRIS assessment ([U.S. EPA, 2011](#)) and reproduces Tables 4-20 through 4-25 from [U.S. EPA \(2011\)](#) as Table_Apx K-2 through Table_Apx K-7, with slight revisions and inclusion of data quality evaluation scores using data quality criteria developed for TSCA risk evaluations. EPA did not present studies that received unacceptable data quality ratings in the tables below. The supplemental file *Data Quality Evaluation of Human Health Hazard Studies – Animal and In Vitro Studies* ([EPA, 2019u](#)) presents the data quality ratings for all acceptable and unacceptable studies, including scores and comments for individual metrics.

Studies not Identified in the IRIS Assessment

Table_Apx K-1 summarizes recent studies and one older study ([Khudoley et al., 1987](#)) not identified in U.S. EPA ([2011](#)).

In peripheral blood lymphocyte/leukocyte samples of an occupational cohort exposed to methylene chloride and other possible/probable carcinogens, [Zeljezic et al. \(2016\)](#) found increased frequencies of micronuclei, nuclear buds and nucleoplasmic bridges as well as DNA damage in exposed subjects when compared with unexposed individuals. After implementing strict use of personal protective equipment (PPE), workers exhibited less genotoxicity than before strict use of PPE ([Zeljezic et al., 2016](#)).

[Suzuki et al. \(2014\)](#) found no increases in micronuclei in reticulocytes or normochromatic erythrocytes or gene mutations (using Pig-a assay) in total red blood cells of B6C3F1 mice exposed by inhalation to methylene chloride concentrations up to 1600 ppm (5615 mg/m³) for 6 weeks. In addition, [Suzuki et al. \(2014\)](#) did not identify an increase in gene mutations or DNA damage in the liver in transgenic *gpt* delta mice exposed to 800 ppm (2808 mg/m³) for 4 weeks. A study by this group also showed no evidence of mutagenicity in the livers of *gpt* delta rats orally exposed to methylene chloride alone (up to 500 mg/kg) or with up to 200 mg/kg-day 1,2-dichloropropane for 4 weeks ([Hirata et al., 2016](#)). Other recent studies reported positive results. In an *in vitro* study of normal rat kidney (NRK) cells, [Yang et al. \(2014\)](#) identified increased DNA damage (via the comet/SCGE assay) in the absence of cytotoxicity, apoptosis or G1 cell cycle arrest. [Mimaki et al. \(2016\)](#) evaluated mutagenicity of methylene chloride in *S. typhimurium* TA100 and found increased revertants/plate and an increased mutation rate in the absence of metabolic activation, similar to previous studies.

Table Apx K-1 Methylene Chloride Genotoxicity Studies not Cited in the 2011 IRIS Assessment

Species	Methylene Chloride Exposure		Outcome	Comments	Reference	Data Quality Evaluation
	Route	Dose/Duration				
Humans: workers in pharmaceutical industry	Inhalation/dermal most likely	8 hrs/day for \geq 8 months of irregular PPE use followed by 8 months of strict PPE use (same 16 worker volunteers for both phases)	<i>Irregular PPE</i> : Micronuclei, nuclear buds and nucleoplasmic bridges were higher in blood lymphocytes of workers exposed to multiple chemicals than controls. Tail length and percent DNA in tail of comet assay did not significantly differ from controls in blood leukocytes.	Workers were exposed to other possible carcinogens in addition to methylene chloride: phenylhydrazine, ethylene oxide, 1,2-dichloroethane; <i>Strict PPE</i> : some effects significantly decreased compared with irregular PPE after the strict use of PPE was implemented	Zeljezic et al. (2016)	NE
Mice: B6C3F1 males	Inhalation	0, 400, 800, 1600 ppm; 6 hrs/day, 5 days/week for 6 weeks	Total red blood cells – no increase in pig-A mutant frequencies Reticulocytes or normochromatic erythrocytes – no increase in micronuclei	Authors note that the results are indicative of lack of mutagenic potential in hematopoietic stem cells, and lack of clastogenicity/aneugenicity in bone marrow of mice	Suzuki et al. (2014)	High
Mice: <i>gpt</i> Delta C57BL/6J males		0, 800 ppm; 6 hrs/day, 5 days/week for 4 weeks	Liver – no increase in DNA damage via comet assay or <i>gpt</i> mutations	DNA damage and <i>gpt</i> mutations were increased after co-exposure of methylene chloride and 1,2-dichloropropane, suggesting that the mutagenic potential of 1,2-dichloropropane may be enhanced by methylene chloride		High
Rats: F344 <i>gpt</i> delta	Gavage	0, 250 or 500 mg/kg-bw via gavage in corn oil every day for 4 weeks	No increase in <i>Gpt</i> and Spi-mutation frequencies; no changes in gene or protein expression of GST-T1 or CYP2E1	The <i>gpt</i> delta rats carry approximately 10 copies of the transgene lambda EG10 per haploid genome	Hirata et al. (2016)	High
Rats: Normal rat kidney (NRK) 52 ^E cell line	<i>In vitro</i> assay	50 to 5000 mg/L (comet assay); 10 to ~10,000 mg/L (cytotoxicity – MTT - viability); 10 to 1000 mg/L (apoptosis assay); 5000 mg/L (cell cycle analysis)	DNA damage at 5×10^3 mg/L ($p < 0.05$) via comet (SCGE) assay; no increased cytotoxicity (MTT/cell viability or apoptotic cells); no changes in cell cycle	None	Yang et al. (2014)	High

Species	Methylene Chloride Exposure		Outcome	Comments	Reference	Data Quality Evaluation
	Route	Dose/Duration				
<i>S. typhimurium</i> TA100	<i>In vitro</i> reverse mutation assay	Up to 3500 ppm vapor concentration	Increased revertants/plate and increased mutation rate	No metabolic activation used; method modified for evaluation of volatile compounds	Mimaki et al. (2016)	High
<i>S. typhimurium</i> TA98, TA100	<i>In vitro</i> reverse mutation assay	Not reported	Increased revertants in the presence of activation	Methods and procedures were cited to other publications	Khudoley et al. (1987)	Medium

Genotoxicity Studies Summarized in the 2011 Methylene Chloride IRIS Assessment

Some overall conclusions from the genotoxicity data on methylene chloride identified by [U.S. EPA \(2011\)](#) are as follows:

- *In vitro* assays in nonmammalian organisms (bacteria, yeast, fungi) ([U.S. EPA, 2011](#)) Table 4-20 slightly revised and reproduced in Table_Apx K-2)
 - In bacteria, methylene chloride mutagenicity is enhanced in the presence of GSH for some strains.
 - In bacteria, consistent induction in TA100 and TA 98 that may be somewhat enhanced but is not markedly influenced by exogenous mammalian liver fractions. Thus, [U.S. EPA \(2011\)](#) suggested that endogenous metabolism in these strains was sufficient to activate methylene chloride.
 - A glutathione-deficient strain variant of TA100 (NG-11) produced 2 times fewer base-pair substitution mutations vs. TA100 that produces normal levels of GSH. Adding 1 mM GSH to NG-11 resulted in numbers of substitutions more similar to results using normal TA100.
 - TA1535, which is deficient in GST, did not develop base-pair mutations.
 - TA1535 transfected with rat GST-T1 showed base-pair substitution mutations at a DCM concentration 60x lower than that needed to induce mutations in TA100.
 - Based on these results, [U.S. EPA \(2011\)](#) notes the likelihood that genotoxicity involves the GST-T1 metabolic pathway, which produces S-(chloromethyl)glutathione and formaldehyde.
 - Fungal assays resulted in some positive results – for mitotic segregation (only seen at 4000 ppm but not 8000 ppm).
 - A yeast assay was positive for gene conversion and recombination at concentrations up to 209 mM.
- *In vitro* assays in mammalian systems ([U.S. EPA \(2011\)](#)) Table 4-21, slightly revised and reproduced in Table_Apx K-3)
 - In human cell lines, methylene chloride exposure yielded positive results in micronucleus and sister chromatid exchange assays but negative for unscheduled DNA synthesis and DNA SSBs.
 - In human lung epithelial cells that showed no GST-T1 activity, DNA damage via the comet assay exhibited a weak trend after methylene chloride exposure.
 - In human peripheral blood mononuclear cells from 20 volunteers that had low, medium or high GST-T1 activity, methylene chloride exposure induced genotoxicity and cytotoxicity at relatively low methylene chloride concentrations (sometimes starting at 30 ppm) that was stronger in the high GST-T1 activity cells. Outcomes included increased sister chromatid exchange, decreased mitotic indices and changes in cell proliferation kinetics.
 - At methylene chloride concentrations from 0.5 to 5 mM, DNA protein cross links exhibited a dose-response in mouse hepatocytes but not in rat, hamster and human hepatocytes.

- DNA single strand breaks (SSBs) were induced by methylene chloride in mouse hepatocytes and club (Clara) cells and SSBs were decreased after addition of a GSH depleter.
- DNA SSBs were induced at lower concentrations in mouse hepatocytes than in rat hepatocytes.
- Chinese hamster ovary cells incubated with GST-competent mouse liver cytosol induced gene mutations, DNA-protein cross-links and DNA SSBs.
- Calf thymus DNA in the presence of 1) methylene chloride dehalogenase/GST from bacteria and GSH 2) human GST-T1, 3) rat GST5-5 or 4) bacterial GST (from DM11) formed DNA adducts. However, calf thymus DNA with methylene chloride in the presence of formaldehyde and GSH did not result in detectable DNA adducts.
- Results of several experiments suggest that the S-(chloromethyl)glutathione intermediate is primarily responsible for methylene chloride's genotoxicity although there is evidence of DNA damage resulting from the formation of formaldehyde.
- *In vivo* assays in insects ([U.S. EPA \(2011\)](#) Table 4-22, slightly revised and reproduced in Table_Apx K-4)
 - In *Drosophila*, two oral methylene chloride studies (sex-linked recessive, somatic w/w+) resulted in positive findings whereas an inhalation study did not result in increased gene mutations.
- *In vivo* assays in mice ([U.S. EPA \(2011\)](#) Table 4-23, slightly revised and reproduced in Table_Apx K-5)
 - Mice exposed to methylene chloride via inhalation:
 - exhibited chromosomal aberrations, DNA SSBs and sister chromatid exchange in liver and lung cells at 2,000 ppm or higher (multiple studies).
 - exhibited DNA-protein cross links in hepatocytes but not in lung cells from 500 to 4,000 ppm for 3 days.
 - exhibited micronuclei in peripheral red blood cells at 2,000 ppm for 12 weeks and 4,000 and 8,000 ppm for 2 weeks.
 - exhibited sister chromatid exchange in peripheral lymphocytes at 8,000 ppm for 2 weeks.
 - Mice exposed to methylene chloride via gavage (single dose of 1,720 mg/kg-bw/day) exhibited DNA damage via the comet assay in liver and lung cells but not stomach, urinary bladder, kidney, brain or bone marrow cells.
 - Mice exposed to methylene chloride at a single 5 mg/kg intraperitoneal dose exhibited no DNA adducts in liver or kidney cells.
 - Chromosomal micronuclei, chromosomal aberrations or sister chromatid exchange were not consistently positive in bone marrow of mice after oral or parenteral exposure; however, GST-activity is minimal in bone marrow and Crebelli et al. (1999) indicates that halogenated hydrocarbons are not very effective in inducing micronucleus formation in mouse bone marrow. Thus, negative findings in bone marrow should not negate positive in vitro findings ([Crebelli et al., 1999](#)).

- The *H-ras* oncogene mutation profile did not differ significantly among spontaneously or methylene chloride induced liver tumors in mice. Other studies of tumor oncogenes and tumor suppressors were not clearly conclusive.
- Unscheduled DNA synthesis was not induced in mice hepatocytes after inhalation of 2,000 or 4,000 ppm methylene chloride for 2 or 6 hrs.
- *In vivo* assays in rats and hamsters ([U.S. EPA \(2011\)](#) Table 4-24, slightly revised and reproduced in Table_Apx K-6)
 - Unlike mice, rats exposed via inhalation did not exhibit DNA SSBs in liver and lung cell homogenates or hepatocytes at 2,000 ppm or higher.
 - Similar to mice, unscheduled DNA synthesis was not induced in rat hepatocytes after inhalation.
 - Similar to mice, rats exposed to methylene chloride at a single 5 mg/kg intraperitoneal dose exhibited no DNA adducts in liver or kidney cells.
 - Rats exhibited DNA SSBs in a liver homogenate via gavage dose of 1,275 mg/kg but not 425 mg/kg methylene chloride.
 - In rats, unscheduled DNA synthesis was not induced after intraperitoneal administration of 400 mg/kg or gavage administration up to 1,000 mg/kg.
 - Unlike mice, hamsters exposed to $\leq 4,000$ ppm methylene chloride via inhalation for 3 days did not exhibit DNA-protein cross links in liver or lung cells
- Comparison of *in vivo* assays targeting lung or liver cells ([U.S. EPA \(2011\)](#) Table 4-25 and reproduced in Table_Apx K-7)
 - This table lists similar studies on the same row if they use different species (mice, rats, hamster) but comparable methods.
 - The table lists studies in separate rows if there are no comparable studies in a second species.
 - All studies described in this table were presented in previous tables.

Table Apx K-2 Results from *in vitro* Genotoxicity Assays of Dichloromethane in Nonmammalian Systems

Endpoint	Test System	Dose/Concentration and Duration	Results ^a		Comments	Reference	Data Quality Evaluation
			-S9	+S9			
Reverse mutation	<i>Salmonella typhimurium</i> TA98, TA100	48-hr exposure to 0, 5,700, 11,400, 17,100, 22,800, and 57,000 ppm	+ (DR)	++ ^b (DR)	Vapor phase exposure in enclosed 37°C system. Toxic at highest dose only.	Jongen et al. (1978)	High
Reverse mutation	<i>S. typhimurium</i> TA100	6-hr exposure to 0, 3,500, 7,000, and 14,000 ppm	+ (DR)	++ ^d (DR)	Vapor phase exposure in enclosed 37°C system.	Jongen et al. (1982)	High
Reverse mutation	<i>S. typhimurium</i> TA100	3-day exposure, up to 84,000 ppm	+	+ ^e	Vapor phase exposure in sealed jars. Peak response at 12 h. Exogenous GST or GSH had no effect.	Green (1983)	Medium
Reverse mutation	<i>S. typhimurium</i> TA100, TA98	24-hr exposure to 0, 0.01, 0.05, 0.1, 0.25, 0.5, and 1.0 mL/chamber	+ (DR)	++ ^f (DR)	Vapor phase exposure in sealed desiccator jars required for positive result. Toxicity at highest dose only.	Zeiger (1990)	High
Reverse mutation	<i>S. typhimurium</i> TA100 <i>S. typhimurium</i> TA100, NG54 <i>E. coli</i> WP2 uvrA pKM101	2- and 6-hr exposures to 0, 2,500, 5,000, 7,500, 10,000 ppm; 6- and 48-hr exposures up to 50,000 ppm 6-hr exposure to 0, 2,500, 5,000, 7,500, 10,000, 20,000, 40,000 ppm 6- and 48-hr exposures to 6,300, 12,500, 25,000, and 50,000 ppm	+ (DR) + (DR) + (DR)	+ ^g (DR) + (DR) + (DR)	Vapor phase exposure in sealed jars. NG54=TA100 with 4-fold lower GSH levels. Exogenous GSH slightly increased mutation frequency. Peak response at 6 h.	Dillon et al. (1992)	High
Reverse mutation	<i>S. typhimurium</i> TA1535 (+GST5-5) TA1535	0–2.0 mM/plate	+ (DR) –	ND ND	5 min preincubation. Transfected with rat GST5-5. Negative with exogenous S-(1-acetoxymethyl)GSH or HCHO. Parental strain negative with exogenous GSH or GST.	Thier et al. (1993)	Medium
Reverse mutation	<i>S. typhimurium</i> TA100 NG-11	3-day exposure, up to 100,000 ppm	++ (DR) + (DR)	ND ND	Vapor phase exposure in sealed jars. NG-11=TA100 without GSH; adding GSH increased mutagenicity of NG-11. Toxic at highest dose.	Graves et al. (1994a)	High
Reverse mutation	<i>S. typhimurium</i> TA1535 (+GST5-5) TA1535	0, 200, 400, 800, and 1600 ppm (0, 0.03, 0.06, 0.13, and 0.26 mM in medium)	+ (DR) – (T)	ND ND	Plate incorporation assay; 24 h exposure in sealed Tedlar bags. Transfected with rat GST5-5. Toxic at highest dose.	Pegram et al. (1997)	High

Endpoint	Test System	Dose/Concentration and Duration	Results ^a		Comments	Reference	Data Quality Evaluation
			-S9	+S9			
Forward mutation	<i>S. typhimurium</i> TA100, RSJ100	Up to 24,000 ppm	+	ND	Plate incorporation assay; 24 h exposure in sealed Tedlar bags. RSJ100=TA1535+transfected rat GSTT1-1; TPT100= nonfunctional GSTT1-1 gene. Toxic at highest dose.	Demarini et al. (1997)	High
	TA1535, TPT100		-(T)	ND			
Forward mutation	<i>S. typhimurium</i> BA13	0, 8, 20, 40, and 85 µmol/plate	+++	+ ^c	Preincubation assay for L-arabinose resistance (AraR test). Toxic ≥85 µmol.	Roldán-Arjona and Pueyo (1993)	High
Forward mutation	<i>E. coli</i> K12 (wild type)	2-hr exposures to 0, 30, 60, and 130 mM/plate (aqueous concentrations)	-	+ ^h	Vapor phase exposure in sealed jars. “+” with mouse liver S9 only, not rat. No cell death in these strains and doses.	Graves et al. (1994a)	High
	<i>E. coli</i> UvrA		-	-			
Fungi and yeasts							
Mitotic segregation	<i>Aspergillus nidulans</i> -diploid strain P1	0, 800, 2,000, 4,000, 6,000, and 8,000 ppm	+(T)	ND	Positive only at 4,000 ppm.	Crebelli et al. (1988)	High
Gene conversion	<i>Saccharomyces cerevisiae</i> -strain D7	0, 104, 157, and 209 mM	+(T)	ND	Total cell death at 209 mM. Positive at 157 mM only with 58% cell death.	Callen et al. (1980)	High
Mitotic recombination			+(T)	ND			
Reverse mutation			+(T) (DR)	ND	Positive dose-response at 104 and 157 mM.		

^a+ = positive, - = negative, (T) = toxicity, ND = not determined, DR = dose-response observed.

^bS9 liver fraction isolated from male Wistar rats induced with phenobarbital.

^cS9 liver fraction isolated from rats induced with Aroclor 1254.

^dS9 liver fraction isolated from male Wistar rats induced with Aroclor 1254 and phenobarbital and separated into microsomal and cytosolic fractions.

^eS9 liver fraction isolated from male Sprague-Dawley rats induced with Aroclor 1254 and separated into microsomal and cytosolic fractions.

^fS9 liver fraction isolated from male Sprague-Dawley rats induced with Aroclor 1254.

^gS9 liver fraction isolated from male Fischer F344 rats induced with Aroclor and separated into microsomal and cytosolic fractions.

^hS9 liver fractions isolated from male B6C3F1 mice or male Alpk:APfSD (AP) rats.

Source: U.S. EPA ([2011](#)), Table 4-20, pp. 104-106

Table Apx K-3 Results from *in vitro* Genotoxicity Assays of Dichloromethane with Mammalian Systems, by Type of Test

Assay	Test System	Concentrations	Results	Reference	Data Quality Evaluation
Human					
Micronucleus test	Human AHH-1, MCL-5, h2E1 cell lines	Up to 10 mM	Positive in MCL-5, h2E1 cell lines, increasing with increasing concentrations from 2 to 10 mM	Doherty et al. (1996)	High
DNA damage by comet assay	Primary human lung epithelial cells	10, 100, 1,000 µM	Weak trend, independent of GST activity (GST enzymatic activity not present in the cultured cells)	Landi et al. (2003)	Medium
DNA SSBs by alkaline elution	Human hepatocytes	5–120 mM	Negative. Cytotoxicity >90 mM as measured by Trypan blue exclusion assay.	Graves et al. (1995)	High
Sister chromatid exchange	Primary human peripheral blood mononuclear cells	0, 15, 30, 60, 125, 250, 500 ppm	Sister chromatid exchanges significantly increased at exposures of 60 ppm and higher, most strongly in the high GST-T1 activity group; Mitotic indices decreased in a dose-dependent manner; changes in cell proliferation kinetics	Olvera-Bello et al. (2010)	High
DNA-protein cross-links	Human hepatocytes	0.5–5 mM	Negative	Casanova et al. (1997)	High
Mouse					
DNA breaks by alkaline elution	Mouse hepatocytes (B6C3F1)	0, 0.4, 3.0, 5.5 mM	Positive with dose-response. No toxicity at these doses as measured by trypan blue exclusion assay.	Graves et al. (1994b)	High
DNA SSBs by alkaline elution	Mouse Clara cells (B6C3F1)	0, 5, 10, 30, 60 mM	Positive with dose-response; DNA damage reduced by addition of GSH depletor. No toxicity at these doses as measured by trypan blue exclusion assay.	Graves et al. (1995)	High
DNA-protein cross-links	Mouse hepatocytes (B6C3F1)	0.5–5 mM	Positive	Casanova et al. (1997)	High
Rat					
DNA SSBs by alkaline elution	Rat hepatocytes (Alpk:APfSD [AP])	0, 30, 60, 90 mM	Positive with dose-response. Cytotoxicity at 90 mM as measured by trypan blue exclusion assay.	Graves et al. (1994b)	High
DNA-protein cross-links	Rat hepatocytes (Fischer-344)	0.5–5 mM	Negative	Casanova et al. (1997)	High
Hamster with GST activity from mouse					
<i>hprt</i> mutation analysis	CHO cells	3,000 and 5,000 ppm	Positive with mouse liver cytosol	Graves and Green (1996)	High

Assay	Test System	Concentrations	Results	Reference	Data Quality Evaluation
<i>hprt</i> mutation analysis	CHO cells	2,500 ppm ^a	Mutation spectrum supports role of glutathione conjugate	Graves et al. (1996)	High
DNA SSBs and DNA-protein cross-links	CHO cells	3,000 and 5,000 ppm	Positive at concentration of 0.5% (v/v) for SSBs in presence of mouse liver cytosol, but increase in DNA-protein cross-links marginal; formaldehyde (in absence of mouse liver cytosol) was positive at 0.5 mM for both DNA SSBs and DNA-protein cross-links; CHO cell cultures were suspended	Graves and Green (1996)	High
Comet assay	Chinese hamster V79 lung fibroblast cells transfected with mouse GST-T1	2.5, 5, 10 mM	A significant, dose-dependent increase in DNA damage resulting from DNA-protein cross-links in V79 cells transfected with mouse GST-T1 compared to parental cells	Hu et al. (2006)	High
DNA-protein cross-links	CHO cells (K1)	60 mM	Positive only with mouse liver S9 added; formaldehyde positive at lower concentrations (0.5–4 mM)	Graves et al. (1994b)	High
Hamster without GST activity from mouse					
Chromosomal aberrations	CHO cells	2 – 15 µl/ml	Positive, independent of rat liver S9	Thilagar and Kumaroo (1983)	High
Forward mutation (<i>hprt</i> locus)	Chinese hamster epithelial cells	10,000, 20,000, 30,000, 40,000 ppm	Negative, without metabolic activation (Experiment was not run with metabolic activation)	Jongen et al. (1981)	Medium
DNA SSBs by alkaline elution	Syrian golden hamster hepatocytes	0.4–90 mM	Negative. Cytotoxicity at 90 mM as measured by Trypan blue exclusion assay.	Graves et al. (1995)	High
Sister chromatid exchange	Chinese hamster V79 cells	10,000, 20,000, 30,000, 40,000 ppm	Weak positive with or without rat-liver microsomal system	Jongen et al. (1981)	High
Sister chromatid exchange	CHO cells	2 – 15 µl/ml	Negative with or without rat liver S9	Thilagar and Kumaroo (1983)	High
DNA-protein cross-links	Syrian golden hamster hepatocytes	0.5–5 mM	Negative	Casanova et al. (1997)	High
Calf					
DNA adducts	Calf thymus DNA	50 mM	Positive in the presence of bacterial GST DM11 and dichloromethane dehalogenase; adducts primarily formed with the guanine residues	Kayser and Vuilleumier (2001)	High

Assay	Test System	Concentrations	Results	Reference	Data Quality Evaluation
DNA adducts	Calf thymus DNA	Up to 60 mM	Positive in the presence of bacterial GST DM11, rat GST5-5, and human GSTT11; adducts primarily formed with the guanine residues	Marsch et al. (2004)	High

CHO = Chinese hamster ovary; *hprt* = hypoxanthine-guanine phosphoribosyl transferase

^aMethods section described concentration as 3,000 ppm (0.3%v/v) but Table I describes it as 2,500 ppm (0.25% v/v).

Source: U.S. EPA ([2011](#)), Table 4-21, pp. 108-110

Table Apx K-4 Results from *in vivo* Genotoxicity Assays of Dichloromethane in Insects

Assay	Test System	Doses	Result	Reference	Data Quality Evaluation
Gene mutation (sex-linked recessive lethal)	Drosophila	125, 620 mM	Positive (feeding exposure)	Gocke et al. (1981)	High
Gene mutation (sex-linked recessive lethal, somatic mutation and recombination)	Drosophila	6 hrs—1,850, 5,500 ppm 1 wk—2,360, 4,660 ppm 2 wks—1,370, 2,360 ppm (all approximate)	Negative (inhalation exposure)	Kramers et al. (1991)	High
Somatic w/w+ assay	Drosophila	50, 100, 250, 500 mM	Positive (feeding exposure)	Rodriguez-Arnaiz (1998)	Medium

Source: U.S. EPA ([2011](#)), Table 4-22, p. 114

Table Apx K-5 Results from *in vivo* Genotoxicity Assays of Dichloromethane in Mice

Assay	Test System	Route and Dose	Duration	Results	Reference	Data Quality Evaluation
Kras and Hras oncogenes	Mouse liver and lung tumors (B6C3F1)	0, 2,000 ppm	Up to 104 wks	No difference in mutation profile between control and dichloromethane-induced liver tumors; number of spontaneous lung tumors (n = 7) limits comparison at this site	Devereux et al. (1993)	High
p53 tumor suppressor gene	Mouse liver and lung tumors (B6C3F1)	0, 2,000 ppm	Up to 104 wks	Loss of heterozygosity infrequently seen in liver tumors from exposed or controls; number of spontaneous lung tumors (n = 7) limits comparison at this site	Hegi et al. (1993)	High
Micronucleus test	Mouse bone marrow (C57BL/6J/A1pk)	Gavage, 1,250, 2,500, and 4,000 mg/kg	Single dose	Negative at all doses	Sheldon et al. (1987)	High
Micronucleus test	Mouse peripheral red blood cells (B6C3F1)	Inhalation 6 hr/d, 5 d/wk, 0, 4,000, 8,000 ppm	2 wk	Positive at 4,000 and 8,000 ppm	Allen et al. (1990)	High
Micronucleus test	Mouse peripheral red blood cells (B6C3F1)	Inhalation, 6 hr/d, 5 d/wk, 0, 2,000 ppm	12 wks	Positive at 2,000 ppm	Allen et al. (1990)	High
Chromosome aberrations	Mouse bone marrow (C57BL/6J)	Intraperitoneal, 100, 1,000, 1,500, 2,000 mg/kg	Single dose	Negative	Westbrook-Collins et al. (1990)	High
Chromosome aberrations	Mouse bone marrow (B6C3F1)	Subcutaneous, 0, 2,500, 5,000 mg/kg	Single dose	Negative	Allen et al. (1990)	High
Chromosome aberrations	Mouse lung and bone marrow cells (B6C3F1)	Inhalation, 6 hr/d, 5 d/wk, 0, 4,000, 8,000 ppm	2 wks	Increase beginning at 4,000 ppm in lung cells; increase only at 8,000 ppm in bone marrow cells	Allen et al. (1990)	High
DNA SSBs by alkaline elution	Mouse hepatocytes (B6C3F1)	Inhalation, 2,000 and 4,000 ppm	3 or 6 hrs	Positive at 4,000 ppm at 3 and 6 hrs	Graves et al. (1994b)	Medium
DNA SSBs by alkaline elution	Mouse liver and lung homogenate (B6C3F1)	Liver: inhalation, 2,000, 4,000, 6,000, 8,000 ppm Lung: inhalation, 1,000, 2,000, 4,000, 6,000 ppm	3 hrs 3 hrs	Liver: positive at 4,000–8,000 ppm Lung: positive at 2,000–4,000 ppm	Graves et al. (1995)	High
DNA damage by comet assay	Mouse stomach, urinary bladder, kidney, brain, bone marrow (CD-1)	Gavage, 1,720 mg/kg; organs harvested at 0 (control), 3, and 24 hrs	Single dose	Negative 3 or 24 hr after dosing	Sasaki et al. (1998a)	High

Assay	Test System	Route and Dose	Duration	Results	Reference	Data Quality Evaluation
DNA damage by comet assay	Mouse liver and lung cells (CD-1)	Gavage, 1,720 mg/kg; organs harvested at 0 (control), 3, and 24 hrs	Single dose	Positive only at 24 hrs after dosing	Sasaki et al. (1998a)	High
DNA adducts	Mouse liver and kidney cells (B6C3F1)	Intraperitoneal, 5 mg/kg	Single dose	Negative	Watanabe et al. (2007)	Medium
DNA-protein cross-links	Mouse liver and lung cells (B6C3F1)	Inhalation, 6 hr/d, 3 d, 4,000 ppm	3 d	Positive in mouse liver cells at 4,000 ppm; negative in mouse lung cells	Casanova et al. (1992)	High
DNA-protein cross-links	Mouse liver and lung cells (B6C3F1)	Inhalation, 6 hr/d, 150, 500, 1,500, 3,000, 4,000 ppm	3 d	Positive in mouse liver cells at 500–4,000 ppm; negative in mouse lung cells	Casanova et al. (1996)	High
Sister chromatid exchange	Mouse bone marrow (C57BL/6J)	Intraperitoneal, 100, 1,000, 1,500, 2,000 mg/kg	Single dose	Negative	Westbrook-Collins et al. (1990)	High
Sister chromatid exchange	Mouse bone marrow (B6C3F1)	Subcutaneous, 0, 2,500, 5,000 mg/kg	Single dose	Negative at all doses	Allen et al. (1990)	High
Sister chromatid exchange	Mouse lung cells and peripheral lymphocytes (B6C3F1)	Inhalation 6 hr/d, 5 d/wk, 0, 4,000, 8,000 ppm	2 wks	Positive at 4,000 and 8,000 ppm for mouse lung cells and at 8,000 ppm for peripheral lymphocytes	Allen et al. (1990)	High
Sister chromatid exchange	Mouse lung cells (B6C3F1)	Inhalation 6 hr/d, 5 d/wk, 0, 2,000 ppm	12 wks	Positive at 2,000 ppm	Allen et al. (1990)	High
DNA synthesis	Mouse liver (B6C3F1)	Gavage, 1,000 mg/kg; inhalation, 4,000 ppm	Single dose; 2 hrs	Negative in both oral and inhalation studies	Lefevre and Ashby (1989)	High
Unscheduled DNA synthesis	Mouse hepatocytes (B6C3F1)	Inhalation, 2,000 and 4,000 ppm.	2 or 6 hrs	Negative	Trueman and Ashby (1987)	Medium

Source: U.S. EPA ([2011](#)), Table 4-23, pp. 115-116

Table Apx K-6 Results from *in vivo* Genotoxicity Assays of Dichloromethane in Rats and Hamsters

Assay	Test System	Route and Dose	Duration	Results	Reference	Data Quality Evaluation
DNA SSBs by alkaline elution	Rat hepatocytes	Inhalation, 3 or 6 hrs, 2,000 and 4,000 ppm	3 or 6 hrs	Negative at all concentrations and time points	Graves et al. (1994b)	Medium
DNA SSBs by alkaline elution	Rat liver homogenate	Gavage, 2 doses, 425 mg/kg and 1,275 mg/kg, administered 4 and 21 hrs before liver harvesting	4 or 21 hrs (time between dosing and liver harvesting)	Positive at 1,275 mg/kg	Kitchin and Brown (1989)	High
DNA SSBs by alkaline elution	Rat liver and lung homogenate	Liver: inhalation, 4,000, 5,000 ppm Lung: inhalation, 4,000 ppm	3 hrs 3 hrs	Negative for both liver and lung at all concentrations	Graves et al. (1995)	High
DNA adducts	Rat liver and kidney cells	Intraperitoneal, 5 mg/kg	Single dose	Negative	Watanabe et al. (2007)	Medium
DNA-protein cross-links	Hamster liver and lung cells	Inhalation, 6 hr/d, 500, 1,500, 4,000 ppm	3 d	Negative at all concentrations	Casanova et al. (1996)	High
Unscheduled DNA synthesis	Rat hepatocytes	Gavage, 100, 500, 1,000 mg/kg	Liver harvested 4 and 12 hrs after dosing	Negative 4 or 12 hrs after dosing	Trueman and Ashby (1987)	Medium
Unscheduled DNA synthesis	Rat hepatocytes	Inhalation, 2 or 6 hrs, 2,000 and 4,000 ppm	2 or 6 hrs	Negative at both concentrations and exposure durations	Trueman and Ashby (1987)	Medium
Unscheduled DNA synthesis	Rat hepatocytes	Intraperitoneal, single dose, 400 mg/kg	Single dose	Negative 48 hrs after dosing	Mirsalis et al. (1989)	High

Source: U.S. EPA ([2011](#)), Table 4-24, p. 120

Table Apx K-7 Comparison of *in vivo* Dichloromethane Genotoxicity Assays Targeted to Lung or Liver Cells, by Species

Assay	Studies in B6C3F ₁ Mice				Data Quality Evaluation	Studies in Rats				Data Quality Evaluation
	Test System	Route, Dose (Duration)	Results	Reference		Test System	Route, Dose (Duration)	Results	Reference	
Chromosome aberrations	Lung cells	Inhalation, 6 hr/d, 5 d/wk, 0, 4,000, 8,000 ppm (2 wks)	Positive at 8,000 ppm	Allen et al. (1990)	High				No studies	N/A
DNA SSBs by alkaline elution	Hepatocytes	Inhalation, 2,000 and 4,000 ppm (3 or 6 hrs)	Positive at 4,000 ppm	Graves et al. (1994b)	Medium	Hepatocytes	Inhalation, 3 or 6 hrs, 2,000 and 4,000 ppm	Negative at all concentrations and time points	Graves et al. (1994b)	Medium
DNA SSBs by alkaline elution	Liver and lung homogenate	Liver: inhalation, 2,000, 4,000, 6,000, 8,000 ppm (3 hrs) Lung: inhalation, 1,000, 2,000, 4,000, 6,000 ppm (3 hrs)	Liver: Positive at 4,000–8,000 ppm Lung: Positive at 2,000–4,000 ppm	Graves et al. (1995)	High	Liver and lung homogenate	Liver: inhalation, 4,000, 5,000 ppm Lung: inhalation, 4,000 ppm	Negative in liver and lung at all concentrations and time points	Graves et al. (1995)	High
DNA SSBs by alkaline elution			No studies	N/A		Liver homogenate	Gavage, 425 mg/kg and 1,275 mg/kg	Positive at 1,275 mg/kg	Kitchin and Brown (1989)	High
DNA damage by comet assay	Liver and lung cells	Gavage, 1,720 mg/kg; organs harvested at 0 (control), 3, and 24 hrs	Positive only at 24 hrs after dosing	Sasaki et al. (1998a)	High				No studies	N/A
DNA-protein cross-links	Liver and lung cells	Inhalation, 6 hr/d, 3 d, 4,000 ppm (3 d) Inhalation, 6 hr/d, 150, 500, 1,500, 3,000, 4,000 ppm (3 d)	Positive in liver 4,000 ppm Positive in liver at 500–	Casanova et al. (1992)	High				No studies	N/A

Assay	Studies in B6C3F ₁ Mice				Data Quality Evaluation	Studies in Rats				Data Quality Evaluation
	Test System	Route, Dose (Duration)	Results	Reference		Test System	Route, Dose (Duration)	Results	Reference	
			4,000 ppm; both studies negative in lung							
DNA adducts	Liver and kidney cells	Intraperitoneal, 5 mg/kg	Negative	Watanabe et al. (2007)	Medium	Liver and kidney cells	Intraperitoneal, 5 mg/kg	Negative	Watanabe et al. (2007)	Medium
Sister chromatid exchange	Lung cells	Inhalation 6 hr/d, 5 d/wk, 0, 4,000, 8,000 ppm (2 wks) Inhalation 6 hr/d, 5 d/wk, 0, 2,000 ppm (12 wks)	Positive at 8,000 ppm Positive at 2,000 ppm	Allen et al. (1990)	High				No studies	N/A
DNA synthesis	Liver	Gavage, 1,000 mg/kg; inhalation, 4,000 ppm (2 hrs)	Negative in oral and inhalation studies	Lefevre and Ashby (1989)	High				No studies	N/A
Unscheduled DNA synthesis	Hepatocytes	Inhalation, 2,000 and 4,000 ppm (2 or 6 hrs)	Negative	Trueman and Ashby (1987)	Medium	Hepatocytes	Inhalation, 2,000 and 4,000 ppm (2 or 6 hrs)	Negative	Trueman and Ashby (1987)	Medium
Unscheduled DNA synthesis				No studies	N/A	Hepatocytes	Intraperitoneal, 400 mg/kg	Negative	Mirsalis et al. (1989)	High

Source: [U.S. EPA \(2011\)](#), Table 4-25, pp. 121-122

Appendix L SUMMARY OF OCCUPATIONAL EXPOSURES AND RISKS FOR PAINT AND COATING REMOVERS

Use of methylene chloride for commercial paint and coating removal were assessed in the TSCA Work Plan Chemical Risk Assessment Methylene Chloride: Paint Stripping Use CASRN: 75-09-2 ([U.S. EPA, 2014](#)). This appendix summarizes the occupational exposures and risk estimates for this use. The majority of this appendix is pulled directly from the 2014 risk assessment in addition to relevant data provided to EPA as described below. This appendix provides detailed analysis of the paint and coating removal scenario and similarly detailed information on other occupational exposure scenarios is provided in the supplemental document titled "*Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) CASRN: 75-09-2, Supplemental Information on Releases and Occupational Exposure Assessment*" ([EPA, 2019b](#)).

Additional occupational exposure monitoring data for paint and coating removal have been provided by DoD ([Defense Occupational and Environmental Health Readiness System - Industrial Hygiene \(DOEHRS-IH\), 2018](#)). The raw data for DoD are summarized in Table Apx L-1. For estimating risks, samples with exactly 15 mins of sampling time were grouped for risks from acute exposure, and samples between >4 and 8 hrs were proportionately scaled to generate 8-hr TWA data for risks from chronic exposure; these acute and chronic estimates are shown in Table Apx L-2.

Table_Apx L-1. Raw Air Sampling Data for Methylene Chloride During DoD Uses in Paint and Coating Removers

Sample Duration Ranges	# of Samples	Exposure Concentrations (mg/m ³)	
		50 th Percentile	95 th Percentile
0 to 15 mins	377	28.7	285
> 15 to 30 mins	184	5.7	151
> 0.5 to 1 hr	101	16.2	230
> 1 to 4 hr	84	9.9	378
> 4 to 8 hr	11	7.7	54

Table_Apx L-2. Acute and Chronic Exposures for Methylene Chloride During DoD Uses in Paint and Coating Removers

TWA Duration	# of Samples	Exposure Concentrations (mg/m ³)	
		50 th Percentile	95 th Percentile
15-minute TWA	324	27.4	289
8-hr TWA Exposure Concentration	11	5.0	47.1
Average Daily Concentration (ADC)		1.1	10.8
Lifetime Average Daily Concentration (LADC)		2.0	24.2

Table Apx L-3 presents modeled dermal exposures during paint and coatings removal uses.

Table_Apx L-3. Summary of Dermal Exposure Doses to Methylene Chloride for Paint and Coatings Removal Uses

Occupational Exposure Scenario	Use Setting (Industrial vs. Commercial)	Maximum Weight Fraction, Y_{derm}^a	<u>Dermal Exposure Dose (mg/day) and Glove Protection Factor (PF)</u>	Calculated Fraction Absorbed, F_{abs}
Paint and Coatings Removal	Industrial	1	180 (PF = 1) 36 (PF = 5) 18 (PF = 10) 9 (PF = 20)	0.08
Paint and Coatings Removal	Commercial	1	280 (PF = 1) 57 (PF = 5) 28 (PF = 10)	0.13

a – The 2016 CDR includes a submission that reports >90% concentration during commercial and consumer use ([U.S. EPA, 2016](#)). EPA assumes up to 100% concentration, and that similar concentrations will be used for industrial paints and coatings removers.

Note on Protection Factors (PFs): All PF values are what-if type values where use of protection factors above 1 is recommended only for glove materials that have been tested for permeation against the methylene chloride-containing liquids associated with the condition of use. For scenarios with only industrial sites, EPA assumes that workers are likely to wear protective gloves and have training on the proper usage of these gloves, which assumes a protection factor of 20. For scenarios covering a broader variety of commercial and industrial sites, EPA assumes either the use of gloves with minimal to no employee training, which assumes a protection factor of 5, or the use of gloves with basic training, which assumes a protection factor of 10. If less-protective gloves are used, a protection factor of 1 may be assumed.

The remainder of this appendix is an unedited excerpt of Chapter 3 sections covering the occupational exposures (Section L.1) and risk estimates (Section 3.4) of the 2014 risk assessment. Table L-6 below summarizes the results of the exposures for the highest exposed population from the risk assessment. Section L.1 refers to appendices in the 2014 risk assessment, which may be accessed for more details ([U.S. EPA, 2014](#)).

L.1 OCCUPATIONAL EXPOSURE ASSESSMENT FOR THE USE OF DCM IN PAINT STRIPPING

Section L.1.1 summarizes the approach and methodology used for estimating occupational inhalation exposures to DCM for the use of DCM-based paint strippers. Section L.1.1.3 lists the occupational exposure estimates for the highest exposed worker population. Additional information is found in Appendices F and G [from the 2014 risk assessment ([U.S. EPA, 2014](#))].

Appendix F [from the 2014 risk assessment ([U.S. EPA, 2014](#))] describes the industries that may use DCM-based paint strippers, worker activities, processes, numbers of sites, and numbers of exposed workers. Appendix G [from the 2014 risk assessment ([U.S. EPA, 2014](#))] provides details about the air concentrations and associated worker Average Daily Concentrations (ADCs) and Lifetime Average Daily Concentrations (LADCs) presented in this section.

L.1.1 Approach and Methodology for Estimating Occupational Exposures

L.1.1.1 Identification of Relevant Industries

Because a variety of industries include paint stripping among their business activities, EPA made the effort to determine and characterize these industries, with a special interest in small commercial shops. EPA's interest in small shops for this assessment is due to the possibility that these shops may have fewer resources or less expertise and awareness of hazards, exposures, or controls as compared to large shops.

There is no standard or universal definition for the term “small shop”. The various meanings of this term can depend upon the industry sector (e.g., metal finishing, furniture repair, foam production, chemical manufacturing) or governmental jurisdiction (e.g., OSHA, EPA, other countries). For the purpose of risk assessment of work plan chemicals, EPA generally refers to entities, businesses, operators, plants, sites, facilities, or shops interchangeably and considers a number of factors to categorize these as small. The factors that have been usually considered include revenue, capacity, throughput, production, use rate of materials, or number of employees. Further characterization to determine which factors best distinguish small shops for all the various industries that perform paint stripping would require more research.

EPA reviewed the published literature and evaluated the 2007 North American Industry Classification System (NAICS) codes to determine industries that likely include paint stripping activities (see Appendix F, Table F-1) [from the 2014 risk assessment ([U.S. EPA, 2014](#))].

The following industries were identified:

- Professional contractors;
- Bathtub refinishing;
- Automotive refinishing;
- Furniture refinishing;
- Art restoration and conservation;
- Aircraft paint stripping;
- Ship paint stripping; and
- Graffiti removal

By identifying these industries, EPA identified corresponding worker subpopulations that may be exposed to DCM due to the use of these paint strippers. Appendix F [from the 2014 risk assessment ([U.S. EPA, 2014](#))] details the industries identified, processes and worker activities that may contribute to workplace exposures. Section L.1.1.2 and Appendix F [from the 2014 risk assessment] provide the estimated number of workers exposed nationwide and average numbers of employees per facility for these industries.

L.1.1.2 Estimation of Potential Workplace Exposures for Paint Stripping Facilities

Workplace exposures based on monitoring data: EPA used air concentration data and estimates found in literature sources to serve as exposure concentrations for occupational inhalation exposures to DCM. These air concentrations were used to estimate the exposure levels for workers exposed to DCM as a result of the use of DCM-based paint strippers.

EPA did not find enough monitoring data to determine complete statistical distributions of actual exposure concentrations for the exposed population of workers in each of the industries. Ideally, EPA would like to know 50th and 95th percentiles for each population, which are considered to be the most important parts of complete statistical exposure distributions. The air concentration means and midpoints (means are preferred over midpoints) served as substitutes for 50th percentiles, and high ends of ranges served as substitutes for 95th percentiles.

Data sources often did not indicate whether monitored exposure concentrations were for occupational users or bystanders. Therefore, EPA assumed that these exposure concentrations were for a combination of users and bystanders. Some bystanders may have lower exposures than users, especially when they are further away from the source of exposure.

Additionally, inhalation exposure data from OSHA and state health inspections were obtained from the OSHA's Integrated Management Information System (IMIS) database. However, OSHA IMIS data were not used to estimate workplace exposures, except where noted, because of the high degree of uncertainty and questionable relevancy of these data to stripping with DCM-containing products. Refer to Appendix G [from the 2014 risk assessment ([U.S. EPA, 2014](#))] for a detailed discussion of the OSHA IMIS data.

Workplace exposure scenarios evaluated in this assessment: Workers performing DCM-based paint stripping might or might not use a respirator and may be exposed to DCM at different exposure frequencies (days per year) or working years. Thus, EPA assessed risks from acute exposure for 4 occupational scenarios and risks from chronic exposure for 16 occupational scenarios based on 8-hr time-weighted average (TWA) exposure concentrations and different variations in exposure conditions. These scenarios were constructed within each industry evaluated in the assessment.

To estimate acute exposure, EPA defined 4 scenarios to reflect a combination of the following (Table Apx L-4):

- No use of a respirator (APF = zero);
- Use of a respirator with an APF of 10, 25, or 50, which would reduce the personal breathing concentration by 10-, 25- or 50-fold (i.e., 0.1, 0.04, 0.02), respectively.

Table ApxL-4. Acute Occupational Exposure Scenarios for the Use of DCM-Based Paint Strippers			
Acute Scenario	Respirator APF^a	8-hr TWA Concentration Multiplier^b	Scenario Description
1	0	1	No respirator
2	10	0.1	Respirator APF 10
3	25	0.04	Respirator APF 25
4	50	0.02	Respirator APF 50

Notes:
^a APF= assigned protection factor. APFs of 10, 25 or 50 mean that the respirator reduced the personal breathing concentration by 10-, 25- or 50-fold (i.e., 0.1, 0.04, 0.02).
^b As indicated in equation 3-2, these multipliers are applied to the 8-hr time-weighted average (TWA) acute exposure concentrations.

To estimate chronic exposure, EPA defined 16 scenarios to reflect a combination of the following (Table Apx L-5):

- No use of a respirator (APF = zero)³⁴;
- Use of a respirator with an APF of 10, 25, or 50;
- An exposure frequency (EF) of the assumed Scenario 1 value of 250 days per year or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 125 days per year); and
- Exposed working years (WY) of the assumed Scenario 1 value of 40 years or half of the assumed Scenario 1 value (the midpoint between the assumed Scenario 1 value and zero: 20 years).

The multipliers in Tables_Apx L-4 and L-5 were used to adjust the exposure estimates of acute and chronic Scenario 1, respectively, to obtain the exposure estimates for the other exposure scenarios. Additional information is presented below about the estimation approach to calculate the acute and chronic exposure estimates.

³⁴ APF assumptions are the same for both acute and chronic scenarios.

Table_Apx L-5. Chronic Occupational Exposure Scenarios for the Use of DCM-Based Paint Strippers					
Chronic Scenario	Respirator APF ^a	Exposure Frequency (EF) (days/yr)	Working Years (WY) (years)	ADC/LAD C Multiplier ^b	Scenario Description
1	0	250	40	1	No respirator, high ends of ranges for EF and WY
2	10	250	40	0.1	Respirator APF 10, high ends of ranges for EF and WY
3	25	250	40	0.04	Respirator APF 25, high ends of ranges for EF and WY
4	50	250	40	0.02	Respirator APF 50, high ends of ranges for EF and WY
5 / 9	0	250/ 125	20/ 40	0.5	No respirator, one midpoint and one high end of range for EF and WY
6 / 10	10	250/ 125	20/ 40	0.05	Respirator APF 10, one midpoint and one high end of range for EF and WY
7 / 11	25	250/ 125	20/ 40	0.02	Respirator APF 25, one midpoint and one high end of range for EF and WY
8 / 12	50	250/ 125	20/ 40	0.01	Respirator APF 50, one midpoint and one high end of range for EF and WY
13	0	125	20	0.25	No respirator, midpoints of ranges for EF and WY
14	10	125	20	0.025	Respirator APF 10, midpoints of ranges for EF and WY
15	25	125	20	0.01	Respirator APF 25, midpoints of ranges for EF and WY
16	50	125	20	0.005	Respirator APF 50, midpoints of ranges for EF and WY
Notes:					
^a APF= assigned protection factor. APFs of 10, 25 or 50 mean that the respirator reduced the personal breathing concentration by 10-, 25- or 50-fold, respectively.					
^b As indicated in equation 3-4, these multipliers are applied to the chronic average daily concentrations (ADCs) and lifetime average daily concentrations (LADCs).					

EPA evaluated scenarios both with and without respirator use and a range of respirator APFs because no data were found about the overall prevalence of the use of respirators to reduce DCM exposures and it was not possible to estimate the numbers of workers who have reduced exposures due to the use of respirators (as described by the data and information sources presented in Appendices F and G [from the 2014 risk assessment ([U.S. EPA, 2014](#))]).

Likewise, EPA made assumptions about the exposure frequencies and working years because data were not found to characterize these parameters. Thus, EPA evaluated occupational risks by developing hypothetical scenarios under varying exposure conditions (i.e., use of respirators with different respiratory protection factors, and different exposure frequencies and working years).

Approach for calculating acute and chronic workplace exposures: To facilitate the exposure calculations for the occupational scenarios, EPA first estimated the acute and chronic exposure estimates for Scenario 1 (highest exposure group). Equations are described below.

The exposure estimates for Acute Scenarios 2 to 4 and Chronic Scenarios 2 to 16 were obtained by adjusting scenario 1 (highest exposure group) with various multipliers (Tables 3-1 and 3-2 for acute and chronic, respectively). The acute multipliers reflected the numerical reduction in exposure levels when respirators were used. The chronic multipliers reflected the numerical reduction in exposure levels when respirators were used and/or other EF and WY values were used. Although 16 chronic scenarios were possible, scenarios 5 through 8 and 9 through 12 resulted in the same multiplier regardless of whether the scenario used an EF of 250 days/yr and a WY of 20 yrs, or an EF of 125 days/yr and a WY of 40 years.

Acute occupational exposure estimates

For single (acute) workplace exposure estimates, the DCM single (acute) exposure concentration was set to the 8-hr TWA air concentration in mg/m^3 reported for the various relevant industries. EPA assumed that some workers could be rotating tasks and not necessarily using DCM-based paint strippers on a daily basis. This type of exposure was characterized as acute in this assessment as the worker would clear DCM and its metabolites before the next encounter with the DCM-containing paint stripper.

Equation L-1 was used to estimate the single (acute) exposure estimates for acute scenario 1 ([EPA, 2009](#)).

(Eq. L-1)

$$EC_{\text{scenario 1}} = C$$

where:

- $EC_{\text{scenario 1}}$ = exposure concentration for a single 8-hr exposure to DCM (mg/m^3) for scenario 1
- C = contaminant concentration in air for relevant industry (central tendency, low- or high-end 8-hr TWA in mg/m^3 from Appendix G, Table G-2 or G-5 [from the 2014 risk assessment ([U.S. EPA, 2014](#))]);

Equation L-2 was used to calculate the acute exposure estimates for scenarios 2 through 4. (Eq. L-2)

$$EC_{\text{scenario } 2 \rightarrow 4} = EC_{\text{scenario } 1} \times M_{\text{acute}}$$

where:

$EC_{\text{scenario } 2 \rightarrow 4}$	=	exposure concentration for a single 8-hr exposure to DCM (mg/m^3) for acute scenarios 2, 3, or 4;
$EC_{\text{scenario } 1}$	=	single (acute) exposure concentration for relevant industry (8-hr TWA in mg/m^3 from Appendix G, Table G-2 or G-5 [from the 2014 risk assessment (U.S. EPA, 2014)]);
M_{acute}	=	Scenario-specific acute exposure multiplier (unit less) for relevant industry (see Table 3-1)

Acute exposure estimates for scenario 1 are presented in Table 3-3. Acute exposure estimates for scenarios 2 through 4 were integrated into the risk calculations by applying the scenario-specific multipliers. Thus, separate tables listing the acute exposure estimates for scenarios 2 through 4 are not provided in this section but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates_081114.xlsx*).

Chronic occupational exposure estimates

The worker exposure estimates for the non-cancer and cancer risk calculations were estimated as ADCs and LADCs, respectively. Both ADC and LADC calculations for Scenario 1 were based on the 8-hr TWA air concentration in mg/m^3 reported for the various relevant industries (Appendix G, Table G-5 [from the 2014 risk assessment ([U.S. EPA, 2014](#))]). EPA assumed that the worker would be doing paint stripping activities during the entire 8-hr work shift on a daily basis. Equation 3-3 was used to estimate the chronic ADCs and LADCs for Scenario 1 ([EPA, 2009](#)).

(Eq. L-3)

$$EC_{\text{scenario } 1} = \frac{C \times ED \times EF \times WY}{AT}$$

where:

$EC_{\text{scenario } 1}$	=	exposure concentration (mg/m^3) for Scenario 1 = ADC for chronic non-cancer risks or LADC for chronic cancer risks for Scenario 1;
C	=	contaminant concentration in air for relevant industry (central tendency, low- or high-end 8-hr TWA in mg/m^3 from Appendix G, Table G-2 [from the 2014 risk assessment (U.S. EPA, 2014)]);
ED	=	exposure duration (hrs/day) = 8 hrs/day;
EF	=	exposure frequency (days/yr) = 250 days/yr for high-end of range for both ADC and LADC calculations;

WY = working years per lifetime (yrs) = 40 yrs for high end of range for both ADC and LADC calculations; and
 AT = averaging time (years × 365 days/year × 24 hrs/day) = 40 yrs for high end of range for ADC calculations; 70 yrs for LADC calculations, which is used to match the years used to calculate EPA's cancer inhalation unit risk (IUR).

Equation L-4 was used to estimate the chronic ADCs and LADCs for scenarios 2 through 16. (Eq. L-4)

$$EC_{\text{scenario 2} \rightarrow 16} = EC_{\text{scenario 1}} \times M_{\text{chronic}}$$

where:

$EC_{\text{scenario 2} \rightarrow 16}$ = exposure concentration for chronic exposure concentration (ADC or LADC) to DCM (mg/m^3) for chronic scenarios 2 through 16
 $EC_{\text{scenario 1}}$ = chronic exposure concentration (ADC or LADC) for relevant industry, chronic scenario 1 (in mg/m^3 from Table 3-3);
 M_{chronic} = scenario-specific ADC/LADC chronic multiplier for relevant industry (see Table 3-2)

Non-cancer and cancer exposure estimates (i.e., ADC and LADC, respectively) for scenario 1 are presented in Table 3-3. The estimates for scenarios 2 through 16 were integrated into the risk calculations by applying the scenario-specific ADC/LADC multipliers. Thus, separate tables listing the chronic exposure estimates for scenarios 2 through 16 are not provided in this section but are available in a supplemental Excel spreadsheet documenting the risk calculations for this assessment (*DCM Exposure and Risk Estimates_081114.xlsx*).

Numbers of exposed workers and shop sizes: Knowing the sizes of exposed populations provides perspective on the prevalence of the health effects. Thus, EPA estimated the current total number of workers in the potentially exposed populations.

EPA found limited data on numbers of workers exposed to DCM in shops that use DCM-based paint strippers. EPA relied on an estimation approach to estimate the total number of exposed workers from the technical support document for the National Emission Standards for Hazardous Air Pollutants (NESHAP) Paint Stripping Operations at Area Sources proposed rule ([U.S. EPA, 2007](#)).

Based on the NESHAP data and analyses, EPA estimates that over 230,000 workers nationwide are directly exposed to DCM from DCM-based paint strippers. This estimate only accounts for workers performing the paint stripping using DCM and does not include other workers ("occupational bystanders") within the facility who are indirectly exposed. EPA cannot estimate the numbers of workers exposed in each of the individual industries that may use DCM-based strippers. EPA also cannot estimate the numbers of workers exposed in small shops. Appendix E [from the 2014 risk assessment ([U.S. EPA, 2014](#))] details the literature search, data found, and assumptions for worker population exposed nationwide.

EPA estimated the average number of employees per facility which can be a factor in determining shop sizes. These estimates were derived by combining the facility and population data obtained from the U.S. Census data, as described in Appendix F [from the 2014 risk assessment ([U.S. EPA, 2014](#))]. The average number of employees for the identified industries based on U.S. Census data were the following:

- Professional contractors (likely to include Bathtub refinishing): 5 workers/facility;
- Automotive refinishing: 6 workers/facility;
- Furniture refinishing: 3 workers/facility;
- Art restoration and conservation (not estimated);
- Aircraft paint stripping: 320 workers/facility (for aircraft manufacturing only);
- Ship paint stripping: 100 workers/facility; and
- Graffiti removal: 8 workers/facility.

These averages give some perspective on shop size but are simple generalizations.

L.1.1.3 Summary of Occupational DCM Exposure Estimates

Table_Apx L-6 shows the DCM air concentrations used in this assessment for estimating risks from acute and chronic exposures for the highest exposed worker scenario group (Scenario 1) within each industry. The statistical issues of these estimates are briefly discussed in section L.5.1.

Acute and chronic DCM exposure estimates for Acute Scenarios 2 through 4 and Chronic Scenarios 2 through 16 were integrated into the risk calculations by applying multipliers to Scenario 1. Separate tables listing the acute and chronic exposure estimates are not provided in this section but can be found in the supplemental Excel spreadsheet - *DCM Exposure and Risk Estimates_081114.xlsx*. Also, Table_Apx L-6 provides a summary of the ranges of acute, ADC and LADC estimates for the various occupational scenarios.

Table_Apx L-6. DCM Acute and Chronic Exposure Concentrations (ADCs and LADCs) for Workers – Scenario 1 – Highest Exposed Scenario Group													
Industry / Activity	Time Range of Studies	ACUTE EXPOSURE ESTIMATES Single 8-hr Concentration (mg/m³)^a				CHRONIC EXPOSURE ESTIMATES USED IN THE NON-CANCER RISK ESTIMATES ADC (mg/m³)^b				CHRONIC EXPOSURE ESTIMATES USED IN THE CANCER RISK ESTIMATES LADC (mg/m³)^b			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Professional Contractors	1981-2004	--	2,980	1,520	60	--	680	347	14	--	389	198	7.8
Bathtub Refinishing		--	--	--	--	--	--	--	--	--	--	--	--
Automotive Refinishing	2003	253	416	253	90	58	95	58	21	33	54	33	12
Furniture Refinishing	1989-2007	499	2,245 (1,266) ^c	1,125	4.0	114	513 (289) ^c	257	0.9	65	293 (165) ^c	147	0.5
Art Restoration and Conservation	2005	2.0				0.5				0.3			
Aircraft Paint Stripping	1977-2006	--	3,802	1,944	86	--	868	444	20	--	496	254	11
Ship Paint Stripping	1980	--	--	--	--	--	--	--	--	--	--	--	--
Graffiti Removal	1993	260	1,188	603	18	59	271	138	4.1	34	155	79	2.3
Non-Specific Workplace Settings - Immersion Stripping of Wood	1980-1994	--	7,000	3,518	35	--	1,598	803	8.0	--	913	459	4.6

Industry / Activity	Time Range of Studies	ACUTE EXPOSURE ESTIMATES Single 8-hr Concentration (mg/m ³) ^a				CHRONIC EXPOSURE ESTIMATES USED IN THE NON-CANCER RISK ESTIMATES ADC (mg/m ³) ^b				CHRONIC EXPOSURE ESTIMATES USED IN THE CANCER RISK ESTIMATES LADC (mg/m ³) ^b			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	1980	--	1,017	825	633	--	232	188	145	--	133	108	83
Non-Specific Workplace Settings - Immersion Stripping of Metal		--	--	--	--	--	--	--	--	--	--	--	--
Non-Specific Workplace Settings – Unknown	1997-2004	357	428	357	285	81	98	81	65	47	56	47	37
Notes: Sources are reported in Table G-2 and discussed in section G-3. ^a Calculated acute single 8-hr concentrations are only estimated from 8-hr TWA exposures; see Equation 3-1. Airborne concentration conversion factor for DCM is 3.47 mg/m ³ per ppm (Niosh, 2011b). ^b Calculated ADCs and LADCs are only calculated from 8-hr TWA exposures; see Equation 3-3. ^c The values in parentheses are the 95 th percentiles of the calculated acute single 8-hr concentrations and the calculated ADCs and LADCs. -- Indicates no data found.													

L.1.1.4 Worker Exposure Limits for DCM

Both regulatory and non-regulatory worker exposure limits have been established for DCM by OSHA, NIOSH, and the American Conference of Government Industrial Hygienists (ACGIH). EPA analysis showed that the OSHA permissible exposure limit (PEL) and Action Level values were exceeded for some industries using DCM-based strippers when the OSHA values were compared to the air concentrations.

Table_Apx L-7 provides a summary of the current occupational exposure values established by OSHA, NIOSH, and ACGIH. Appendix F [from the 2014 risk assessment ([U.S. EPA, 2014](#))] presents additional background on processes, respiratory protection, facilities and worker populations.

OSHA's amended regulatory occupational exposure limits for DCM were effective April 10, 1997. The amendments included reducing the PEL, reducing and changing the averaging time of the short-term exposure limit (STEL), adding an Action Level, and removing the ceiling limit ([OSHA, 1997a](#)). See Appendix G, section G-2-3, for more details [from the 2014 risk assessment([U.S. EPA, 2014](#))].

Source	Limit Type	Exposure Limit
OSHA PEL	PEL (8-hr TWA) ^b	25 ppm ^c
	STEL (15-minute TWA)	125 ppm
	Action Level (8-hr TWA)	12.5 ppm
NIOSH exposure limits	IDLH ^d	2,300 ppm
	Recommended Exposure Limit ^e	Ca
ACGIH TLV ^f	8-hr TWA	50 ppm

Notes:
^a Source: ([OSHA, 1997a](#))
^b PEL= Permissible exposure limit ; TWA= Time-weighted average
^c Airborne concentration conversion factor for DCM is 3.47 mg/m³ per ppm ([Niosh, 2011b](#)).
^d IDLH = Immediately dangerous to life or health. IDLH values are based on effects that might occur from a 30-minute exposure.
^e The Recommended Exposure Limit notation "Ca" is for a potential occupational carcinogen. The NIOSH Pocket Guide website has detailed policy recommendations for chemicals with "Ca" notations ([Niosh, 2011b](#)).
^f TLV = Threshold limit value

L.4 HUMAN HEALTH RISK CHARACTERIZATION

Exposure to DCM is associated with adverse effects on the nervous system, liver and lung. These non-cancer adverse effects are deemed important for acute and chronic risk estimation for the scenarios and populations addressed in this risk assessment.

DCM is likely to be carcinogenic to humans. The cancer risk assessment uses the IUR derived in the 2011 DCM IRIS assessment based on liver and lung tumors in rodents. The weight-of-evidence analysis for the cancer endpoint was sufficient to conclude that DCM-induced tumor development operates through a mutagenic mode of action ([U.S. EPA, 2011](#)).

L.4.1 Risk Estimation Approach for Acute and Repeated Exposures

Tables_Apx L-8 and L-9 show the use scenarios, populations of interest and toxicological endpoints that were used for estimating acute or chronic risks, respectively.

Use Scenarios	OCCUPATIONAL USE	RESIDENTIAL USE
Populations And Toxicological Approach		
Population of Interest and Exposure Scenario: <i>Users</i>	Adults of both sexes (>16 years old) exposed to DCM during an 8-hr workday ^{1, 2}	Adults of both sexes (>16 years old) typically exposed to DCM for 1 hr. Other shorter (10-min, 30-min) or longer exposure times (4-hr, 8-hr) were also assumed when comparing DCM air concentrations with AEGLs.
Population of Interest and Exposure Scenario: <i>Bystander</i>	Adults of both sexes (>16 years old) indirectly exposed to DCM while being in the same building during product use.	Individuals of any age indirectly exposed to DCM while being in the rest of the house during product use.

Table_Apx L-8. Use Scenarios, Populations of Interest and Toxicological Endpoints for Assessing Acute Risks to DCM-containing Paint Strippers		
Use Scenarios	OCCUPATIONAL USE	RESIDENTIAL USE
Populations And Toxicological Approach		
Health Effects of Concern, Concentration and Time Duration	<p><u>Non-Cancer Health Effects:</u> CNS effects and COHb formation in the blood (see Table 3-10).</p> <p><i>Hazard Values (PODs) for Occupational Scenarios:</i>³ 8-hr California REL POD= 290 mg/m³ 8-hr AEGL-2 POD = 210 mg/m³</p> <p><i>Hazard Values (PODs) for Residential Scenarios:</i> 1-hr SMAC POD= 350 mg/m³ 1-hr California REL POD= 840 mg/m³ 10-min AEGL-1 POD= 3,000 mg/m³ 30-min AEGL-1 POD = 2,400 mg/m³ 1-hr AEGL-1 POD = 2,130 mg/m³ 10-min AEGL-2 POD = 6,000 mg/m³ 30-min AEGL-2 POD = 4,200 mg/m³ 1-hr AEGL-2 POD = 2,000 mg/m³ 4-hr AEGL-2 POD = 350 mg/m³ 8-hr AEGL-2 POD = 210 mg/m³</p> <p><u>Cancer Health Effects:</u> Acute cancer risks were not estimated. Relationship is not known between a single short-term exposure to DCM and the induction of cancer in humans.</p>	
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	UF for SMAC PODs= 10 UF for California REL POD= 60 UF for AEGL-1 PODs= 3 UF for AEGL-2 PODs= 1	
Notes: ¹ It is assumed no substantial buildup of DCM in the body between exposure events due to DCM's short biological half-life (~40 min). ² EPA believes that the users of these products are generally adults, but younger individuals may be users of DCM-based paint strippers. ³ AEGL-1 POD for 8-hr is not available since the DCM AEGL technical support document did not derive AEGL-1 values for 8-hrs.		

Table_Apx L-9. Use Scenarios, Populations of Interest and Toxicological Endpoints for Assessing Chronic Risks to DCM-containing Paint Strippers					
Use Scenarios	OCCUPATIONAL USE				
Populations And Toxicological Approach					
Population of Interest and Exposure Scenario: <i>Users</i>	Adults of both sexes (>16 years old) exposed to DCM during an 8-hr workday for up to 250 days per year for 40 working years depending on the occupational scenario ^{1,2}				
Population of Interest and Exposure Scenario: <i>Bystander</i>	Adults of both sexes (>16 years old) indirectly exposed to DCM while being in the same building during product use. ³				
Health Effects of Concern, Concentration and Time Duration	<table border="0"> <tr> <td style="text-align: center;"><i>Hazard Value (PODs) for Non-Cancer Effects (liver effects):</i></td> <td style="text-align: center;"><i>Hazard Value (PODs) for Cancer Effects (liver and lung tumors):</i></td> </tr> <tr> <td style="text-align: center;">1st percentile human equivalent concentration (HEC) i.e., the HEC₉₉: 17.2 mg/m³ (4.8 ppm)</td> <td style="text-align: center;">Inhalation Unit Risk (IUR): 4 x 10⁻⁵ per ppm (1 x 10⁻⁵ per mg/m³)</td> </tr> </table>	<i>Hazard Value (PODs) for Non-Cancer Effects (liver effects):</i>	<i>Hazard Value (PODs) for Cancer Effects (liver and lung tumors):</i>	1 st percentile human equivalent concentration (HEC) i.e., the HEC ₉₉ : 17.2 mg/m ³ (4.8 ppm)	Inhalation Unit Risk (IUR): 4 x 10 ⁻⁵ per ppm (1 x 10 ⁻⁵ per mg/m ³)
<i>Hazard Value (PODs) for Non-Cancer Effects (liver effects):</i>	<i>Hazard Value (PODs) for Cancer Effects (liver and lung tumors):</i>				
1 st percentile human equivalent concentration (HEC) i.e., the HEC ₉₉ : 17.2 mg/m ³ (4.8 ppm)	Inhalation Unit Risk (IUR): 4 x 10 ⁻⁵ per ppm (1 x 10 ⁻⁵ per mg/m ³)				
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	UF for the HEC ₉₉ = 10 UF is not applied for the cancer risk calculations.				
Notes:					
¹ It is assumed no substantial buildup of DCM in the body between exposure events due to DCM's short biological half-life (~40 min).					
² EPA believes that the users of these products are generally adults, but younger individuals may be users of DCM-based paint strippers.					
³ Data sources did not often indicate whether exposure concentrations were for occupational users or bystanders. Therefore, EPA assumed that exposures were for a combination of users and bystanders. Some bystanders may have lower exposures than users, especially when they are further away from the source of exposure.					

Acute or chronic MOEs (MOE_{acute} or $MOE_{chronic}$) were used in this assessment to estimate non-cancer risks (Table_Apx L-10).

Table_Apx L-10. Margin of Exposure (MOE) Equation to Estimate Non-Cancer Risks Following Acute or Chronic Exposures to DCM	
$MOE_{acute\ or\ chronic} = \frac{\text{Non-cancer Hazard value (POD)}}{\text{Human Exposure}}$	
MOE = Hazard value (POD) = Human Exposure =	Margin of exposure (unitless) derived from various toxicological documents (see Tables 3-10, 3-11, 3-12) Exposure estimate (in ppm) from occupational or consumer exposure assessment. ADCs were used for non-cancer risks associated with chronic exposures to DCM. Acute concentrations as expressed as 8-hr TWA DCM air concentrations were used for acute risks.

Study-specific UFs were identified for each hazard value (i.e., POD). These UFs accounted for (1) the variation in susceptibility among the members of the human population (i.e., inter-individual or intraspecies variability); (2) the uncertainty in extrapolating animal data to humans (i.e., interspecies uncertainty); and (3) the uncertainty in extrapolating from a LOAEL rather than from a NOAEL.

The total UF for each non-cancer hazard value was the benchmark MOE used to interpret the MOE risk estimates for each use scenario. The MOE estimate was interpreted as human health risk if the MOE estimate was less than the benchmark MOE (i.e., the total UF). On the other hand, the MOE estimate indicated negligible concerns for adverse human health effects if the MOE estimate exceeded the benchmark MOE. Typically, the larger the MOE, the more unlikely it is that a non-cancer adverse effect would occur.

Cancer risks for repeated exposures to DCM were estimated using the equation in Table_Apx L-11. Estimates of cancer risks should be interpreted as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e., incremental or excess individual lifetime cancer risk).

Table_Apx L-11. Equation to Calculate Cancer Risks	
$\text{Risk} = \text{Human Exposure} \times \text{IUR}$	
Risk = Human exposure = IUR =	Cancer risk (unitless) Exposure estimate (LADC in ppm) from occupational exposure assessment Inhalation unit risk 4×10^{-5} per ppm (1×10^{-5} per mg/m^3) (U.S. EPA, 2011)

L.4.1 Acute Non-Cancer Risk Estimates for Inhalation Exposures to DCM

The acute inhalation risk assessment used CNS effects to evaluate the acute risks for consumer and occupational use of DCM-containing paint strippers. Health hazard values were derived from the SMAC and the California acute REL hazard/dose-response assessments. This assessment gives preferences to those acute risk estimates derived from the SMAC hazard/dose-response assessment because the SMAC POD was based on multiple human observations

reporting increased COHb levels after DCM exposure, coupled with the knowledge of what would be considered a NOAEL COHb level based on the extensive CO database ([Nrc, 1996](#)).

Hazard values based on the AEGL hazard/dose-response assessment were also included in the acute risk assessment. As discussed in section 3.3.1.3.3, AEGL PODs for the respective tiers (discomfort/non-disabling effects = AEGL-1 threshold; disability = AEGL-2 threshold; and death = AEGL-3 threshold) are selected to represent an estimated point of transition between one defined set of symptoms or adverse effects in one tier and another defined set of symptoms or adverse effects in the next tier ([NRC, 2001](#)). Although the AEGL PODs and total UFs do not have the degree of conservatism that other values have, EPA used them in this assessment to gauge how far the acute consumer and occupational exposure are from the thresholds for discomfort/non-disabling effects (AEGL-1) and disability (AEGL-2). These comparisons provide an indicator of whether the exposure estimates would be expected to produce human adverse effects following DCM exposure.

L.4.1.1 Acute Risks for Consumer Exposure Scenarios

Acute inhalation risks for CNS effects were reported for all of the consumer exposure scenarios when risks were evaluated with the SMAC and the California acute REL PODs and respective benchmark MOEs. These risks were reported for both the product user and the residential bystanders exposed to DCM, irrespective of the type of product used (i.e., brush-on vs. spray-on paint stripper) (Table_Apx L-12).

Consumers using DCM-based paint strippers reported risk concerns for non-disabling effects (AEGL-1) during the first hour of product use (i.e., 10-min, 30-min or 1-hr exposure). For instance, MOEs based on the AEGL-1 PODs were lower than the benchmark MOE for users using brush-on and spray-on products in those scenarios constructed with upper-end estimates for either the user or the user and bystanders (Scenarios 2, 3, 5 and 6) (Table_Apx L-13).

Likewise, risk concerns for incapacitating effects (AEGL-2) in product users were observed in Scenarios 2, 3, 5 and 6 at longer exposure times (i.e., 4-hr or 8-hrs). Interestingly, these risks were also reported for residential bystanders in Scenarios 3 and 6, where upper end user and bystander parameters were used to construct the scenarios (Table_Apx L-13).

The bathroom scenario (#7) was constructed to simulate a human fatality case during a bathtub refinishing project. It was included in the assessment to estimate the DCM air concentrations to residential occupants outside the use zone (i.e., bystanders) under conditions of high product use in the room of use. As expected, risk concerns for incapacitating effects (AEGL-2) were seen in users exposed to DCM for 4- and 8-hrs. Similarly, the users showed risks for non-disabling effects (AEGL-1) during the first hour of product use (i.e., 10-min, 30-min or 1-hr). Bystanders did not show risk concerns for non-disabling (AEGL-1) and incapacitating (AEGL-2) effects at any of the exposure durations (i.e., 10-min, 30-min, 1-hr, 4-hr or 8-hr) (Table_Apx L-13).

Table_Apx L-12. Acute Risk Estimates for Residential Exposures to DCM-Based Paint Strippers: SMAC and California's REL PODs. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text				
Exposure Scenario	Individual	Maximum Value for 1-hr Averaging Period (mg/m³)	Margin of Exposure (MOE)	
			1-hr SMAC POD Total UF or Benchmark MOE=10*Preferred Approach	1-hr California REL POD Total UF or Benchmark MOE=60
Scenario #1 Brush application in workshop, central parameter values	User	220	1.6	3.8
	Bystander	120	2.9	7.0
Scenario #2 Brush application in workshop, upper-end values for user	User	1,100	0.3	0.8
	Bystander	210	1.7	4.0
Scenario #3 Brush application in workshop, upper-end values for user and bystander estimates	User	760	0.5	1.1
	Bystander	460	0.8	1.8
Scenario #4 Spray application in workshop, central parameter values	User	490	0.7	1.7
	Bystander	280	1.3	3.0
Scenario #5 Spray application in workshop, upper-end values for user	User	1,600	0.2	0.5
	Bystander	310	1.1	2.7
Scenario #6 Spray application in workshop, upper-end values for user and bystander estimates	User	1,100	0.3	0.8
	Bystander	700	0.5	1.2
Scenario #7 Brush application in bathroom, simulation	User	799	0.4	1.1
	Bystander	218	1.6	3.9

Table_Apx L-13. Acute Risk Estimates for Residential Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text															
Scenario #1: Brush application in workshop, central parameter estimates	User	380	270	220	120	69	7.9	8.9	9.7	15.8	15.6	9.1	2.9	3.0	
	Bystander	130	130	120	82	49	23.1	18.5	17.8	46.2	32.3	16.7	4.3	4.3	
Scenario #2: Brush application in workshop, upper-end user estimates	User	1,300	1,100	1,100	420	220	2.3	2.2	1.9	4.6	3.8	1.8	0.8	1.0	
	Bystander	220	220	210	140	82	13.6	10.9	10.1	27.3	19.1	9.5	2.5	2.6	
Scenario #3: Brush application in workshop, upper-end user and bystander estimates	User	1,200	900	760	560	400	2.5	2.7	2.8	5.0	4.7	2.6	0.6	0.5	
	Bystander	470	470	460	380	290	6.4	5.1	4.6	12.8	8.9	4.3	0.9	0.7	
Scenario #4: Spray application in workshop, central parameter estimates	User	780	600	490	270	150	3.8	4.0	4.3	7.7	7.0	4.1	1.3	1.4	
	Bystander	300	300	280	190	110	10.0	8.0	7.6	20.0	14.0	7.1	1.8	1.9	
Scenario #5: Spray application in workshop, upper-end user estimates	User	1,900	1,800	1,600	620	330	1.6	1.3	1.3	3.2	2.3	1.3	0.6	0.6	
	Bystander	330	320	310	200	120	9.1	7.5	6.9	18.2	13.1	6.5	1.8	1.8	

Table_Apx L-13. Acute Risk Estimates for Residential Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text														
Scenario #6: Spray application in workshop, upper-end user and bystander estimates	User	1,600	1,300	1,100	810	580	1.9	1.8	1.9	3.8	3.2	1.8	0.4	0.4
	Bystander	710	710	700	580	430	4.2	3.4	3.0	8.5	5.9	2.9	0.6	0.5
Scenario #7: Brush application in bathroom, simulation	User	1,455	887	799	536	340	2.1	2.7	2.7	4.1	4.7	2.5	0.7	0.6
	Bystander	224	222	218	187	150	13.4	10.8	9.8	26.8	18.9	9.2	1.9	1.4

L.4.1.1 Acute Risks for Occupational Exposure Scenarios

Acute inhalation risks for CNS effects were reported for most of the relevant industries when occupational risks were evaluated with the California acute REL POD and respective benchmark MOE. These risks were irrespective of the absence or presence of respirators and were observed with central tendency or high-end DCM air concentrations (Table_Apx L-14).

Workers handling DCM-containing paint strippers with no respirator showed risks for incapacitating effects (AEGL-2) when employed in all of the relevant industries, except the art restoration and conservation industry (Table_Apx L-14). These risks were present with either central tendency or high-end DCM air concentrations of DCM.

Workers employed in industries with high exposure to DCM [i.e., professional contractors, furniture refinishing, aircraft paint stripping, and immersion stripping of wood (non-specific workplace settings)] typically showed risks for incapacitating (AEGL-2) effects when using APF 10 respirators (Scenario 2) during high exposure conditions. The use of APF 25 respirators (Scenario 3) was not protective for workers employed in the immersion stripping of wood (non-specific workplace settings) when DCM air concentrations were as high as 7,000 mg/m³.

Table_Apx L-14. Acute Risk Estimates for Occupational Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text												
Professional Contractors	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		2,980	1,520	60		0.1	0.2	5		0.07	0.1	4
Scenario 2 (Respirator, APF 10)		298	152	6		1	2	48		0.7	1.4	35
Scenario 3 (Respirator, APF 25)		119	61	2		2	5	121		1.8	4	88
Scenario 4 (Respirator, APF 50)		60	30	1		5	10	242		4	7	175
Automotive Refinishing	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	253	416	253	90	1	0.7	1	3	0.8	0.5	0.8	2
Scenario 2 (Respirator, APF 10)	25	42	25.3	9	12	7	12	32	8	5	8	23
Scenario 3 (Respirator, APF 25)	10	17	10	4	29	17	29	81	21	13	21	58
Scenario 4 (Respirator, APF 50)	5	8	5	2	57	35	57	161	42	25	42	117
Furniture Refinishing	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	499	2,245	1,125	4	0.6	0.1	0.3	73	0.4	0.1	0.2	53
Scenario 2 (Respirator, APF 10)	49.9	225	113	0.4	6	1.3	2.6	725	4	0.9	2	525
Scenario 3 (Respirator, APF 25)	20	90	45	0.2	15	3	6	1813	11	2	5	1312

Table_Apx L-14. Acute Risk Estimates for Occupational Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text												
Scenario 4 (Respirator, APF 50)	10	45	23	0.1	29	6	13	3625	21	5	9	2625
Art Restoration and Conservation	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	2				145				105			
Scenario 2 (Respirator, APF 10)	0.2				1450				1050			
Scenario 3 (Respirator, APF 25)	0.1				3625				2625			
Scenario 4 (Respirator, APF 50)	0.04				7250				5250			
Aircraft Paint Stripping	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		3,802	1,944	86		0.1	0.2	3		0.1	0.1	2
Scenario 2 (Respirator, APF 10)		380	194	9		1	1.5	34		0.6	1	24
Scenario 3 (Respirator, APF 25)		152	78	3		2	4	84		1	3	61
Scenario 4 (Respirator, APF 50)		76	39	2		4	7	167		3	5	122
Graffiti Removal	Acute 8-hr concentration (mg/m ³)				Acute MOE (8hr-REL POD=290 mg/m ³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m ³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	260	1,188	603	18	1	0.2	0.5	16	0.8	0.2	0.4	12

Table_Apx L-14. Acute Risk Estimates for Occupational Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text												
Scenario 2 (Respirator, APF 10)	26	118.8	60.3	1.8	11	2	5	161	8	2	3	117
Scenario 3 (Respirator, APF 25)	10	48	24	0.7	28	6	12	403	20	4	9	292
Scenario 4 (Respirator, APF 50)	5	24	12	0.4	56	12	24	806	40	9	17	583
Non-Specific Workplace Settings - Immersion Stripping of Wood	Acute 8-hr concentration (mg/m³)				Acute MOE (8hr-REL POD=290 mg/m³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		7,000	3,518	35		0.04	0.1	8		0.03	0.1	6
Scenario 2 (Respirator, APF 10)		700	352	4		0.4	0.8	83		0.3	0.6	60
Scenario 3 (Respirator, APF 25)		280	141	1		1	2	207		0.8	1.5	150
Scenario 4 (Respirator, APF 50)		140	70	0.7		2	4	414		2	3	300
Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	Acute 8-hr concentration (mg/m³)				Acute MOE (8hr-REL POD=290 mg/m³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)		1,017	825	633		0.3	0.4	0.5		0.2	0.3	0.3
Scenario 2 (Respirator, APF 10)		101.7	83	63		3	4	5		2	3	3
Scenario 3 (Respirator, APF 25)		41	33	25		7	9	11		5	6	8
Scenario 4 (Respirator, APF 50)		20	17	13		14	18	23		10	13	17

Table_Apx L-14. Acute Risk Estimates for Occupational Exposures to DCM-Based Paint Strippers: AEGL-1 and AEGL-2 PODs for Various Exposure Durations. MOEs below benchmark MOE indicate potential health risks and are denoted in bold text

Non-Specific Workplace Settings – Unknown	Acute 8-hr concentration (mg/m³)				Acute MOE (8hr-REL POD=290 mg/m³) Total UF or Benchmark MOE=60				Acute MOE (8hr-AEGL-2 POD=210 mg/m³) Total UF or Benchmark MOE=1			
	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Scenario 1 (No respirator, APF=0)	357	428	357	285	0.8	0.7	0.8	1	0.6	0.5	0.6	0.7
Scenario 2 (Respirator, APF 10)	36	43	36	29	8	7	8	10	6	5	6	7
Scenario 3 (Respirator, APF 25)	14	17	14	11	20	17	20	25	15	12	15	18
Scenario 4 (Respirator, APF 50)	7	9	7	6	41	34	41	51	29	25	29	37

L.4.1 Non-Cancer and Cancer Risk Estimates for Chronic Inhalation Exposures to DCM

Non-cancer and cancer risk estimates for inhalation exposures to DCM were only derived for occupational scenarios since the exposures for consumer uses were not considered chronic in nature. Hazard values were obtained from the EPA IRIS *Toxicological Review of Methylene Chloride* ([U.S. EPA, 2011](#)).

L.4.1.1 Cancer Risks for Occupational Exposure Scenarios

The cancer risk assessment evaluated the incremental individual lifetime cancer risks for continuous exposures to DCM occurring during the use of paint stripping products. Excess cancer risks were calculated by multiplying the EPA inhalation unit risk for DCM ([U.S. EPA, 2011](#)) by the exposure estimate (i.e., LADC). Cancer risks were expressed as number of cancer cases per million.

Occupational scenarios assumed that the exposure frequency (i.e., the number of days per year workers or bystanders are exposed to DCM) was either 125 or 250 days per year for an occupational exposure duration of 20 or 40 years over a 70-yr lifespan. It is recognized that the combination of these assumptions may yield conservative cancer risk estimates for some of the occupational scenarios evaluated in this assessment. Nevertheless, EPA does not have additional information for further refinement of the exposure assumptions.

EPA typically uses a benchmark cancer risk level between 1×10^{-4} and 1×10^{-6} for determining the acceptability of the cancer risk in a population. Since the benchmark cancer risk level will be determined during risk management, the occupational cancer risk estimates were compared to three benchmark levels within EPA's acceptability range. The benchmark levels were:

1. 1×10^{-6} : the probability of 1 chance in 1 million of an individual developing cancer;
2. 1×10^{-5} : the probability of 1 chance in 100,000 of an individual developing cancer, which is equivalent to 10 cancer cases in 1 million;
3. 1×10^{-4} : the probability of 1 chance in 10,000 of an individual developing cancer, which is equivalent to 100 cancer cases in 1 million.

Tables_Apx L-15 to L-23 show the excess cancer risks calculated for workers of different industries handling DCM-based paint strippers. Selected scenarios ranging from the highest exposure scenario (i.e., no respiratory protection and high end values for EF and WY—i.e., Scenario 1) to the lowest exposure scenario (e.g., respiratory protection APF 50 and midpoints for EF and WY—Scenario 16) were included in the tables. Calculations of cancer risks for the full set of industries and scenarios are provided in the supplemental Excel spreadsheet, *DCM Exposure and Risk Estimates_081114.xlsx*.

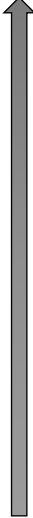
Workers showed excess cancer risks for all of the industries evaluated when working with DCM-based paint strippers for 250 days/year for 40 years with no respiratory protection (Scenario 1). Generally, Scenario 1 exceeded the three target cancer levels with the exception of art restoration and conservation that only exceeded the 1×10^{-6} target level.

On the other hand, workers showed a reduction in cancer risks when working for 125 days/year for 20 years with adequate respiratory protection (Scenario 16). That reduction in excess cancer

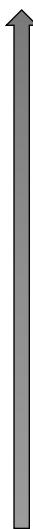
risk was one or two orders of magnitude depending on the industry involved in paint stripping activities when compared with Scenario 1.

For Scenarios 3 and 15, occupational cancer risks for the different industries fell between the risks calculated for Scenario 1 and 16, and generally exceeded one or more benchmark cancer levels when workers were exposed to high or midpoint DCM air concentrations.

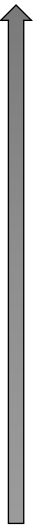
Table_Apx L-15. Occupational Cancer Risks for Professional Contractors (Scenarios 1, 3, 15 and 16)

	Professional Contractors	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier			Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	389	198	8	3.9E-03	2.0E-03	7.8E-05
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	16	8	0.31	1.6E-04	7.9E-05	3.1E-06
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	4	2	0.08	3.9E-05	2.0E-05	7.8E-07
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	2	1	0.04	1.9E-05	9.9E-06	3.9E-07

Table_Apx L-16. Occupational Cancer Risks for Automotive Refinishing (Scenarios 1, 3, 15 and 16)

	Automotive Refinishing	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier				Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	33	54	33	12	3.3E-04	5.4E-04	3.3E-04	1.2E-04
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	1	2	1	0.48	1.3E-05	2.2E-05	1.3E-05	4.8E-06
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.3	1	0.33	0.12	3.3E-06	5.4E-06	3.3E-06	1.2E-06
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.3	0.2	0.1	1.7E-06	2.7E-06	1.7E-06	6.0E-07


Table_Apx L-17. Occupational Cancer Risks for Furniture Refinishing (Scenarios 1, 3, 15 and 16)

	Furniture Refinishing	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier				Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	65	293	147	0.5	6.5E-04	2.9E-03	1.5E-03	5.0E-06
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	3	12	6	0.02	2.6E-05	1.2E-04	5.9E-05	2.0E-07
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	3	1	0.01	6.5E-06	2.9E-05	1.5E-05	5.0E-08
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	1.5	0.7	0.003	3.3E-06	1.5E-05	7.4E-06	2.5E-08


Lowest Exposure Highest Exposure	Aircraft Paint Stripping	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier			Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)		
		High	Midpoint	Low	High	Midpoint	Low
	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	496	254	11	5.0E-03	2.5E-03	1.1E-04
Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	20	10	0.44	2.0E-04	1.0E-04	4.4E-06	
Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	5	3	0.11	5.0E-05	2.5E-05	1.1E-06	
Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	2	1	0.06	2.5E-05	1.3E-05	5.5E-07	

Lowest Exposure Highest Exposure	Graffiti Removal	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier				Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	34	155	79	2.3	3.4E-04	1.6E-03	7.9E-04	2.3E-05
Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	1	6	3	0.092	1.4E-05	6.2E-05	3.2E-05	9.2E-07	
Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.340	2	1	0.023	3.4E-06	1.6E-05	7.9E-06	2.3E-07	
Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.8	0.4	0.012	1.7E-06	7.8E-06	4.0E-06	1.2E-07	


Table_Apx L-20. Occupational Cancer Risks for Non-Specific Workplace Settings—Immersion Stripping of Wood (Scenarios 1, 3, 15 and 16)

	Non-Specific Workplace Settings - Immersion Stripping of Wood	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier			Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	913	459	4.6	9.1E-03	4.6E-03	4.6E-05
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	37	18	0.184	3.7E-04	1.8E-04	1.8E-06
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	9	5	0.046	9.1E-05	4.6E-05	4.6E-07
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	5	2	0.023	4.6E-05	2.3E-05	2.3E-07

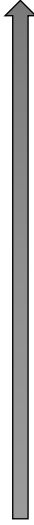
Table_Apx L-21. Occupational Cancer Risks for Non-Specific Workplace Settings—Immersion Stripping of Wood and Metal (Scenarios 1, 3, 15 and 16)

	Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier			Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	133	108	83	1.3E-03	1.1E-03	8.3E-04
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	5	4	3	5.3E-05	4.3E-05	3.3E-05
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	1.3E-05	1.1E-05	8.3E-06
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	1	1	0.415	6.7E-06	5.4E-06	4.2E-06

Table_Apx L-22. Occupational Cancer Risks for Non-Specific Workplace Settings—Unknown (Scenarios 1, 3, 15 and 16)

	Non-Specific Workplace Settings - Unknown	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier				Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	47	56	47	37	4.7E-04	5.6E-04	4.7E-04	3.7E-04
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	2	2	1	1.9E-05	2.2E-05	1.9E-05	1.5E-05
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.5	1	0.5	0.4	4.7E-06	5.6E-06	4.7E-06	3.7E-06
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.2	0.3	0.2	0.2	2.4E-06	2.8E-06	2.4E-06	1.9E-06

Table_Apx L-23. Occupational Cancer Risks for Art Restoration and Conservation (Scenarios 1, 3, 15 and 16)

	Art Restoration and Conservation	LADC (mg/m ³) ** LADCs for scenarios 2 to 16 have been adjusted with the multiplier				Excess Cancer Risk (Inhalation Unit Risk = 1x10 ⁻⁵ per mg/m ³)			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	0.3				3.0E-06			
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	0.012				1.2E-07			
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.003				3.0E-08			
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.0015				1.5E-08			

L.4.1.1 Non-Cancer Risks for Occupational Exposure Scenarios Following Chronic Exposure to DCM

EPA estimated non-cancer risks for the occupational use of DCM-containing paint strippers. Chronic exposure to DCM has been associated with liver effects. As previously discussed, the DCM IRIS assessment developed a non-cancer hazard value (i.e., POD) based on hepatic effects. EPA used the PBPK-derived 1st percentile HEC i.e., the HEC₉₉ the concentration at which there is 99% likelihood an individual would have an internal dose less than or equal to the internal dose of hazard reported in the DCM IRIS assessment ([U.S. EPA, 2011](#)) to calculate non-cancer risks associated with the repeated use of DCM-based strippers at different workplace settings.


Tables_Apx 3-24 to 3-32 show the non-cancer MOE estimates calculated for workers of different industries handling DCM-based paint strippers on a repeated basis. Selected scenarios ranging from the highest exposure scenario (i.e., no respiratory protection and high end values for EF and WY—i.e., Scenario 1) to the lowest exposure scenario (e.g., respiratory protection APF 50 and midpoints for EF and WY—Scenario 16) were included in the tables. Calculations of non-cancer risks for the full set of industries and scenarios are provided in the supplemental Excel spreadsheet, *DCM Exposure and Risk Estimates_081114.xlsx*.

Most workers using DCM-based paint strippers showed non-cancer risks for liver effects, with the exception of workers employed in the art renovation and conservation industry (Table_Apx L-33). For instance, risk concerns for liver effects were reported for most workers handling DCM-based paint strippers. These risk findings were reported with or without respiratory protection and using the product

in a repeated nature at facilities usually reporting central tendency or high-end DCM air levels. Among all of the occupational scenarios, the greatest risk concern is for workers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection.

Non-cancer risks were not observed for workers that reduce their exposure to DCM-based strippers by doing all of the following: (1) wearing adequate respiratory protection (i.e., APF 50 respirator), (2) limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years) and (3) working in facilities with low-end DCM air concentrations. This observation was reported in all of the relevant industries.

Table_Apx L-24. Occupational Non-Cancer Risks for Professional Contractors Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

	Professional Contractors	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier			Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	680	347	14	0.025	0.050	1
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	27	14	1	1	1	31
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	7	3	0.1	3	5	123
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	3	2	0.1	5	10	246

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.

Table_Apx L-25. Occupational Non-Cancer Risks for Automotive Refinishing Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

	Automotive Refinishing	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	58	95	58	21	0.3	0.2	0.3	0.8

Table_Apx L-25. Occupational Non-Cancer Risks for Automotive Refinishing Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

↑	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	4	2	1	7	5	7	20
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	0.2	30	18	30	82
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	0.5	0.3	0.1	59	36	59	164

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.

Table_Apx L-26. Occupational Non-Cancer Risks for Furniture Refinishing Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

↑ Lowest Exposure Highest Exposure	Furniture Refinishing	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	114	513	257	0.9	0.2	0.03	0.1	19
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	5	21	10	0.04	4	0.8	2	478
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	5	3	0.01	15	3	7	1911
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.6	3	1	0.005	30	7	13	3822

Table_Apx L-27. Occupational Non-Cancer Risks for Art Restoration and Conservation Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

	Art Restoration/ Conservation	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier		Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10	
		Mean ^a		Mean ^a	
↑ Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	0.5		34	
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	0.02		860	
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	0.005		3440	
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.0025		6880	


Note:^a Based on one 8-hr TWA data point reported in the OSHA IMIS database.**Note: MOEs below benchmark MOE indicating risk are denoted in bold text.****Table_Apx L-28. Occupational Non-Cancer Risks for Aircraft Stripping Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)**

	Aircraft Paint Stripping	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier			Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10		
		High	Midpoint	Low	High	Midpoint	Low
↑ Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	868	444	20	0.02	0.04	0.9
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	35	18	1	0.5	1	22
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	9	4	0.2	2	4	86
	Scenario 16	4	2	0.1	4	8	172

Table_Apx L-28. Occupational Non-Cancer Risks for Aircraft Stripping Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)


(Respirator APF 50, midpoints of ranges for EF and WY)							
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Table_Apx L-29. Occupational Non-Cancer Risks for Graffiti Removal Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)


	Graffiti Removal	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
	 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	59	271	138	4	0.3	0.1	0.1
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	2	11	6	0.2	7	2	3	105
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	3	1	0.04	29	6	12	420
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.3	1	0.7	0.02	58	13	25	839

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.


Table_Apx L-30. Occupational Non-Cancer Risks for Non-Specific Workplace Settings (Immersion Stripping of Wood) Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

	Non-Specific Workplace Settings - Immersion Stripping of Wood	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier			Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	1,598	803	8	0.01	0.02	2
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	64	32	0.3	0.3	0.5	54
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	16	8	0.08	1	2	215
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	8	4	0.04	2	4	430

Table_Apx L-31. Occupational Non-Cancer Risks for Non-Specific Workplace Settings (Immersion Stripping of Wood and Metal) Following Chronic Exposure to DCM (Scenarios 1, 3, 15 and 16)

	Non-Specific Workplace Settings - Immersion Stripping of Wood and Metal	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier			Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10		
		High	Midpoint	Low	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	232	188	145	0.07	0.1	0.1
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	9	8	6	2	2	3
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	2	2	1	7	9	12
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	1	1	1	15	18	24

Note: MOEs below benchmark MOE indicating risk are denoted in bold text.

	Non-Specific Workplace Settings - Unknown	ADC (mg/m ³) ** ADCs for scenarios 2 to 16 have been adjusted with the multiplier				Chronic MOE (24hr HEC ₉₉ = 17.2 mg/m ³) Total UF or Benchmark MOE=10			
		Mean	High	Midpoint	Low	Mean	High	Midpoint	Low
 Lowest Exposure Highest Exposure	Scenario 1 [No respirator, high ends of ranges for exposure frequency (EF) and working years (WY)]	81	98	81	65	0.21	0.18	0.21	0.27
	Scenario 3 (Respirator APF 25, high ends of ranges for EF and WY)	3	4	3	3	5	4	5	7
	Scenario 15 (Respirator APF 25, midpoints of ranges for EF and WY)	1	1	1	0.65	21	18	21	26
	Scenario 16 (Respirator APF 50, midpoints of ranges for EF and WY)	0.41	0.49	0.41	0.33	42	35	42	53

L.4.1 Human Health Risk Characterization Summary

This risk assessment focused on the occupational and consumer uses of DCM-containing paint strippers. The population of interest consisted of workers and consumers with direct (users) or indirect (bystander) exposure to DCM. Only the inhalation route of exposure was considered in this risk assessment.

The occupational and consumer exposure assessments generated the DCM exposure levels required to derive non-cancer risk estimates associated with acute and chronic exposures to DCM. In addition, cancer risks were estimated for occupational scenarios and expressed as lifetime risks, meaning the risk of developing cancer as a result of the occupational exposure over a normal lifetime of 70 yrs. Lifetime cancer risks from DCM exposure were compared to benchmark cancer risks ranging from 10^{-6} to 10^{-4} .

Many of the occupational scenarios exceeded the target cancer risks of 10^{-6} , 10^{-5} and 10^{-4} when workers employed at various industries handled DCM-paint strippers for 250 days/year for 40 years with no respiratory protection. Adequate respiratory protection and reduced exposure conditions (e.g., exposure to 125 day/year for 20 years) resulted in reduced cancer risks for workers when compared to conditions of no respiratory protection while working with paint strippers for a 250 days/year for a working lifetime (i.e., 40 years).

To characterize the risks of adverse health effects other than cancer, MOEs were used to evaluate non-cancer risks for both acute and chronic exposures using hazard values derived from peer-reviewed

hazard/dose-response assessments. Health protective hazard values were derived from the SMAC and the California acute REL hazard/dose-response assessments, whereas hazard values for non-disabling (AEGL-1) and incapacitating (AEGL-2) effects were obtained from the AEGL hazard/dose-response assessment for DCM.

Workers employed at most industries showed non-cancer risks for liver effects when using DCM-based strippers on a repeated basis. The exception was the art renovation and conservation industry which did not show non-cancer risks for the different scenarios evaluated in the assessment.

Most workers handling DCM-based paint strippers are at risk of developing non-cancer effects when they handle the product on a repeated basis with or without wearing respiratory protection. These observations were seen under various exposure conditions (i.e., exposure frequency and working years) in facilities reporting central tendency or high-end DCM air levels. Of special interest are workers using DCM-containing paint strippers engaging in long-term use of the product (i.e., 250 days/year for 40 years) with no respiratory protection as they showed the greatest risk concern for non-cancer risks.

On the contrary, non-cancer risks were not observed in workers that reduced their chronic exposure to DCM by doing all of the following: (1) wearing adequate respiratory protection (i.e., APF 50 respirator), (2) limiting exposure to central tendency exposure conditions (i.e., 125 days/year for 20 years), and (3) working in facilities with low-end DCM air concentrations.

Most occupational and residential users of DCM-based paint strippers reported acute risks for CNS effects when the SMAC and California's acute REL hazard values were used for risk estimation. These risks were observed in workers with or without respiratory protection and residential bystanders indirectly exposed to DCM.

There were concerns for discomfort/non-disabling (AEGL-1) and incapacitating (AEGL-2) effects for residential users exposed to DCM for shorter (10-min, 30-min, 1-hr) or longer exposure durations (4-hr, 8-hr) while doing the product application or staying in the residence after completion of the stripping task. These concerns were present for upper-end exposure conditions in the residential scenario as well as some of the upper-end exposure scenarios for affected bystanders.

Moreover, there were concerns for incapacitating effects (AEGL-2 effects) in workers handling DCM-containing paint strippers on an acute/short-term basis with no respiratory protection while employed in most industries involved in paint stripping. Concerns for incapacitating effects (AEGL-2 effects) were also observed for workers wearing respirators (i.e., APF 10 or APF 25) while performing paint stripping activities in industries with high DCM air concentrations [i.e., professional contractors, furniture refinishing, aircraft paint stripping, and immersion stripping of wood (non-specific workplace settings)].

The bathroom consumer modeling indicated that application of DCM-based paint strippers in a bathroom generate unsafe exposure conditions for the user of the product. Risk concerns for discomfort/non-disabling (AEGL-1) and incapacitating effects (AEGL-2) were seen in users exposed to DCM for shorter (10-min, 30-min, 1-hr) or longer exposure durations (4-hr, 8-hr) while doing the product application or staying in the residence after completion of the stripping task. However, residential bystanders did not report risk concerns for AEGL-1 and AEGL-2 effects.

Appendix M EVIDENCE INTEGRATION OF IMMUNE SYSTEM EFFECTS

Table_Apx M-1. Synthesis of Epidemiological Evidence

Endpoint	OR/HR/SMR (95% CI)	Important study characteristics	Study Confidence Rating	Reference
Mortality from infectious and parasitic diseases	SMR all divisions: 0.0 (0.0-0.66) ^a SMR roll coat: 0.67 (0.14-1.97) ^a	MeCl exposure quantified and duration-adjusted; MeCl was primary exposure for all divs; other chemical exposures possible (not controlled) for roll coat; dissimilar comparison group for all divs;	High	Hearne and Pifer (1999)
Mortality from influenza and pneumonia	SMR males: 1.25 (N/A) SMR females: 4.36 (N/A)	MeCl exposure quantified; Other chemical exposures not controlled; dissimilar comparison group	Medium	hoechst celanese corp (1992)
Mortality from bronchitis (non-specific)	HR: 9.21 (1.03–82.69)	MeCl exposure estimated based on job duties; Other chemical exposures identified (~ 21 solvents) but not controlled	Medium	Radican et al. (2008)
Mortality from non-malignant respiratory disease	SMR: 0.97 (0.42-1.90)	MeCl exposure quantified; methanol and acetone exposure not controlled; dissimilar comparison group	Medium	Lanes et al. (1993)
Sjorgen's Syndrome (autoimmune)	OR: 9.28 (2.60-33.0) 3.04 [cum.] (0.50 – 18.3)	MeCl exposure estimated based on job duties; Other chemical exposures not controlled	Medium	Chaigne et al. (2015)

^a SMRs reported in study on different scale: SMR all divs = 0 (0 - 66) and SMR roll coat = 67 (14 – 197)

Table_Apx M-2. Synthesis of Animal Evidence

Species	Exposure Route	Doses/Concentration	Duration	NOAEL ^a	Effect	Study Confidence Rating	Reference
Rat, SD	Inhalation	0, 5187 ppm	6 hrs/day, 5 days/wk, 28 days	5187 ppm	No IgM antibody response after sheep RBC injection; Decreased spleen wts (females)	High	Warbrick et al. (2003)
Mouse, CD-1 (female)	Inhalation	0, 52, 95 ppm	3 hrs	52 ppm	Acute: ↑ mortality (12.2%; p < 0.01) from <i>S. zooepidemicus</i> ; ↓ bactericidal activity (12%; p < 0.001)	Medium	Aranyi et al. (1986)
		0, 51 ppm	3 hrs/day for 5 days	51 ppm	None re: mortality or bactericidal activity		
Rat, F344	Inhalation	0, 1000, 2000, 4000 ppm	6 hrs/day, 5 days/wk, 2 years	1000 ppm	Splenic fibrosis; no patterns in inflammatory cells in respiratory tract	High	NTP (1986)
Mouse, B6C3F1	Inhalation	0, 2000, 4000 ppm	6 hrs/day, 5 days/wk, 2 years	2000 ppm	Splenic follicular atrophy; no patterns in inflammatory cells in respiratory tract	High	NTP (1986)
Rat, SD	Inhalation	0, 50, 200, 500 ppm	6 hrs/day, 5 days/wk, 2 years	500 ppm	No histopathological or other changes in lymph nodes, thymus or spleens; no patterns in inflammatory cells in respiratory tract	High	Nitschke et al. (1988a)

Species	Exposure Route	Doses/Concentration	Duration	NOAEL ^a	Effect	Study Confidence Rating	Reference
Rats, hamsters	Inhalation	0, 500, 1500, 3500 ppm	6 hrs/day, 5 days/wk, 2 years	3500 ppm	No histopathological or other changes in lymph nodes, thymus or spleens; no patterns in inflammatory cells in respiratory tract	High	Burek et al. (1984)

^aEPA-derived as related to immune endpoint

Table_Apx M-3. Synthesis of Mechanistic Evidence

System	Effect	Study Confidence Rating	Reference
Male were rats treated with hemin arginate (HAR), which induces heme oxygenase-1 (HO-1). Hemorrhage was then induced in the mice. In part of the experiment, the mice were then treated with a heme oxygenase-1 blocker, and then administered 100 mg/kg-bw methylene chloride.	<ul style="list-style-type: none"> HAR resulted in ↓ pro-inflammatory cytokine TNF-alpha and ↑ anti-inflammatory cytokine IL-10. The HO-1 blocker abolished this effect but then administration of methylene chloride restored the anti-inflammatory response. The authors suggest that the anti-inflammatory response is partly due to carbon monoxide release from administration of methylene chloride (in addition to the HAR administration/HO-1 induction) 	N/A	Kubulus et al. (2008)
Evaluation of peripheral blood mononuclear cells in carp after exposure to 0.004-40 mg/kg-bw methylene chloride by i.p.	↑ mitochondrial activity and H ₂ O ₂ of peripheral blood mononuclear cells in a dose-dependent fashion suggesting an immunomodulatory effect related to an acute pro-inflammatory state. Also, ↑ apoptosis and generation of other ROS was observed. Exact immunomodulatory effects are unclear.	N/A	Uraga-Tovar et al. (2014)

Table_Apx M-4. Evidence Integration Summary Judgment: Immunotoxicity

Summary of Human, Animal, and Mechanistic Evidence					Inferences across evidence streams
Evidence from Studies of Exposed Humans					<ul style="list-style-type: none"> Bacterial resistance and histopathological changes in the spleen are assumed to be relevant to humans Some evidence for decreased resistance to infection (bactericidal assay in rats; increased mortality in humans from flu/pneumonia) but lack of support from IgM RBC assay Autoimmunity evaluated in only one study Effects on spleen common to multiple studies Susceptible populations may include people with compromised immune systems and the elderly Other solvents have been associated with effects on the immune system
Studies, outcomes, and confidence	Factors that increase strength or certainty	Factors that decrease strength or certainty	Key findings and interpretation	Evidence stream summary	
<ul style="list-style-type: none"> Mortality from infectious disease –SMRs > and < 1 Autoimmunity – OR > 1 Mortality from non-specific respiratory disease – SMR/HR > and < 1 Hearne and Pifer 1999: high confidence; all others: medium confidence Lack of quantitative methylene chloride air concentration measurements and use of dissimilar comparison groups in most studies, Lack of control for other chemicals, some of which are solvents and may also be associated with immunotoxicity 	<ul style="list-style-type: none"> <u>Magnitude of effect</u> Large OR for one of the autoimmunity measurements One large SMR for mortality from bronchitis (but a non-specific effect) SMRs > 1 for study of mortality from flu/pneumonia (a severe outcome) 	<ul style="list-style-type: none"> <u>Inconsistency</u> Infectious disease: one SMR > 1 and another is < 1 <u>Imprecision</u> Lack of information on precision for one study (Gibbs); imprecise association for cum exposure odds ratio for autoimmunity (Chaigne) <u>Dose-response</u> Insufficient information to judge gradient <u>Coherence across types of immunity</u> Inconsistency within types of studies and limited study numbers make it difficult to judge coherence 	<ul style="list-style-type: none"> Mortality from infectious disease: Possible association with methylene chloride but results are inconsistent and outcome is severe (mortality) Autoimmunity: Possible strong association with methylene chloride but only one study is available Some study designs may limit ability to discern effects associated specifically with methylene chloride 	<ul style="list-style-type: none"> Results across human epidemiological studies suggest that methylene chloride may be associated with immunosuppression and autoimmunity Inconsistencies across studies, severity of outcome (mortality) and limitations of study design preclude firm conclusions <u>Mechanistic evidence</u>: Support unclear given the limited database 	
Evidence from In vivo Animal Studies					
Studies, outcomes, and confidence	Factors that increase strength or certainty	Factors that decrease strength or certainty	Key findings and interpretation	Evidence stream summary	
<ul style="list-style-type: none"> Bacterial resistance assay – effect observed Functional immune (IgM) assay – no effect observed Clinical chemistry/histopathology results (multiple studies) – change in histopathology 	<ul style="list-style-type: none"> <u>Effect size/precision</u>: Bacterial resistance assay showed two statistically-significant possibly related results of similar magnitude <u>Consistency</u> 	<ul style="list-style-type: none"> Only a single study of bacterial resistance is available Burek didn't identify histopathological changes in the spleen at a concentration identified with splenic changes in other studies 	<ul style="list-style-type: none"> One study positive for bactericidal activity but limited support Support from animal studies only includes histopathological changes in the spleen in some studies. 	<ul style="list-style-type: none"> Limited information based on a single study of bactericidal resistance with some changes in spleens in some studies. However, lack of support from IgM RBC assay 	

Summary of Human, Animal, and Mechanistic Evidence				Inferences across evidence streams
<p>of spleen within some studies</p> <ul style="list-style-type: none"> Aranyi et al. 1986: medium confidence; all others: high confidence 	<p>Several studies showed effects on spleen (decreased weight, atrophy, fibrosis)</p> <ul style="list-style-type: none"> <u>Dose-response gradient</u> – spleen effects observed at higher concentrations 	<ul style="list-style-type: none"> Splenic fibrosis showed somewhat unclear dose-response trend (2%, 10%, 20%, 14% at 0, 1000, 2000 and 4000 ppm) Two-year studies didn't identify effects on immune cells and organs than the spleen No increased rates of infection were identified in 13-week and 2-year studies RBC study to determine IgM response was negative. 	<ul style="list-style-type: none"> <u>Mechanistic evidence</u>: Support is unclear given the limited database 	
Mechanistic Evidence or Supplemental Information				
Biological events or pathways (or other information)	Species or model systems	Key findings, limitations, and interpretation (for each row below)	Evidence stream summary	
<ul style="list-style-type: none"> Pro-inflammatory, but somewhat non-specific, changes (one study) Anti-inflammatory changes (one study) 	<ul style="list-style-type: none"> Two <i>in vivo</i> studies Rat and carp 	<p>The limited number of studies, differences in species, types of cells and substances studied as well as differences in processes evaluated make it difficult to make any conclusions regarding these studies.</p>	<p>Little can be concluded from these two studies that have very different study protocols. It is not clear whether the studies suggest opposite effects or are just two aspects of a coordinated immune response.</p>	

ATTACHMENT B

link to log on and submit the intervention or protests.

Persons unable to file electronically may mail similar pleadings to the Federal Energy Regulatory Commission, 888 First Street NE, Washington, DC 20426. Hand delivered submissions in docketed proceedings should be delivered to Health and Human Services, 12225 Wilkins Avenue, Rockville, Maryland 20852.

In addition to publishing the full text of this document in the **Federal Register**, the Commission provides all interested persons an opportunity to view and/or print the contents of this document via the internet through the Commission's Home Page (<http://ferc.gov>) using the eLibrary link. Enter the docket number excluding the last three digits in the docket number field to access the document. At this time, the Commission has suspended access to the Commission's Public Reference Room, due to the proclamation declaring a National Emergency concerning the Novel Coronavirus Disease (COVID-19), issued by the President on March 13, 2020. For assistance, contact the Federal Energy Regulatory Commission at FERCOnlineSupport@ferc.gov or call toll-free, (886) 208-3676 or TTY, (202) 502-8659.

Dated: June 18, 2020.

Nathaniel J. Davis, Sr.,
Deputy Secretary.

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ENVIRONMENTAL PROTECTION AGENCY

[EPA-HQ-ORD-2015-0765; FRL-10011-20-ORD]

Board of Scientific Counselors (BOSC) Executive Committee Meeting—July 2020

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of public meeting.

SUMMARY: The Environmental Protection Agency (EPA), Office of Research and Development (ORD), gives notice of a meeting of the Board of Scientific Counselors (BOSC) Executive Committee (EC) to review the Chemical Safety and Sustainability and Health and Environmental Risk Assessment (CSS-HERA) Subcommittee's report on the Strategic Research Action Plan (StRAP) of ORD's HERA research program. The committee will also receive a briefing on ORD research on SARS-COV-2 and EPA's new approach

methods (NAMs) work plan to reduce animal testing.

DATES: The videoconference meeting will be held on Tuesday, July 7, 2020, from 11:00 a.m. to 5:15 p.m. (EDT). Meeting times are subject to change. This meeting is open to the public. Those who wish to attend must register by July 6, 2020. Comments must be received by July 6, 2020, to be considered by the subcommittee. Requests for the draft agenda or making a presentation at the meeting will be accepted until July 3, 2020.

ADDRESSES: Instructions on how to connect to the videoconference will be provided upon registration at <https://epa-bosc-executive-committee.eventbrite.com>. Attendees should register no later than July 6, 2020.

Submit your comments to Docket ID No. EPA-HQ-ORD-2015-0765 by one of the following methods:

- www.regulations.gov: Follow the online instructions for submitting comments.
 - *Note:* comments submitted to the www.regulations.gov website are anonymous unless identifying information is included in the body of the comment.
 - *Email:* Send comments by electronic mail (email) to: ORD.Docket@epa.gov, Attention Docket ID No. EPA-HQ-ORD-2015-0765.
 - *Note:* comments submitted via email are not anonymous. The sender's email will be included in the body of the comment and placed in the public docket which is made available on the internet.

Instructions: All comments received, including any personal information provided, will be included in the public docket without change and may be made available online at www.regulations.gov. Information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute will not be included in the public docket, and should not be submitted through www.regulations.gov or email. For additional information about the EPA's public docket visit the EPA Docket Center homepage at <http://www.epa.gov/dockets/>.

Public Docket: Publicly available docket materials may be accessed *Online* at www.regulations.gov. Copyrighted materials in the docket are only available via hard copy. The telephone number for the ORD Docket Center is (202) 566-1752.

FOR FURTHER INFORMATION CONTACT: The Designated Federal Officer (DFO), Tom Tracy, via phone/voice mail at: (202)

564-6518; or via email at: tracy.tom@epa.gov. Any member of the public interested in receiving a draft agenda, attending the meeting, or making a presentation at the meeting should contact Tom Tracy.

SUPPLEMENTARY INFORMATION: The Board of Scientific Counselors (BOSC) is a federal advisory committee that provides advice and recommendations to EPA's Office of Research and Development on technical and management issues of its research programs. Meeting agenda and materials will be posted to <https://www.epa.gov/bosc>. Proposed agenda items for the meeting include but are not limited to the following: Review of the CSS-HERA report, ORD research on SARS-COV-2, and EPA's NAMs work plan.

Information on Services Available: For information on translation services, access, or services for individuals with disabilities, please contact Tom Tracy at (202) 564-6518 or tracy.tom@epa.gov. To request accommodation of a disability, please contact Tom Tracy at least ten days prior to the meeting to give the EPA adequate time to process your request.

Authority: Pub. L. 92-463, 1, Oct. 6, 1972, 86 Stat. 770.

Dated: June 19, 2020.

Mary Ross,
Director, Office of Science Advisor, Policy, and Engagement.

[FR Doc. 2020-13620 Filed 6-23-20; 8:45 am]

BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

[EPA-HQ-OPPT-2019-0437; FRL-10011-16]

Methylene Chloride (MC); Final Toxic Substances Control Act (TSCA) Risk Evaluation; Notice of Availability

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The Environmental Protection Agency (EPA) is announcing the availability of the final Toxic Substances Control Act (TSCA) risk evaluation of methylene chloride (MC). The purpose of conducting risk evaluations under TSCA is to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment under the conditions of use, including an unreasonable risk to a relevant potentially exposed or susceptible subpopulation. EPA has determined that specific conditions of use of methylene

chloride present an unreasonable risk of injury to health. For those conditions of use for which EPA has found an unreasonable risk, EPA must move to address that unreasonable risk through risk management measures enumerated in TSCA. EPA has also determined that specific conditions of use do not present unreasonable risk of injury to health or the environment. For those conditions of use for which EPA has found no unreasonable risk to health or the environment, the Agency's determination is a final Agency action and is issued via order in the risk evaluation.

ADDRESSES: The docket for this action, identified by docket identification (ID) number EPA-HQ-OPPT-2019-0437, is available online at <http://www.regulations.gov> or in-person at the Office of Pollution Prevention and Toxics Docket (OPPT Docket), Environmental Protection Agency Docket Center (EPA/DC), West William Jefferson Clinton Bldg., Rm. 3334, 1301 Constitution Ave., NW, Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the OPPT Docket is (202) 566-0280. Please review the visitor instructions and additional information about the docket available at <http://www.epa.gov/dockets>.

Please note that due to the public health emergency the EPA Docket Center (EPA/DC) and Reading Room was closed to public visitors on March 31, 2020. Our EPA/DC staff will continue to provide customer service via email, phone, and webform. For further information on EPA/DC services, docket contact information and the current status of the EPA/DC and Reading Room, please visit <https://www.epa.gov/dockets>.

FOR FURTHER INFORMATION CONTACT:

For technical information contact: Dr. Stan Barone, Office of Pollution Prevention and Toxics (7403M), Environmental Protection Agency, 1200 Pennsylvania Ave. NW, Washington, DC 20460-0001; telephone number: (202) 564-1169; email address: barone.stan@epa.gov.

For general information contact: The TSCA-Hotline, ABVI-Goodwill, 422 South Clinton Ave., Rochester, NY 14620; telephone number: (202) 554-1404; email address: TSCA-Hotline@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

This action is directed to the public in general. This action may be of interest to persons who are or may be interested in risk evaluations of chemical substances under TSCA, 15 U.S.C. 2601 *et seq.* Since other entities may also be interested in this final risk evaluation, the EPA has not attempted to describe all the specific entities that may be affected by this action.

B. What is EPA's authority for taking this action?

TSCA section 6, 15 U.S.C. 2605, requires EPA to conduct risk evaluations to "determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of costs or other nonrisk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." 15 U.S.C. 2605(b)(4)(A). TSCA sections 6(b)(4)(A) through (H) enumerate the deadlines and minimum requirements applicable to this process, including provisions that provide instruction on chemical substances that must undergo evaluation, the minimum components of a TSCA risk evaluation, and the timelines for public comment and completion of the risk evaluation. TSCA also requires that EPA operate in a manner that is consistent with the best available science, make decisions based on the weight of the scientific evidence and consider reasonably available information. 15 U.S.C. 2625(h), (i), and (k). TSCA section 6(i) directs that a determination of "no unreasonable risk" shall be issued by order and considered to be a final Agency action, while a determination of "unreasonable risk" is not considered to be a final Agency action. 15 U.S.C. 2605(i).

The statute identifies the minimum components for all chemical substance risk evaluations. For each risk evaluation, EPA must publish a document that outlines the scope of the risk evaluation to be conducted, which includes the hazards, exposures, conditions of use, and the potentially exposed or susceptible subpopulations that EPA expects to consider. 15 U.S.C. 2605(b)(4)(D). The statute further provides that each risk evaluation must also: (1) Integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance, including information that is relevant to specific risks of injury to health or the environment and

information on relevant potentially exposed or susceptible subpopulations; (2) describe whether aggregate or sentinel exposures were considered and the basis for that consideration; (3) take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use; and (4) describe the weight of the scientific evidence for the identified hazards and exposures. 15 U.S.C. 2605(b)(4)(F)(i)-(ii) and (iv)-(v). Each risk evaluation must not consider costs or other nonrisk factors. 15 U.S.C. 2605(b)(4)(F)(iii).

The statute requires that the risk evaluation process be completed within a specified timeframe and provide an opportunity for public comment on a draft risk evaluation prior to publishing a final risk evaluation. 15 U.S.C. 2605(b)(4).

In conducting risk evaluations, "EPA will determine whether the chemical substance presents an unreasonable risk of injury to health or the environment under each condition of use within the scope of the risk evaluation . . ." 40 CFR 702.47. Pursuant to TSCA section 6(i)(1), a determination of "no unreasonable risk" shall be issued by order and considered to be final Agency action. Under EPA's implementing regulations, "[a] determination by EPA that the chemical substance, under one or more of the conditions of use within the scope of the risk evaluation, does not present an unreasonable risk of injury to health or the environment will be issued by order and considered to be a final Agency action, effective on the date of issuance of the order." 40 CFR 702.49(d). Subsection 5.4.1 of the final risk evaluation for MC constitutes the order required under TSCA section 6(i)(1), and the "no unreasonable risk" determinations in that subsection are considered to be a final Agency action effective on the date of issuance of the order.

C. What action is EPA taking?

EPA is announcing the availability of the risk evaluation of the chemical substance identified in Unit II. In this risk evaluation EPA has made unreasonable risk determinations on all the conditions of use within the scope of the risk evaluation for this chemical. For those conditions of use for which EPA has found an unreasonable risk of injury to health or the environment, EPA must move to address those risks through risk management measures enumerated in 15 U.S.C. 2605(a). For those conditions of use for which EPA has found no unreasonable risk of injury to health or the environment, the Agency's determination is a final

Agency action and is issued via order, per 15 U.S.C. 2605(i)(1), in the risk evaluation, subsection 5.4.1.

EPA is also announcing the availability of the information required to be provided publicly with each risk evaluation. 40 CFR 702.51. Specifically, EPA has provided:

- The scope document and problem formulation (in Docket EPA-HQ-OPPT-2016-0742);
- Draft risk evaluation, and final risk evaluation (in Docket EPA-HQ-OPPT-2019-0437);
- All notices, determinations, findings, consent agreements, and orders (in Docket EPA-HQ-OPPT-2019-0437);
- Any information required to be provided to the Agency under 15 U.S.C. 2603 (in Docket EPA-HQ-OPPT-2016-0742 and Docket EPA-HQ-OPPT-2019-0437);
- A nontechnical summary of the risk evaluation (in Docket EPA-HQ-OPPT-2019-0437);
- A list of the studies, with the results of the studies, considered in carrying out each risk evaluation (Risk Evaluation for Methylene Chloride (Dichloromethane, DCM) in Docket EPA-HQ-OPPT-2019-0437);
- The final peer review report, including the response to peer review and public comments received during peer review (in Docket EPA-HQ-OPPT-2019-0437); and
- Response to public comments received on the draft scope and the draft risk evaluation (in Docket EPA-HQ-OPPT-2019-0437).

II. TSCA Risk Evaluation

A. What is EPA's risk evaluation process for existing chemicals under TSCA?

The risk evaluation process is the second step in EPA's existing chemical process under TSCA, following prioritization and before risk management. As this chemical is one of the first ten chemical substances undergoing risk evaluation, the chemical substance was not required to go through prioritization (81 FR 91927, December 19, 2016) (FRL-9956-47). The purpose of conducting risk evaluations is to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment under the conditions of use, including an unreasonable risk to a relevant potentially exposed or susceptible subpopulation. As part of this process, EPA must evaluate both hazard and exposure, not consider costs or other nonrisk factors, use reasonably available information and approaches in a manner that is consistent with the

requirements in TSCA for the use of the best available science, and ensure decisions are based on the weight of scientific evidence.

The specific risk evaluation process that EPA has established by rule to implement the statutory process is set out in 40 CFR part 702 and summarized on EPA's website at <http://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-evaluations-existing-chemicals-under-tsca>. As explained in the preamble to EPA's final rule on procedures for risk evaluation (82 FR 33726, July 20, 2017) (FRL-9964-38), the specific regulatory process set out in 40 CFR part 702, subpart B is being followed for the first ten chemical substances undergoing risk evaluation to the maximum extent practicable.

Prior to the publication of this final risk evaluation, a draft risk evaluation was subject to peer review and public comment. EPA reviewed the report from the peer review committee and public comments and has amended the risk evaluation in response to these comments as appropriate. The public comments, peer review report, and EPA's response to comments is in Docket EPA-HQ-OPPT-2019-0437. Prior to the publication of the draft risk evaluation, EPA made available the scope and problem formulation, and solicited public input on uses and exposure. EPA's documents and the public comments are in Docket EPA-HQ-OPPT-2016-0732. Additionally, information about the scope, problem formulation, and draft risk evaluation phases of the TSCA risk evaluation for this chemical is at <http://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-evaluation-methylene-chloride-0>.

B. What is methylene chloride?

Methylene chloride (MC), also known as dichloromethane and DCM, is a volatile chemical used as a solvent in a wide range of industrial, commercial and consumer applications. The primary uses for methylene chloride are for paint removal, adhesives, metal cleaning, aerosol solvents, chemical processing and flexible polyurethane foam manufacturing. Information from the 2016 Chemical Data Reporting (CDR) for MC indicates the reported production volume is more than 260 million lbs per year (manufacture and import).

Authority: 15 U.S.C. 2601 *et seq.*

Dated: June 17, 2020.

Andrew Wheeler,
Administrator.

[FR Doc. 2020-13581 Filed 6-23-20; 8:45 am]

BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

[FRL-10010-92-Region 5]

Clean Air Act Operating Permit Program; Petition for Objection to State Operating Permit for Riverview Energy Corporation; Petition for Objection to State Operating Permit for ESSROC Cement Corporation

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of final orders on petitions for objection to two Clean Air Act title V operating permits.

SUMMARY: The Environmental Protection Agency (EPA) Administrator signed an Order dated March 26, 2020, denying a Petition dated August 6, 2019 from Southwestern Indiana Citizens for Quality of Life, Inc. and Valley Watch, Inc. The Petition requested that EPA object to a Clean Air Act (CAA) title V operating permit issued by the Indiana Department of Environmental Management (IDEM) to Riverview Energy Corporation for its direct coal hydrogenation facility located in Dale, Spencer County, Indiana. The EPA Administrator also signed an Order dated April 1, 2020, denying a Petition dated January 4, 2017 from Vicki L. Whittinghill. The Petition requested that EPA object to a CAA title V operating permit issued by IDEM to ESSROC Cement Corporation for its Portland cement manufacturing plant located in Clark County, Indiana.

ADDRESSES: The final Orders, the Petitions, and other supporting information are available for public inspection during normal business hours at the following address: U.S. Environmental Protection Agency, Region 5, Air and Radiation Division, 77 West Jackson Boulevard, Chicago, Illinois 60604. This facility is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding Federal holidays and facility closures due to COVID-19. We recommend that you telephone Michael Langman, Environmental Scientist, at (312) 886-6867 before visiting the Region 5 office. Additionally, the final Orders and Petitions are available electronically at: <https://www.epa.gov/title-v-operating-permits/title-v-petition-database>.