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Draft Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro)

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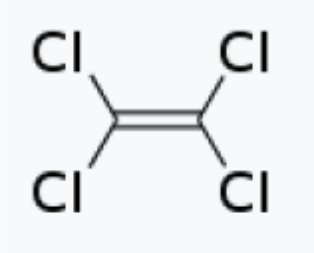

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ABBREVIATIONS

°C	Degrees Celsius
µg	Microgram(s)
1-BP	1-Bromopropane
1Q10	Lowest 1-day average flow that occurs (on average) once every 10 years
30Q5	Lowest 30-day average flow that occurs (on average) once every 5 years
7Q10	Lowest 7-day average flow that occurs (on average) once every 10 years
AAP	Alanine aminopeptidase
ABC	ATP Binding Cassette
AC	Acute Concentration
ACGIH®	American Conference of Government Industrial Hygienists
ADC	Average Daily Concentrations
ADME	Absorption/Distribution/Metabolism/Elimination
ADR	Acute Dose Rate
AEGL	Acute Exposure Guideline Level
AF	Assessment Factor
ALS	Amyotrophic Lateral Sclerosis
ALT	Aminotransferase
AML	Acute Myeloid Leukemia
ANCA	Antineutrophil-Cytoplasmic Antibody
APF	Assigned Protection Factor
ASD	Autism Spectrum Disorder
Atm	Atmosphere(s)
ATSDR	Agency for Toxic Substances and Disease Registries
AUC	Area Under the Curve
Avg	Average
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BIOWIN	EPI Suite biodegredation module
BLS	US Bureau of Labor Statistics
BMD	Benchmark Dose
BMDL/BMCL	Benchmark Dose/Concentration Lower Bound
BMR	Benchmark Dose Response
BW	Body Weight
CAA	Clean Air Act
CARB	California Air Resources Board
CASRN	Chemical Abstracts Service Registry Number
CBI	Confidential Business Information
CCI	Color Confusion Index
CCL_4	Carbon Tetrachloride
CD	Cluster of Differentiation
CDC	Centers for Disease Control
CDR	Chemical Data Reporting

CDSMF	California Death Statistical Master File
CEHD	Chemical Exposure Health Data
CEM	Consumer Exposure Model
CEPA	Canadian Environmental Protection Agency/Act
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
CF	Conversion Factor
CFC	Chlorofluorocarbon
CFR	Code of Federal Regulations
CHIRP	Chemical Risk Information Platform
ChV	Chronic Toxicity Value
CI	Confidence Interval
cm^3	Cubic Centimeter(s)
CNS	Central Nervous System
CoA	Coenzyme A
COC	Concentration of Concern
COPD	Chronic Obstructive Pulmonary Disease
CoRAP	Community Rolling Action Plan
COU	Condition of Use
cP	Centipoise
CPCat	Chemical and Product Categories
CPS	Current Population Survey
CPSC	Consumer Product Safety Commission
CSCL	Chemical Substances Control Law
CT	central tendency
CWA	Clean Water Act
CYP	Cytochrome P
DCA	Dichloroacetic Acid
DF	Dilution Factor
DLBCL	Diffuse Large B-cell Lymphoma
DMR	Discharge Monitoring Report
DNA	Deoxyribonucleic Acid
DNAPL	Dense Non-Aqueous Phase Liquid
DNP	Dinitrophenol
DoD	Department of Defense
DQE	Data Quality Evaluation
EC50	Half Maximal Effective Concentration
ECHA	European Chemicals Agency
ECHO	Enforcement and Compliance History Online
ECOTOX	ECOTOXicology knowledgebase
EDC	Ethylene Dichloride
EEG	Electrocochleogram
E-FAST	Exposure and Fate Assessment Screening Tool
EG	Effluent Guidelines

ELCR	Excess Lifetime Cancer Risk
EPA	Environmental Protection Agency
EPANET	EPA water distribution system model
EPCRA	Emergency Planning and Community Right-to-Know Act
EPISuite	Estimation Programs Interface (EPI) Suite
ESD	Emission Scenario Documents
EU	European Union
FDA	Food and Drug Administration
FFDCA	Federal Food, Drug and Cosmetic Act
FHSA	Federal Hazardous Substance Act
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FR(s)	Federal Regulation
G	Gram(s)
GACT	Generally Available Control Technology
GD	Gestation Day
GIS	Geographical Information System
GM	Geometric Mean
GPS	Global Positioning System
GS	Generic Scenario
GSD	Geometric Standard Deviation
GSH	Glutathione
GST	Glutathione S-transferase
HAP	Hazardous Air Pollutant
HCFC	Hydrochlorofluorocarbon
HCl	Hydrochloric Acid
HE	High End
HEC	Human Equivalent Concentration
HED	Human Equivalent Dose
HERO	Health and Environmental Research Online (database)
HFC	Hydrofluorocarbon
HPV	High Production Volume
Hr	Hour(s)
HRs	Hazard Ratios
HSIA	Halogenated Solvents Industry Association
HUC	Hydrologic Unit Codes
i.p.	Intraperitoneal
IARC	International Agency for Research on Cancer
ICD	International Classification of Diseases
IDLH	Immediately Dangerous to Life and Health
IgA	Immunoglobulin A
IgE	Immunoglobulin E
IRIS	Integrated Risk Information System
IRTA	Institute for Research and Technical Assistance

ISHA	Industrial Safety and Health Act
IUR(s)	Inhalation Unit Risk(s)
kg	Kilogram(s)
L	Liter(s)
LADC	Lifetime Average Daily Concentration
lb	Pound(s)
LC50	Lethal Concentration 50
LDH	Lactate Dehydrogenase
LOAEC	Lowest Observable Adverse Effect Concentration
LOAEL	Lowest Observed Adverse Effect Level
LOD	Limit of Detection
LOEC	Lowest Observed 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
Max.	Maximum
MCCEM	Multi-Chamber Concentration Exposure Model
MCL	Mononuclear Cell Leukemia (Hazard sections)
MCL	Maximum Contaminant Level (Surface Water sections)
MCLG	Maximum Contaminant Level Goal
MF	Mycosis Fungoides
Mfg	Manufacturing
mg	Milligram(s)
Min	Minute
Min.	Minimum
MLD	Million Liters per Day
MM	Multiple Myeloma
mmHg	Millimeter(s) of Mercury
MOA	Mode of Action
MOE	Margin of Exposure
mRNA	Messenger RNA
MSDS	Material Safety Data Sheet
n	Number variable (also N)
N/A	Not Available; Not Applicable
NAAQS	National Ambient Air Quality Standards
NAC	National Advisory Committee
NAcTCVC	N-acetylate TCVC
NAG	N-acetyl glucuronidase
NAICS	North American Industry Classification System
NATA	National Air Toxics Assessment
NAWQA	National Water-Quality Assessment

NCEA	National Center for Environmental Assessment
NCHS	National Center for Health Statistics
ND	Non-detect
NDI	National Death Index
NEI	National Emissions Inventory
NESHAP	National Emission Standards for Hazardous Air Pollutants
NHANES	National Health and Nutrition Examination Survey
NHD	National Hydrological Dataset
NHEXAS	National Human Exposure Assessment 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
NOACC	Nordic Occupational Cancer Study
NOAEC	No Observable Adverse Effect Concentration
NOAEL	No Observed Adverse Effect Level
NOEC	No Observable Effect Concentration
NOEL	No Observable Effect Level
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulations
NPL	National Priorities List
NR	Not Reported
NRC	National Research Council
NTP	National Toxicology Program
NWIS	National Water Information Systems
OAQPS	Office of Air Quality Planning and Standards
OCPSF	Organic Chemicals, Plastics and Synthetic Fibers
OCSPP	Office of Chemical Safety and Pollution Prevention
ODS	Ozone Depleting Substance
OECD	Organisation for Economic Co-operation and Development
OEHHA	Office of Environmental Health Hazard Assessment
OEL	Occupational Exposure Limit
OEM	Original Equipment Manufacturer
OES	Occupational Exposure Scenarios
ONU	Occupational Non-User
OPPT	Office of Pollution Prevention and Toxics
ORs	Odds Ratios
OSHA	Occupational Safety and Health Administration
OTPR	Oily Type Paint Removers
OTVD	Open Top Vapor Degreasing
PAPR	Power Air-Purifying Respirator
RPB	Retinol-binding protein

PBPK	Physiologically Based Pharmacokinetic
PBZ	Personal Breathing Zone
PCA	Passive Cutaneous Anaphylaxis
PCE	Perchloroethylene
PCO	Palmitoyl CoA Oxidation
PDM	Probabilistic Dilution Model
PECO	Populations, Exposures, Comparators and Outcomes
PEL	Permissible Exposure Limit
PESS	Potentially Exposed Susceptible Subpopulation
PF	Protection Factor
pH	Potential for Hydrogen (also Power of Hydrogen)
PND	Postnatal Day
POD	Point of Departure
POTW	Publicly Owned Treatment Works
PPARα	Peroxisome Proliferator-Activated Receptor alpha
ppb	Part(s) per Billion
PPE	Personal Protective Equipment
ppm	Part(s) per Million
P_{trend}	P-value trend
PWS	Public Water System
RCRA	Resource Conservation and Recovery Act
RDD	Relative Delivered Dose
RESO	Receptors, Exposure, Setting (or Scenario), Outcome
RfC(s)	Reference Concentration(s)
RQ	Risk Quotient
RR	Risk Ratio
S9	Fraction of an organ tissue homogenate used in biological assays to add metabolic activity
SAR	Supplied-Air Respirator
SARA	Superfund Amendments and Reauthorization Act
SCBA	Self-Contained Breathing Apparatus
SCEs	Sister Chromatid Exchange(s)
SCHER	Scientific Committee on Health and Environmental Risks
SD	Standard Deviation
SDS	Safety Data Sheet
SDWA	Safe Drinking Water Act
SEMS	Superfund Enterprise Management System
SF	Stream Flow
SHIELD	School Health Initiative: Environment, Learning, Disease
SIC	Standard Industry Classification
SIDS	Screening Information Data Set
SIR	Standardized Incidence Ratios
SMR	Standard Mortality Ratio

SNAP	Significant New Alternatives Policy
SpERC	Specific Environmental Release Category
SSADMF	Social Security Administration Death Master File
STEL	Short-Term Exposure Limit
STEWARDS	USDA ARS Sustaining the Earth's Watersheds - Agricultural research Database System
STORET	EPA STORage and RETrieval data warehouse
STP	Standard Temperature and Pressure
SUSB	U.S. Census Statistics of US Businesses
SWC	Surface Water Concentration
$t_{1/2}$	Half-life
TCA	Trichloroacetic Acid
TCAC	Trichloroacetyl Chloride
TCCR	Transparent, Clear, Consistent, and Reasonable
TCE	Trichloroethylene
TCOH	Trichloroethanol
TCVC	S-(1,2,2-trichlorovinyl) cysteine
TCVCS	TCVC sulfoxide
TCVG	S-(1,2,2-trichlorovinyl) glutathione
TCVMA	N-acetyl-S-(trichlorovinyl)-l-cystine
TEAM	Total Exposure Assessment Methodology
TLV®	Threshold Limit Value
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TTO	Total Toxic Organics
TWA	Time-Weighted Average
U.S.	United States
UFs	Uncertainty Factors
USGS	United States Geological Survey
VA	Veteran's Affairs
VACCR	Veteran's Affairs Central Cancer Registry
VOC	Volatile Organic Compound
WBC	White Blood Cells
WESTAT	National solvent usage survey (Westat 1987)
WHO	World Health Organization
WOE	Weight of Evidence
WQP	Water Quality Portal
WQX	Water Quality Exchange
WWR	Waste Water Release
WWTP	Wastewater Treatment Plants
Yr	Year(s)

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EXECUTIVE SUMMARY

This draft risk evaluation for perchloroethylene was performed in accordance with the Frank R. Lautenberg Chemical Safety for the 21st Century Act and is being disseminated for 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. As per EPA's final rule, Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act (82 FR 33726), EPA is taking comment on this draft, and will also obtain peer review on this draft risk evaluation for PCE. All conclusions, findings, and determinations in this document are preliminary and subject to comment. The final risk evaluation may change 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. The preliminary conclusions, findings, and determinations in this draft risk evaluation are for the purpose of identifying whether the chemical substance presents unreasonable risk of injury to health or the environment under the conditions of use, including unreasonable risk to a potentially exposed or susceptible subpopulation (PESS) in accordance with TSCA section 6, and are not intended to represent any findings under TSCA section 7.

PCE is subject to federal and state regulations and reporting requirements. PCE has been a reportable 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) and as such is subject to effluent limitations.

PCE is currently manufactured, processed, distributed, used, and disposed of as part of industrial, commercial, and consumer conditions of use. PCE has a wide-range of uses, including production of fluorinated compounds, and as a solvent in dry cleaning and vapor degreasing. A variety of consumer and commercial products use PCE such as adhesives (arts and crafts, as well as light repairs), aerosol degreasing, brake cleaners, aerosol lubricants, sealants, stone polish, stainless steel polish and other wipe cleaners (cleaners used for wiping surfaces). EPA evaluated the following categories of conditions of use: manufacturing; processing; distribution in commerce, industrial, commercial and consumer uses and disposal. The yearly aggregate production volume ranged from 388 to 324 million pounds between 2012 and 2015.

Approach

EPA used reasonably available information (defined in 40 CFR 702.33 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 analyses 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 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 2018b).

In the problem formulation, EPA identified the conditions of use and presented three conceptual models and an analysis plan for this draft risk evaluation. These have been carried into the draft 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

draft risk evaluation) and exposure pathways within the scope of the risk evaluation. While PCE is present in various environmental media, such as groundwater, surface water, and air, EPA stated in the problem formulation that EPA did not expect to include in the risk evaluation certain exposure pathways that are under the jurisdiction of other EPA-administered statutes in this draft risk evaluation as described in Section 1.4.

EPA quantitatively evaluated the risk to aquatic species from exposure to surface water from the manufacturing, processing, use, or disposal of PCE. EPA used environmental fate parameters, physical-chemical properties, modelling, and monitoring data to assess ambient water exposure to aquatic species. During the systematic review process, EPA identified and evaluated studies that warranted further evaluation. Therefore, exposures to aquatic organisms from ambient surface water, are assessed and presented in this draft risk evaluation and used to inform the risk determination. These analyses are described in Sections 2.1, 2.3, 4.1.

EPA evaluated exposures to PCE 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 occupational users (workers) and occupational non-users (ONUs)[1], and acute and chronic dermal exposures to workers. EPA used inhalation monitoring data from literature sources, where reasonably available and that met data evaluation criteria, as well as modeling approaches, where reasonably available, to estimate potential inhalation exposures. Dermal doses for workers were estimated in these 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 was estimated since inhalation and dermal monitoring data were not reasonably available. These analyses are described in Section 2.4 of this draft 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 2018b). EPA concluded that PCE poses a hazard to environmental aquatic receptors with algae being the most sensitive taxa for 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 (U.S. EPA 2014c) to evaluate, extract, and integrate PCE's human health hazard and dose-response information. EPA reviewed key and supporting information from previous hazard assessments, EPA IRIS Toxicologic Review (U.S. EPA 2012e), an ATSDR Toxicological Profile (ATSDR 2019), AEGL (NAC/AEGL 2009), 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 2012 – 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 PCE exposure analyses are described in Section 3.2.

[1] ONUs are workers who do not directly handle PCE but perform work in an area where PCE is present.

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. The results of the risk characterization are in Section 4.1, 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. The estimated releases from specific facilities result in modeled surface water concentrations that were equal to or exceed the aquatic benchmark (RQ ≥ 1) for seven conditions of use, indicating that exposures resulting from environmental concentrations were greater than the effect concentration or the concentration of concern. Details of these estimates are in Section 4.1.2.

Human Health Risks

Risks were estimated following both acute and chronic exposure for representative endpoints from every hazard domain. EPA identified potential cancer and non-cancer human health risks. The studies that support the health concerns address neurotoxicity (CNS) effects from acute exposures, and neurological, kidney, liver, immune system and developmental effects from chronic exposures and cancer.

EPA estimated risk to workers from inhalation and dermal exposures, and risk to occupational non-users (ONUs) from inhalation exposures by comparing the estimated exposures to acute and chronic human health hazards For workers and ONUs, EPA estimated the cancer risk as the product of the chronic exposure to PCE and the inhalation Unit Risk value for each COU. For dermal exposure to workers, cancer risk was estimated as the product of the dermal exposure and the cancer slope factor for each COU. For workers and ONUs, EPA estimated exposure and used the MOE approach to assess the margin of exposure (MOE) for non-cancer health effects. 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 PCE. More information on respiratory and dermal protection, including EPA's approach regarding the occupational exposure scenarios for PCE, is in Section 2.4.1.

For occupational scenarios, using the MOE approach for non-cancer endpoints, risks were indicated for all conditions of use, except for use of laboratory chemicals, under high-end inhalation or dermal exposure scenarios if PPE was not used. For the majority of exposure scenarios, risk to workers were identified for multiple endpoints in both acute and chronic exposure scenarios. Based on the PODs selected from among the acute and chronic endpoints, acute and chronic non-cancer and cancer risks were indicated for all but one exposure scenarios and occupational conditions of use under high-end inhalation or dermal exposure levels without the use of PPE. Use of PPE during the assessed conditions of use is expected to reduce worker exposure. This resulted in fewer conditions of use with estimated risks for acute, chronic non-cancer, or cancer inhalation or dermal exposures. With assumed use of respiratory protection, cancer risks from chronic inhalation exposures were not indicated for most conditions of use. With assumed use of dermal protection, acute, chronic non-cancer, and cancer risks were not indicated for some 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 assumed PPE (i.e., respirators APF 10, 25 or 50). EPA's estimates for worker risks for each occupational exposure scenario are presented in Section 4.2.1 and summarized in Table 4-112.

ONUs are expected to have lower exposure levels than workers in most instances but exposures could

not always be quantified based on reasonably available data and risk estimates for ONUs may be similar to workers in some settings. While the difference between the exposures of ONUs and the exposures of workers directly handling PCE generally cannot be quantified, ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical. In these instances, EPA considered the ONU exposures to be equal to the central tendency risk estimates for workers when determining ONU risk attributable to inhalation. While this is likely health protective as it assumes ONU exposure is as high as it is for the majority of workers (greater numbers are likely to be exposed near the middle of the distribution), this is uncertain. Dermal exposures are not expected because ONUs do not typically directly handle PCE, nor they are in the immediate proximity of PCE.

Based on central-tendency exposure levels, acute and chronic non-cancer risks to ONUs were indicated for the majority of exposure scenarios. ONUs are not assumed to be using PPE to reduce exposures to PCE used in their vicinity. ONUs are not expected to be dermally exposed to PCE and therefore dermal risks to ONUs were not assessed. EPA's estimates for ONU risks for each occupational exposure scenario are presented alongside worker risk estimates in Section 4.2.2.

EPA also evaluated the risk to consumers from inhalation and dermal exposures, and to bystanders, from inhalation exposures, by comparing the estimated exposures to acute human health hazards. 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 2.4.1.30. EPA assumed that consumers or bystanders would not use PPE and that all exposures would be acute rather than chronic.

For consumer users and bystanders, risks identified for acute exposures were indicated for some conditions of use. For consumers, medium and high intensity acute inhalation and dermal exposure scenarios indicated risk. Conditions of use that indicated risks following acute exposures to consumer users (for inhalation and dermal exposure) also indicated risks to bystanders (primarily for inhalation exposures only). One scenario, dry cleaning solvent, presented risks for bystanders in the dermal scenario. Some consumer conditions of use did not indicate risks for consumer or bystanders. EPA's estimates for consumer and bystander risks for each consumer use exposure scenario are presented in Section 4.2.4 and summarized in Table 4-113 in Section 4.5.2.

Uncertainties

Key assumptions and uncertainties in the environmental risk estimation include the uncertainty around modeled releases that have surface water concentrations greater than the highest concentration of concern for algae. Data were reasonably available for three algal species and may not represent the most sensitive species at a given site. 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. Assumptions and key sources of uncertainty for consumer exposure are detailed in Section 2.4.2.3 for consumer products, Section 2.4.2.4 for consumer articles, and Section 2.4.2.6 for overarching uncertainties.

Potentially Exposed and Susceptible Subpopulations

TSCA sec. 6(b)(4) requires that EPA evaluate risk to relevant potentially exposed or susceptible subpopulations (PESS). TSCA sec. 3(12) states that "[*t]he 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*."

In developing the risk evaluation, EPA analyzed the 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 a chemical. For consideration of the most highly exposed groups, EPA considered PCE exposures among both workers using PCE and ONUs in the vicinity of PCE use to be higher than the exposures experienced by the general population. Consumer users and bystanders are also expected to be more highly exposed than the general population. Potentially susceptible subpopulations include the developing fetus (and by extension, women of childbearing age) as well as those with pre-existing health conditions, higher body fat content, or particular genetic polymorphisms.

Aggregate and Sentinel Exposures

Section 6 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). Exposures to PCE 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 utilize additivity of exposure pathways at this time 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.

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 this risk evaluation, the EPA considered sentinel exposure the highest exposure given the details of the conditions of use and the potential exposure scenarios.

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); 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 risk determination is discussed in Section 5.1.

Environmental Risks

EPA evaluated environmental exposures for aquatic organisms and determined whether any risks are unreasonable. The drivers for EPA's draft determination of unreasonable risks to aquatic organisms are immobilization from acute exposure, growth effects from chronic exposure, and mortality to algae. Algae was assessed separately and not incorporated into acute or chronic COCs, because durations normally considered acute for other species (e.g., 48, 72 hours) can encompass several generations of algae. EPA estimated site-specific surface water concentrations for discharges using upper and lower bounds for the range of predicted surface water concentrations. For the percentage of the chemical

removed from wastewater during treatment before discharge to a body of water, EPA estimated 80% removal of PCE from indirect discharging facilities and estimated 0% removal of PCE for direct releases to surface water. PCE has low bioaccumulation potential and moderate potential to accumulate in wastewater biosolids, soil, or sediment.

For risks to the environment, EPA preliminarily determined that the conditions of use for PCE that present unreasonable risks are processing as a reactant/intermediate, recycling, use as a processing aid in petroleum production, and disposal. A full description of EPA's draft determination for each condition of use is in Section 5.3.

Risks of Injury to Health

EPA's draft determination of unreasonable risk for specific conditions of use of PCE listed below are based on health risks to workers, occupational non-users, consumers, or bystanders from consumer use. As described below, risks to general population were not evaluated. PCE has a large database of human health toxicity data. For each hazard domain there are several endpoints, and often a single endpoint was examined by multiple studies. The non-cancer effects selected for risk estimation were neurotoxicity (i.e., increased latencies for pattern reversal visual-evoked potentials) from acute exposure and multiple effects including CNS, kidney, liver, immune system and developmental toxicity from repeated and chronic exposures. The evaluation of cancer includes estimates of risk of lung and liver tumors.

Risk to the General Population

General population exposures to PCE may occur from industrial and/or commercial uses; industrial releases to air, water or land; and other conditions of use. As part of the problem formulation for PCE, EPA found those exposure pathways are covered by other statutes and consist of: the ambient air pathway (i.e., PCE is listed as a hazardous air pollutant (HAP) in the Clean Air Act (CAA)), the drinking water pathway (i.e., National Primary Drinking Water Regulations (NPDWRs) are promulgated for PCE under the Safe Drinking Water Act), ambient water pathways (i.e., PCE is a priority pollutant with recommended water quality criteria for protection of human health under the CWA), and disposal pathways (RCRA and SDWA regulations minimize further environmental exposure and associated risks related to the disposal of PCE). As described in the problem formulation for PCE, other environmental statutes administered by EPA adequately assess and effectively manage these exposures. EPA believes that the TSCA risk evaluation should focus on those exposure pathways associated with TSCA conditions of use that are not subject to the regulatory regimes discussed above because those pathways are likely to represent the greatest areas of concern to EPA. Therefore, EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population.

Risk to Workers

EPA evaluated workers' acute and chronic inhalation and dermal exposures for cancer and non-cancer risks and determined whether any risks are unreasonable. The drivers for EPA's draft determination of unreasonable risk for workers are neurotoxicity from acute and chronic inhalation exposures, neurotoxicity from chronic dermal exposures, and cancer resulting from chronic inhalation and dermal exposures.

The determinations reflect the effects associated with the occupational exposures to PCE and incorporate consideration of assumed PPE (frequently estimated to be a respirator of APF 10, 25, or 50 and gloves with PF 5, 10, or 20). Some conditions of use did not assume the use of respiratory PPE. For workers, EPA determined that all applicable conditions of use for PCE presented unreasonable risks, except for distribution in commerce, the industrial use of lubricants and greases (e.g., penetrating

lubricants, cutting tool coolants), the industrial use of laboratory chemicals, the commercial use of lubricants and greases (e.g., penetrating lubricants, cutting tool coolants), and the commercial use of laboratory chemicals. A full description of EPA's draft determination of unreasonable risk for each condition of use is in Section 5.3.

Risk to Occupational Non-Users (ONUs)

EPA evaluated ONU acute and chronic inhalation exposures for cancer and non-cancer risks and determined whether any risks are unreasonable. The drivers for EPA's draft determination of unreasonable risks to ONUs are neurotoxicity from acute and chronic inhalation, and cancer resulting from chronic inhalation exposure. The draft determinations reflect the effects associated with the occupational exposures to PCE and the assumed absence of PPE for ONUs. For dermal exposures, because ONUs are not expected to be dermally exposed to PCE, dermal risks to ONUs were not evaluated. For inhalation exposures, EPA, where possible, used monitoring or modeling information to estimate ONU exposures and to describe the risks separately from workers directly exposed. For some conditions of use, EPA did not separately calculate risk estimates for ONUs and workers. For these conditions of use, there is uncertainty in the ONU risk estimates since the data or modeling did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency risk estimate when determining ONU risk for those conditions of use for which ONU exposures were not separately estimated. EPA determined that most applicable conditions of use do not present unreasonable risks. Estimated numbers of occupational non-users are in Section 2.4.1.2.

Risk to Consumers

EPA evaluated consumer acute inhalation and dermal exposures for non-cancer risks and determined whether any risks are unreasonable. The driver for EPA's draft determination of unreasonable risk is neurotoxicity from acute inhalation and dermal exposure. Generally, risks for consumers were indicated by acute inhalation and dermal exposure at low, medium, and high intensity use.

For consumers, EPA determined that most consumer conditions of use present unreasonable risks, except for use of livestock grooming adhesive, aerosol paints and coatings, and metallic overglaze.

A full description of EPA's draft determination for each condition of use is in Section 5.3.

Risk to Bystanders (from consumer uses)

EPA evaluated bystander acute inhalation exposures for non-cancer risks and determined whether any risks are unreasonable. The driver for EPA's determination of unreasonable risk are neurotoxicity from acute inhalation exposure. Generally, risks for bystanders were indicated by acute inhalation exposure scenarios at low, medium, and high intensity use. Because bystanders are not expected to be dermally exposed to PCE, dermal non-cancer risks to bystanders were not evaluated. For bystanders, EPA determined that most consumer conditions of use present unreasonable risks, except for use of dry cleaned articles, arts and crafts adhesive, livestock grooming adhesive, caulks and sealants, aerosol coatings and primers, liquid rust primer and sealant, and metallic overglaze.

A full description of EPA's draft determination for each condition of use is in Section 5.3.

Summary of Risk Determinations

EPA has preliminarily determined that the following conditions of use of PCE do not present an unreasonable risk of injury under any scenarios. The details of these determinations are presented in Table 5-1 in Section 5.2.

Conditions of Use that Do Not Present an Unreasonable Risk
• Distribution in commerce • Industrial use of lubricants and greases (e.g., penetrating lubricants, cutting tool coolants) • Industrial use of laboratory chemicals • Commercial use of lubricants and greases (e.g., penetrating lubricants, cutting tool coolants) • Commercial use of laboratory chemicals • Consumer use of livestock grooming adhesive • Consumer use of aerosol coating and primers • Consumer use of metallic overglaze

EPA has preliminarily determined that the following conditions of use of PCE present an unreasonable risk to the environment or unreasonable risk of injury to health to workers (including, in some cases, occupational non-users) or to consumers (including, in some cases, bystanders). The details of these determinations are presented in Table 5-1 in Section 5.2.

Manufacturing that Presents an Unreasonable Risk
• Domestic Manufacture • Import (includes repackaging and loading/unloading)

Processing that Presents an Unreasonable Risk
• Processing as a reactant/intermediate • Incorporation into formulation, mixture or reaction product (cleaning and degreasing products) • Incorporation into formulation, mixture or reaction product (adhesive and sealant products) • Incorporation into formulation, mixture or reaction product (paint and coating products) • Incorporation into formulation, mixture or reaction product (other chemical products and preparations) • Repackaging • Recycling

Industrial Uses that Present an Unreasonable Risk
• As a solvent for batch vapor degreasing (open-top) • As a solvent for batch vapor degreasing (closed-loop) • As a solvent for in-line vapor degreasing (conveyorized) • As a solvent for in-line vapor degreasing (web-cleaner) • As a solvent for cold cleaning • As a solvent for aerosol spray degreaser/cleaner • In dry cleaning and spot cleaning (Post-2006 dry cleaning) • In dry cleaning and spot cleaning (4th/5th Gen only dry cleaning)

- As a lubricants and grease (aerosol lubricants)
- As a solvent-based adhesive and sealant
- As a solvent-based paint and coating
- As a maskant for chemical milling
- As a processing aids for pesticide, fertilizer and other agricultural chemical manufacturing
- As a processing aids specific to petroleum production (catalyst regeneration in petrochemical manufacturing)
- In textile processing (spot cleaning)
- In textile processing (other)
- In wood furniture manufacturing
- As a laboratory chemical
- In foundry applications

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Commercial Uses that Present an Unreasonable Risk

- As a cleaner and degreaser (wipe cleaning)
- As a cleaner and degreaser (other spot cleaning/spot removers (including carpet cleaning))
- As a cleaner and degreaser (mold release)
- In dry cleaning and spot cleaning (Post-2006 dry cleaning)
- In dry cleaning and spot cleaning (4th/5th Gen only dry cleaning)
- In automotive care products (e.g., engine degreaser and brake cleaner)
- As an aerosol cleaner
- As a non-aerosol cleaner
- As a lubricant and grease (aerosol lubricants)
- As a light repair adhesive
- As a solvent-based paint and coating
- In carpet cleaning
- In metal (e.g., stainless steel) and stone polishes
- In inks and ink removal products (printing)
- In inks and ink removal products (photocopying)
- In welding
- In photographic film
- In mold cleaning, release and protectant products

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Consumer Uses that Present an Unreasonable Risk

- As a cleaner and degreaser (other)
- In dry cleaning
- In automotive care products (brake cleaner)
- In automotive care products (parts cleaner)
- In aerosol cleaner (vandalism mark and stain remover, mold cleaner, weld splatter protectant)
- In non-aerosol cleaner (e.g., marble and stone polish)
- In lubricants and greases (cutting fluid)
- In lubricants and greases (lubricants and penetrating Oils)
- In adhesives for arts and crafts (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant)
- In adhesives for arts and crafts (column adhesive, caulk and sealant)

- In solvent-based paints and coatings (outdoor water shield (liquid))
- In rust primer and sealant (liquid)
- In metal (e.g., stainless steel) and stone polishes
- In inks and ink removal products; welding; mold cleaning, release and protectant products

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Disposal that Presents an Unreasonable Risk
• Disposal

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1 INTRODUCTION

This document presents for comment the draft risk evaluation for PCE 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, the Nation's primary chemicals management law in June 2016.

The Agency published the Scope of the Risk Evaluation for PCE in June 2017 (U.S. EPA 2017i), and the problem formulation in June, 2018 (U.S. EPA 2018d). These 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 (U.S. EPA 2014c). The problem formulation identified conditions of use within the scope of the risk evaluation 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 aquatic receptors exposed via surface water, workers, and consumers. The conclusions of the problem formulation were that risk would not be evaluated for sediment, soil and land-applied biosolid pathways leading to exposure to terrestrial and aquatic organisms. Risks would not be evaluated for land-applied biosolids because PCE is currently being addressed in the Clean Water Act (CWA) regulatory analytical process. EPA also excluded from risk evaluation ambient air, drinking water, land disposal, ambient water, and waste incineration pathways leading to exposures to the general population and terrestrial organisms since those pathways are regulated under other environmental statutes administered by EPA which adequately assess and effectively manage exposure**s**. EPA received comments on the published problem formulation for PCE and has considered the comments specific to PCE, as well as more general comments regarding EPA's chemical risk evaluation approach for developing the draft risk evaluations for the first 10 chemicals EPA is evaluating.

In this draft risk evaluation, Section 1 presents the basic physical-chemical characteristics of PCE, 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 problem formulation. This section also includes a discussion of the systematic review process utilized in this draft risk evaluation. Section 2 provides a discussion and analysis of the exposures, both human health and environmental, that can be expected based on the conditions of use for PCE. Section 3 discusses environmental and health hazards of PCE. 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 draft risk evaluation. Section 5 presents EPA's proposed 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)).

As per EPA's final rule, (U.S. EPA 2017c), this draft risk evaluation will be subject to both public comment and peer review, which are distinct but related processes. EPA is providing 60 days for public comment on any and all aspects of this draft 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 PCE. 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 will be conducted in accordance with EPA's regulatory procedures for chemical risk evaluations, including using the EPA Peer Review Handbook (U.S. EPA 2015a) and other methods consistent with section 26 of TSCA (*See* 40 CFR 702.45). As explained in the Risk Evaluation Rule (U.S. EPA 2017c), the purpose of peer review is for the independent review of the science underlying the risk assessment. Peer review will therefore address 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 (U.S. EPA 2017c), 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 believes peer reviewers will be most effective in this role if they receive the benefit of public comments on draft risk evaluations prior to peer review. The final risk evaluation may change 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 will respond to public and peer review comments received on the draft risk evaluation and will explain changes made to the draft risk evaluation for PCE in response to those comments in the final risk evaluation.

EPA 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 PCE. Thus, in addition to any new comments on the draft risk evaluation, the public should re-submit or clearly identify at this point any previously filed comments, modified as appropriate, that are relevant to this risk evaluation and that the submitter feels have not been addressed. EPA does not intend to further respond to comments submitted prior to the publication of this draft risk evaluation unless they are clearly identified in comments on this draft risk evaluation.

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 intends to consider. For scope development, EPA considered the measured or estimated physical-chemical properties set forth in Table 1-1; EPA found no additional information during problem formulation or risk evaluation that would change these values.

Table 1-1 Physical and Chemical Properties of PCE

Property	Value[a]	References	Data Quality Rating
Molecular formula	C_2Cl_4		
Molecular weight	165.833		
Physical form	Colorless liquid; chloroform-like odor	Lewis (2007); NIOSH (2005); U.S. Coast Guard (1984)	High
Melting point	-22.3°C	Lide (2007)	High
Boiling point	121.3°C	Lide (2007)	High
Density	1.623 g/cm^3 at 20°C	Lide (2007)	High
Vapor pressure	18.5 mmHg at 25°C	Riddick et al. (1985)	High
Vapor density	5.83 (relative to air)	(Lewis 1992)	High
Water solubility	206 mg/L at 20°C	Horvath (1982)	High
Octanol:water partition coefficient (K_{OW})	3.40	Hansch et al. (1995)	High
Henry's Law constant	0.0177 atm-m^3/mole	Gossett (1987)	High
Flash point	Not applicable	Nfpa (2010)	High
Autoflammability	Not readily available		
Viscosity	0.839 cP at 25°C	Hickman (2000)	High
Refractive index	1.4775	Lide (2007)	High
Dielectric constant	2.30 at 25°C	(Lange and Dean 1985)	High

[a] Measured unless otherwise noted.

1.2 Uses and Production Volume

The uses of PCE include the production of fluorinated compounds, dry cleaning and vapor degreasing, as well as a number of less produced uses. Nearly 65% of the production volume of PCE is used as an intermediate in industrial gas manufacturing, more specifically to produce fluorinated compounds, such as hydrofluorocarbons (HFCs) and hydrochlorofluorocarbons (HCFCs) (NTP 2014) (Icis 2011). HFCs 134a and 125 are alternatives to chlorofluorocarbons (CFCs) and HCFCs, which are ozone depleting substances (ODSs), and the subject of a phase-out (https://www.epa.gov/ods-phaseout). HCFCs are transitional substances in the phase-out of ODSs (Icis 2011), (Fay 2017). Previously, PCE was widely used to manufacture CFCs (especially trichlorotrifluoroethane (CFC-113)) until production and importation of CFCs for most uses were phased out in the United States by regulations implementing the Montreal Protocol (40 CFR part 82). A relatively small amount of CFC-113 is still produced for exempted uses (van Hook 2017).

The second largest use of PCE (~15%) is as a solvent in dry cleaning facilities (NTP 2014). PCE is non-flammable and effectively dissolves fats, greases, waxes and oils, without harming natural or human-made fibers. These properties enabled it to replace traditional petroleum solvents (ATSDR 2014; Dow Chemical Co 2008; Tirsell 2000). The demand for PCE dry cleaning solvents has steadily declined as a result of the improved efficiency of dry cleaning equipment, increased chemical recycling and the popularity of wash-and-wear fabrics that eliminate the need for dry cleaning (ATSDR 2019). PCE is also used in dry cleaning detergent and dry cleaning sizing.

Approximately 60% of dry cleaning machines now use PCE as a solvent (DLI/NCA 2017). In 1991, EPA estimated that 83% of all dry cleaning facilities used PCE as solvent (U.S. EPA 1991). In 2008, the Halogenated Solvents Industry Association (HSIA) estimated that 70% of dry cleaners used PCE as dry cleaning solvent (Graul 2017). Similarly, in 2011, King County, WA conducted a profile of the dry cleaning industry and found that 69% of respondents (105 of the 152 respondents) used PCE in their primary machine (Whittaker and Johanson 2011). Hence, there appears to be a trend towards alternatives to PCE in dry cleaning. According to the dry cleaning industry, a majority of new PCE dry cleaning machines are sold in locations where "local fire codes preclude the use of Class III combustible alternative solvents or [where] the nature of the operation demands the use of PCE" (DLI/NCA 2017).

The third most prevalent use of PCE (~10%) is as a vapor degreasing solvent (NTP 2014). PCE can be used to dissolve many organic compounds, select inorganic compounds and high-melting pitches and waxes making it ideal for cleaning contaminated metal parts and other fabricated materials (ATSDR 2019). It is a very good solvent for greases, fats, waxes, oils, bitumen, tar and many natural and synthetic resins for use in chemical cleaning systems, degreasing light and heavy metals, degreasing pelts and leather (tanning), extraction of animal and vegetable fats and oils and textile dyeing (solvent for dye baths) (Stoye 2000). PCE is also used in cold cleaning, which is similar to vapor degreasing, except that cold cleaning does not require the solvent to be heated to its boiling point in order to clean a given component. Vapor degreasing and cold cleaning scenarios may include a range of open-top or closed systems, conveyorized/enclosed/inline systems, spray wands, dip containers and wipes.

PCE has many other uses, which collectively constitute ~10% of the production volume. EPA's search of safety data sheets, government databases and other sources found over 375 products containing PCE. These uses include (but are not limited to):

- Adhesives
- Aerosol degreasing
- Brake cleaner
- Laboratories
- Lubricants
- Mold cleaners, releases and protectants
- Oil refining
- Sealants
- Stainless steel polish
- Tire buffers and cleaners
- Vandal mark removers

Many of these uses include consumer products, such as adhesives (arts and crafts, as well as light repairs), aerosol degreasing, brake cleaners, aerosol lubricants, sealants, sealants for gun ammunition, stone polish, stainless steel polish and wipe cleaners. The uses of PCE in consumer adhesives and brake cleaners are especially prevalent; EPA has found 16 consumer adhesive products and 14 consumer brake cleaners containing PCE (see (U.S. EPA 2017g)).

The Chemical Data Reporting (CDR) Rule under TSCA requires U.S. manufacturers and importers to provide EPA with information on the chemicals they manufacture or import into the United States. 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 at each of their sites during the 2015 calendar year were required to report information under the CDR rule (U.S. EPA 2016d).

The 2016 CDR reporting data for PCE are provided in Table 1-2 from EPA's CDR database (U.S. EPA 2016c). This information has not changed from that provided in the scope document.

Table 1-2 Production Volume of PCE in CDR Reporting Period (2012 to 2015) [a]

Reporting Year	2012	2013	2014	2015
Total Aggregate Production Volume (lbs)	387,623,401	391,403,540	355,305,850	324,240,744

[a] The CDR data for the 2016 reporting period is available via ChemView (https://java.epa.gov/chemview) (ChemView 2019). The CDR data presented in the problem formulation is more specific than currently available in ChemView.

1.3 Regulatory and Assessment History

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

Federal Laws and Regulations

PCE is subject to federal statutes or regulations, other than TSCA, 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.

State Laws and Regulations

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

Laws and Regulations in Other Countries and International Treaties or Agreements

PCE is subject to statutes or regulations in countries other than the United States. A summary of these laws and regulations is provided in Appendix A.

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. EPA found no additional assessments beyond those listed in the Problem Formulation document.

Table 1-3 Assessment History of PCE

Authoring Organization	Assessment
EPA Assessments	

Authoring Organization	Assessment
Integrated Risk Information System (IRIS)	Toxicological Review of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) (U.S. EPA 2012e)
Office of Air Quality Planning and Standards (OAQPS)	Perchloroethylene Dry Cleaners Refined Human Health Risk Characterization (U.S. EPA 2005b)
National Center for Environmental Assessment (NCEA)	Sources, Emission and Exposure for Trichloroethylene (TCE) and Related Chemicals (U.S. EPA 2001)
Office of Air Toxics	Tetrachloroethylene (PCE, Perchloroethylene); 127-18-4 (U.S. EPA 2000)
Office of Pesticides and Toxic Substances (now, Office of Chemical Safety and Pollution Prevention [OCSPP])	Occupational Exposure and Environmental Release Assessment of Tetrachloroethylene (U.S. EPA 1985b)
Office of Health and Environmental Assessment	Final Health Effects Criteria Document for Tetrachloroethylene (U.S. EPA 1985a)
Office of Water (OW)	Update of Human Health Ambient Water Quality Criteria: Tetrachloroethylene (Perchloroethylene) 127-18-4 (U.S. EPA 2015b)
Office of Water (OW)	Ambient Water Quality Criteria for Tetrachloroethylene (U.S. EPA 1980)
Other U.S.-Based Organizations	
California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (OEHHA), Air Toxics Hot Spots Program	Perchloroethylene Inhalation Cancer Unit Risk Factor (OEHHA 2016)
Agency for Toxic Substances and Disease Registry (ATSDR)	Toxicological Profile for Tetrachloroethylene (PERC) (ATSDR 2019)
National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL Committee)	Tetrachloroethylene (NAC/AEGL 2009)
California Environmental Protection Agency, OEHHA, Pesticide and Environmental Toxicology Section	Public Health Goal for Tetrachloroethylene in Drinking Water (OEHHA 2001)
National Toxicology Program (NTP)	Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene); (CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (NTP 1986a)
International	

Authoring Organization	Assessment
International Agency for Research on Cancer (IARC)	IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Tetrachloroethylene (IARC 2014)
European Union (EU), Scientific Committee on Health and Environmental Risks (SCHER)	SCHER, Scientific Opinion on the Risk Assessment Report on Tetrachloroethylene, Human Health Part, CAS No.: 127-18-4, 12 (Scher 2008)
World Health Organization (WHO)	Concise International Chemical Assessment Document 68; Tetrachloroethylene (WHO 2006a)
EU, European Chemicals Bureau (ECB)	EU Risk Assessment Report; Tetrachloroethylene, Part 1 - environment (ECB 2005)
National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Australia	Tetrachloroethylene; Priority Existing Chemical Assessment Report No. 15 (NICNAS 2001)

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 (COUs) 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.” The conditions of use are described below in Table 1-4. No additional information was received by EPA following the publication of the problem formulation that would update or otherwise require changes to the use document conditions of use (U.S. EPA 2018d) Table 2-4) or the life cycle diagram as presented in the problem formulation (U.S. EPA 2018d). The life cycle diagram is presented in Figure 1-1.

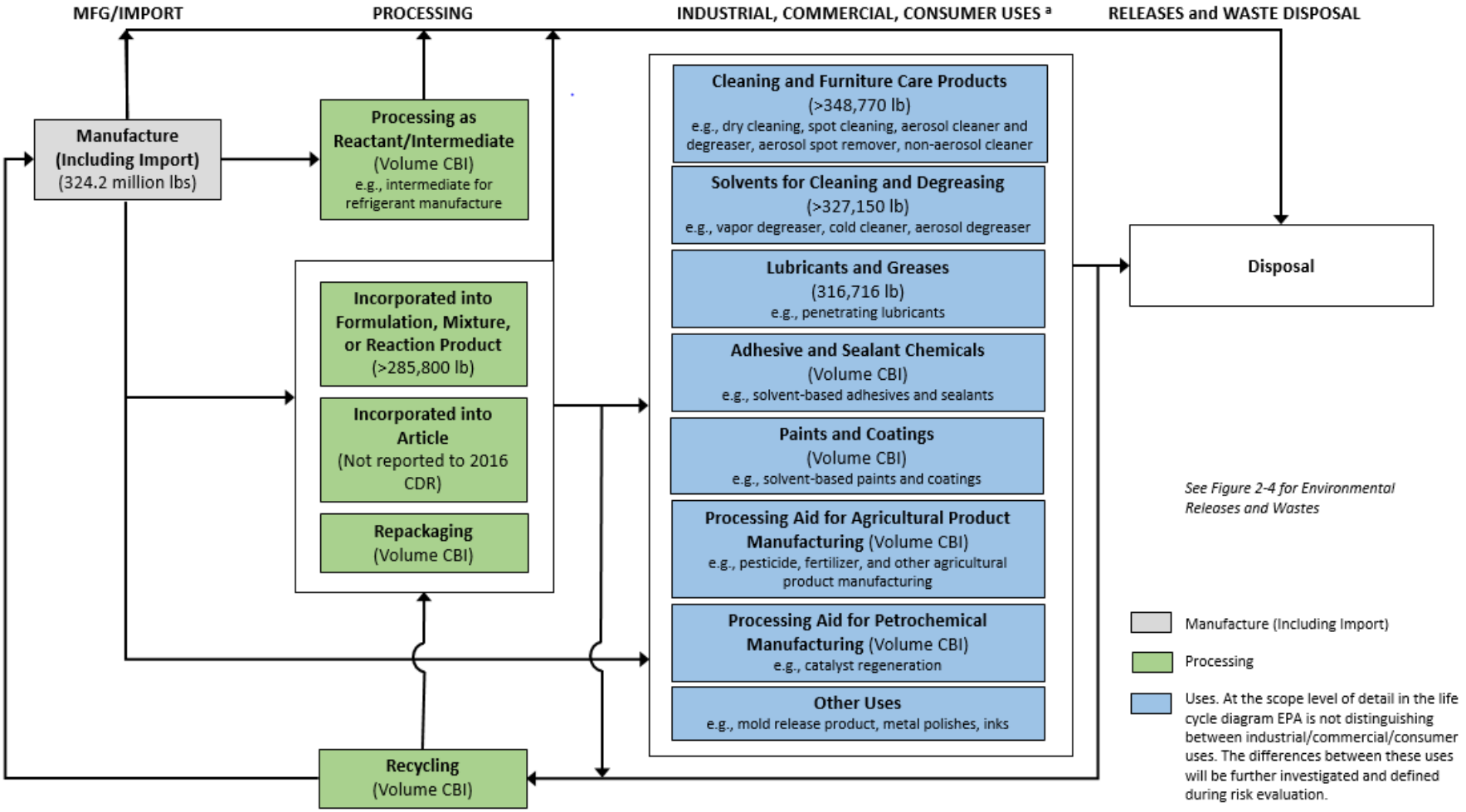


Figure 1-1. PCE Life Cycle Diagram

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 or commercial) and disposal. The production volumes shown are for reporting year 2015 from the 2016 CDR reporting period (Table 1-2) (U.S. EPA 2016c). Activities related to distribution (e.g., loading, unloading) will be considered throughout the PCE 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
Manufacture	Domestic manufacture	Domestic manufacture	(U.S. EPA 2016c)
	Import	Import	(U.S. EPA 2016c)
Processing	Processing as a reactant or intermediate	Intermediate in industrial gas manufacturing	(U.S. EPA 2016c); (U.S. EPA 2017g); (Krock 2017a); (Krock 2017b); (Cooper 2017); (Fay 2017)
		Intermediate in basic organic chemical manufacturing	(U.S. EPA 2016b), (U.S. EPA 2017g);
		Intermediate in petroleum refineries	(U.S. EPA 2016b); (U.S. EPA 2017g);(Cooper 2017)
		Residual or byproduct	(Krock 2017a); (Krock 2017b);
	Incorporated into formulation, mixture or reaction product	Cleaning and degreasing products	(U.S. EPA 2016b); (Rudnick 2017a), (Rudnick 2017b)
		Adhesive and sealant products	(U.S. EPA 2016b)
		Paint and coating products	(U.S. EPA 2016b)
		Other chemical products and preparations	(U.S. EPA 2016b)
	Repackaging	Solvent for cleaning or degreasing	(U.S. EPA 2016b)
		Intermediate	(U.S. EPA 2016b)
	Recycling	Recycling	(U.S. EPA 2016b)
Distribution in commerce	Distribution	Distribution	(U.S. EPA 2017g)
Industrial use	Solvents (for cleaning or degreasing)	Solvents and/or Degreasers (cold, aerosol spray or vapor degreaser; not specified in comment)	(U.S. EPA 2017g); (Holmes 2017); (Tatman 2017)
		Batch vapor degreaser (e.g., open-top, closed-loop)	(U.S. EPA 1985b); (Riegle 2017); (HSIA 2018b)
		In-line vapor degreaser (e.g., conveyorized, web cleaner)	(U.S. EPA 1985b); (Dowell 2017)
	Solvents (for cleaning or degreasing)	Cold cleaner	(U.S. EPA 2017g); (Rudnick 2017a), (Rudnick 2017b)
		Aerosol spray degreaser/cleaner	(U.S. EPA 2017g); (U.S. EPA 2017g); (Sass 2017); (Rudnick 2017a), (Rudnick 2017b)

Life Cycle Stage	Category [a]	Subcategory [b]	References
		Dry cleaning solvent	(U.S. EPA 2017g); (U.S. EPA 2006a)
		Spot cleaner	(U.S. EPA 2017g); (Sass 2017)
	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	(U.S. EPA 2016b); (U.S. EPA 2017g); (HSIA 2018b); (Tatman 2017); (HSIA 2018b); (Tatman 2017)
	Adhesive and sealant chemicals	Solvent-based adhesives and sealants	(U.S. EPA 2016b); (U.S. EPA 2017g); (U.S. EPA 2017g); (Sass 2017); (Riegle 2017); (Holmes 2017); (HSIA 2018b)
	Paints and coatings including paint and coating removers	Solvent-based paints and coatings, including for chemical milling	(U.S. EPA 2016b); (U.S. EPA 2017g); (Sass 2017); (Riegle 2017); (Davis 2017); (HSIA 2018b); (U.S. DOD 2017)
	Processing aids, not otherwise listed	Pesticide, fertilizer and other agricultural chemical manufacturing	(U.S. EPA 2016b)
	Processing aids, specific to petroleum production	Catalyst regeneration in petrochemical manufacturing	(U.S. EPA 2016b); (U.S. EPA 2017g); (Dow Chem 2008); (Cooper 2017); (HSIA 2018b)
	Other uses	Textile processing	(U.S. EPA 2017g)
		Wood furniture manufacturing	(U.S. EPA 2017g)
		Laboratory chemicals	(U.S. EPA 2017g); (Riegle 2017)
		Foundry applications	(U.S. EPA 2017g)
Commercial/consumer use	Cleaning and furniture care products	Cleaners and degreasers (other)	(U.S. EPA 2017g); (Sass 2017); (Rudnick 2017a), (Rudnick 2017b); (Holmes 2017); (McCormick 2017); (HSIA 2018b); (Tatman 2017)
		Dry cleaning solvent	(U.S. EPA 2017g);(U.S. EPA 2006a); (DLI/NCA 2017); (Sass 2017)

Life Cycle Stage	Category [a]	Subcategory [b]	References
		Spot cleaner	(U.S. EPA 2017g); (U.S. EPA 2006a); (Sass 2017)
		Automotive care products (e.g., engine degreaser and brake cleaner)	U.S. EPA (2016d), (U.S. EPA 2017g); (Rudnick 2017a), (Rudnick 2017b); (HSIA 2018b)
		Aerosol cleaner	(U.S. EPA 2017g); (Sass 2017)
		Non-aerosol cleaner	(U.S. EPA 2017g); (Sass 2017)
	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	(U.S. EPA 2016b); (U.S. EPA 2017g); (HSIA 2018b); (Tatman 2017)
	Adhesives and sealant chemicals	Adhesives for arts and crafts	(U.S. EPA 2016b); (U.S. EPA 2017g); (Sass 2017)
		Light repair adhesives	(U.S. EPA 2016b); (U.S. EPA 2017g)
	Paints and coatings	Solvent-based paints and coatings	(U.S. EPA 2016b); (U.S. EPA 2017g); (Sass 2017); (Davis 2017); (HSIA 2018b)
	Other uses	Carpet cleaning	(U.S. EPA 2017g); (Sass 2017)
		Laboratory chemicals	(U.S. EPA 2017g)
		Metal (e.g., stainless steel) and stone polishes	(U.S. EPA 2017g)
		Inks and ink removal products	(U.S. EPA 2017g)
		Welding	(U.S. EPA 2017g)
		Photographic film	(U.S. EPA 2017g)
		Mold cleaning, release and protectant products	(U.S. EPA 2017g); (Rudnick 2017a), (Rudnick 2017b)
Disposal	Disposal	Industrial pre-treatment	(U.S. EPA 2017g)
		Industrial wastewater treatment	
		Publicly owned treatment works (POTW)	
		Underground injection	
		Municipal landfill	
		Hazardous landfill	
		Other land disposal	

Life Cycle Stage	Category [a]	Subcategory [b]	References
		Municipal waste incinerator	
		Hazardous waste incinerator	
		Off-site waste transfer	
		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 for PCE in consumer, industrial, and/or commercial settings.
[b] These subcategories reflect more specific uses of PCE.

1.4.2 Conceptual Models

The conceptual models for this risk evaluation are shown in Figure 1-2, Figure 1-3, and Figure 1-4. EPA considered the potential for hazards to human health and the environment resulting from exposure pathways outlined in the preliminary conceptual models of the PCE scope document (U.S. EPA 2017i). These conceptual models considered potential exposures resulting from industrial and commercial activities, consumer activities and uses and environmental releases and wastes. The problem formulation documents refined the initial conceptual models and analysis plans that were provided in the PCE scope document (U.S. EPA 2018d).

For the purpose of this evaluation, EPA considered workers and occupational non-users, which includes men and women of reproductive age (Figure 1-2). Consumer exposure was assessed for various pathways for users age 11 and older along with bystanders of all ages (Figure 1-3).

The potential pathways that were determined to be included in the risk evaluation but not to warrant further analysis in this draft risk evaluation were: exposure to both humans and ecological organisms due to land application of biosolids following wastewater treatment leading to exposure terrestrial organisms. In the problem formulation, EPA determined that risks would not be evaluated for land-applied biosolids because PCE is currently being addressed in the Clean Water Act (CWA) regulatory analytical process. Also, as outlined in Section 1.3 and Appendix A, PCE is regulated in various environmental media.

The potential pathways that were determined to be included in the risk evaluation and further analyzed include:

- Exposure to aquatic species (e.g. aquatic plants) via contaminated surface water.
- Inhalation and dermal exposures to workers and consumer users, and inhalation exposures to ONUs and consumer bystanders, from industrial/commercial activities and consumer activities.
- Inhalation and dermal exposures to workers and inhalation exposures to ONUs from waste handling, treatment and disposal.

Review and evaluation of reasonably available information on PCE confirmed the preliminary conclusions in the problem formulation and as a result, the EPA confirms further analysis of the pathways outlined in the conceptual models. The conceptual models for this risk evaluation are shown in Figure 1-2, Figure 1-3, and Figure 1-4.

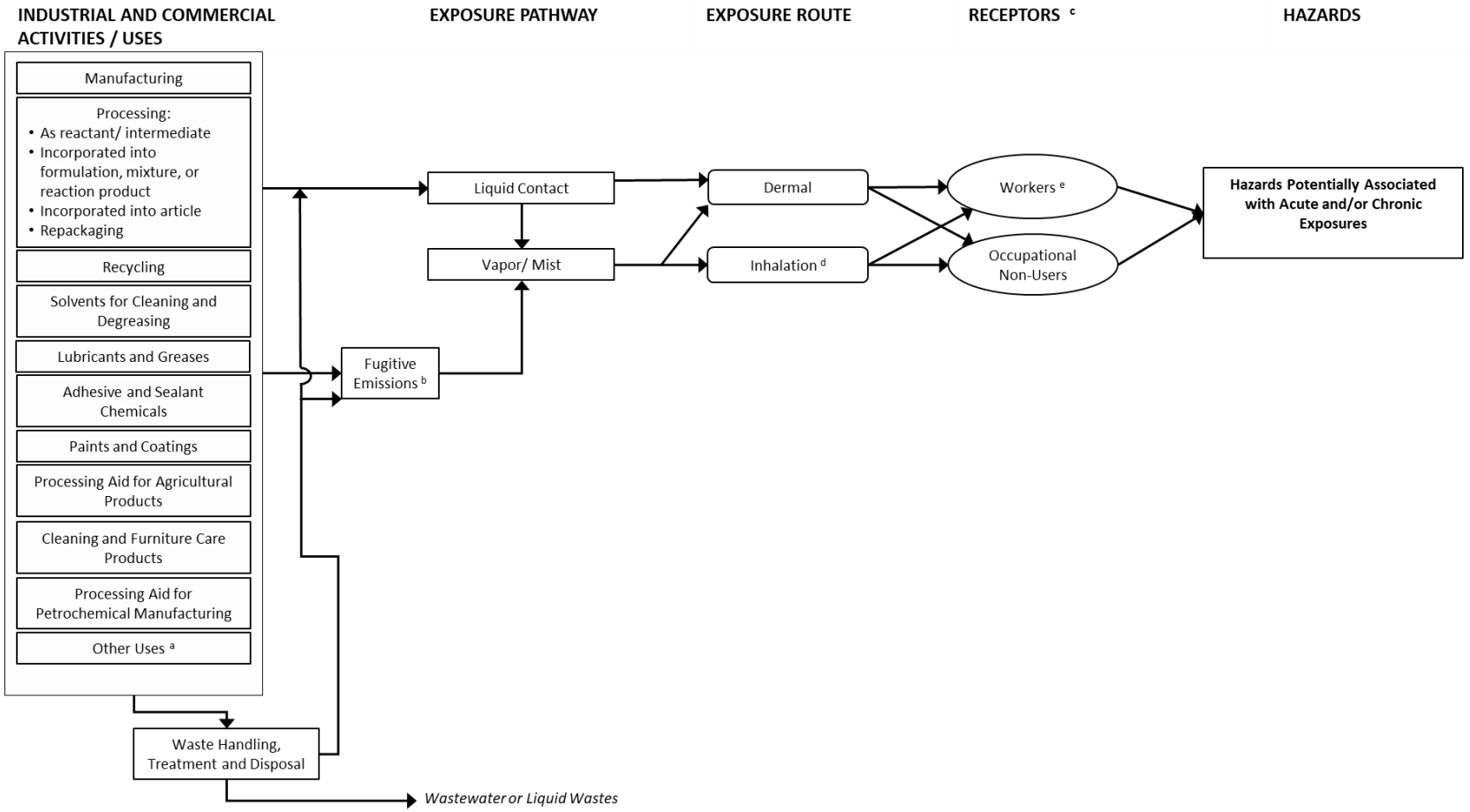


Figure 1-2. PCE Conceptual Model for Industrial and Commercial Activities and Uses: Potential Exposures and Hazards

The conceptual model presents the exposure pathways, exposure routes and hazards to human receptors from industrial and commercial activities and uses of PCE.

[a] Some products are used in both commercial and consumer applications such adhesives and sealants. Additional uses of PCE 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] Receptors include potentially exposed or susceptible subpopulations.

[d] Oral exposure may occur through mists that deposit in the upper respiratory tract however, based on physical chemical properties, mists of PCE will likely be rapidly absorbed in the respiratory tract or evaporate and will be considered as an inhalation exposure.

[e] When data and information are available to support the analysis, EPA also considers the effect that engineering controls and/or personal protective equipment have on occupational exposure levels

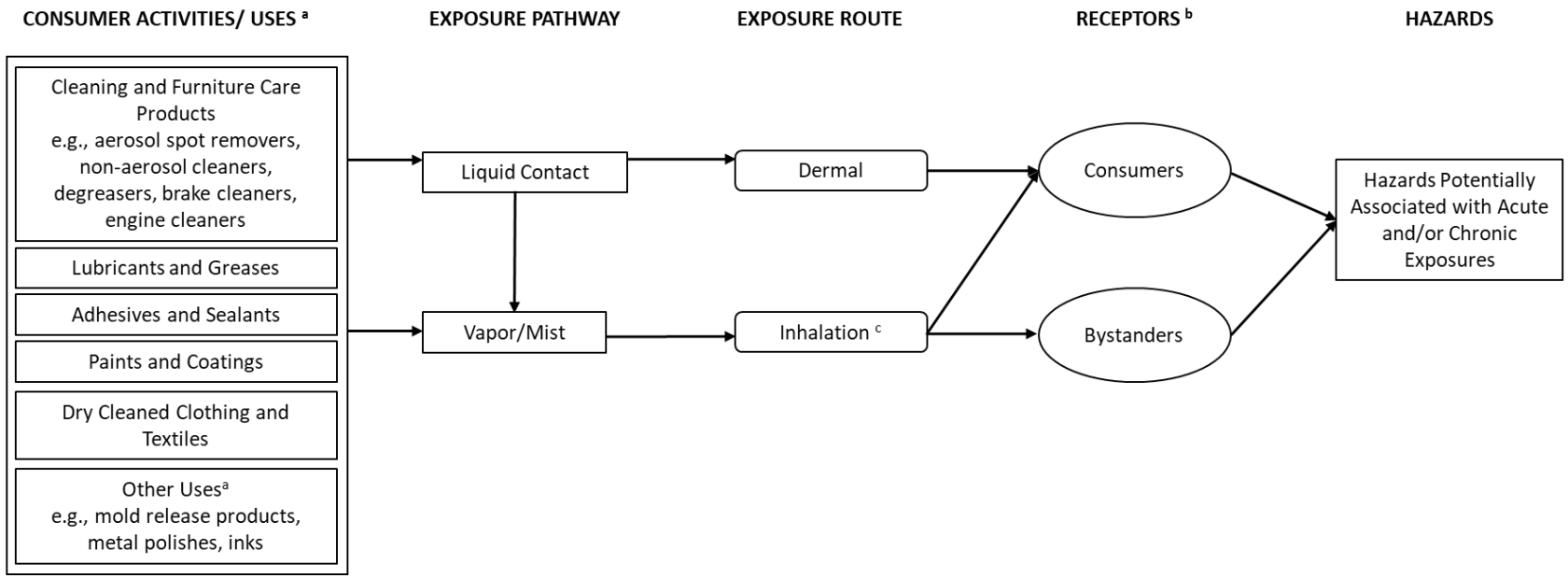


Figure 1-3. PCE Conceptual Model for Consumer Activities and Uses: Potential Exposures and Hazards

The conceptual model presents the exposure pathways, exposure routes and hazards to human receptors from consumer activities and uses of PCE.

[a] Some products are used in both commercial and consumer applications. Additional uses of PCE are included in Table 1-2.

[b] Receptors include potentially exposed or susceptible subpopulations.

[c] Consumers oral exposure may occur through mists that deposit in the upper respiratory tract however, based on physical chemical properties, mists of PCE will likely be rapidly absorbed in the respiratory tract or evaporate and will be considered as an inhalation exposure.

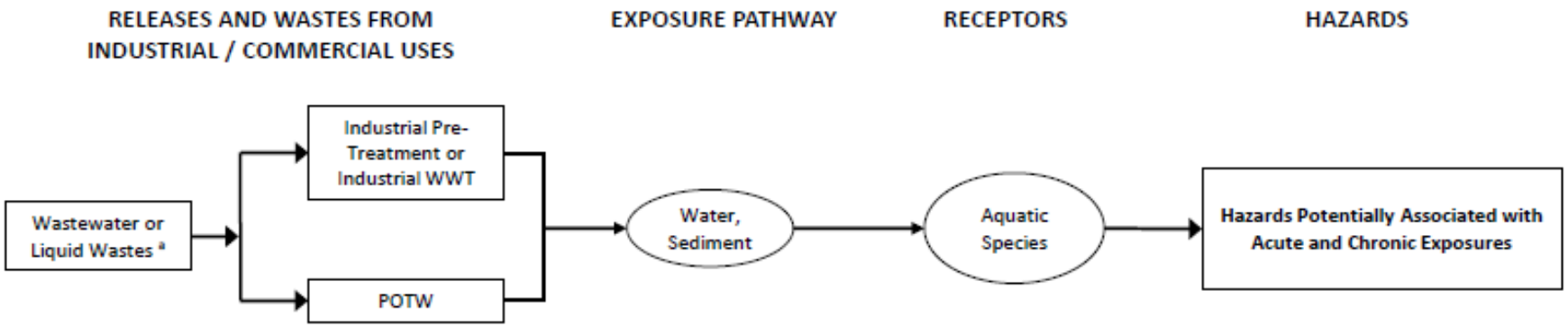


Figure 1-4. PCE Conceptual Model for Environmental Releases and Wastes: Potential Ecological Exposures and Hazards

The conceptual model presents the exposure pathways, exposure routes and hazards to human and environmental receptors from environmental releases and wastes of PCE.

[a] Industrial wastewater or liquid wastes 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 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 2018c). 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 will make an effort to adopt as many best practices as practicable from the systematic review community, EPA expects modifications to 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 PCE is described in *Strategy for Conducting Literature Searches for Perchloroethylene (PCE) Supplemental File to the TSCA Scope Document* (U.S. EPA 2017j) and the results of the title and abstract screening process were published in *PCE (CASRN 127-18-4) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA 2017e).

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

framework[2]. Data sources that met the criteria were carried forward to the data evaluation stage. The inclusion and exclusion criteria for full text screening for PCE are available in in Appendix F of the *Problem Formulation of the Risk Evaluation for PCE* (U.S. EPA 2018d).

Although EPA conducted a comprehensive search and screening process as described above, EPA made the decision to leverage the literature published in previous assessments[3] to identify key and supporting data[4] and information for developing the PCE risk evaluation. This is discussed *Strategy for Conducting Literature Searches for Perchloroethylene (PCE) Supplemental File to the TSCA Scope Document* (U.S. EPA 2017j). In general, many of the key and supporting data sources were identified in the comprehensive *Perchloroethylene (CASRN 127-18-4) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA 2017e). 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 was discussed in Section 4 of *Application of Systematic Review for TSCA Risk Evaluations* (U.S. EPA 2018c). Other relevant key and supporting references were identified through targeted supplemental searches to support the analytical approaches and methods in the PCE 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 Perchloroethylene (PCE) Supplemental File to the TSCA Scope Document* (U.S. EPA 2017j). 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.

[2] 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.

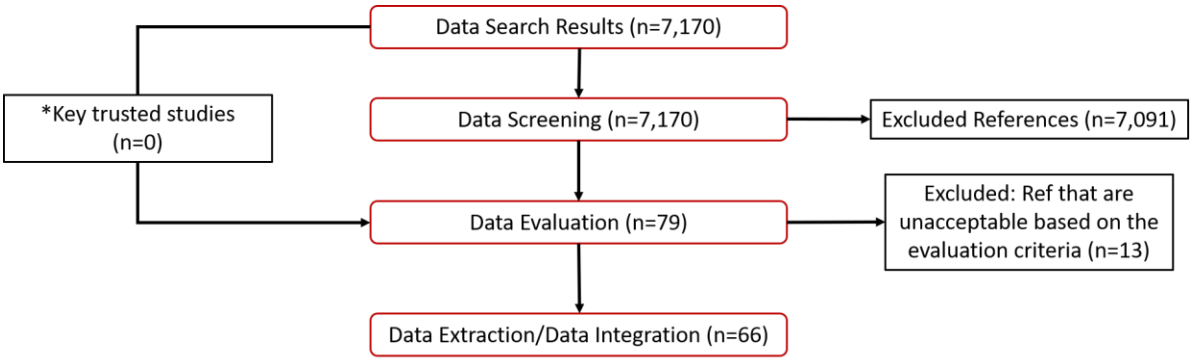
[3] Examples of existing assessments are EPA's chemical assessments (e.g., previous work plan risk assessments, problem formulation documents), ATSDR's Toxicological Profiles and EPA's IRIS assessments. This is described in more detail in *Strategy for Conducting Literature Searches for PCE (PCE) Supplemental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

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

This pragmatic approach allowed EPA to maximize the scientific and analytical efforts of other regulatory and non-regulatory agencies by accepting for the most part the relevant scientific knowledge gathered and analyzed by others except for influential information sources that may have an impact on the weight of the scientific evidence and ultimately the risk findings. The influential information (i.e., key/supporting) came from a smaller pool of sources subject to the rigor of the TSCA systematic review process to ensure that the risk evaluation uses the best available science and the weight of the scientific evidence.

The figures below depict literature flow diagrams illustrating the results of this process for each scientific discipline-specific evidence supporting the draft risk evaluation (Figure 1-5, Figure 1-6, Figure 1-7, Figure 1-8 and Figure 1-9). 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 draft 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.



*Any relevant studies from prior assessments that were identified as potentially relevant for TSCA assessment needs bypassed the data screening step and moved directly to the data evaluation step (e.g. key supporting studies from IRIS assessments, ATSDR assessments, ECHA dossiers, etc.).

Figure 1-5. Literature Flow Diagram for Environmental Fate Information

Note: Literature search results for the environmental fate and transport of PCE yielded 7,170 studies. During problem formulation, following data screening, most environmental exposure pathways were removed from the conceptual models. As a result, 7,091 studies were deemed off-topic and excluded. The remaining 79 studies related to environmental exposure pathways retained in the conceptual models entered data evaluation, where 13 studies were deemed unacceptable and 66 moved into data extraction and integration. Note: Data sources identified relevant to physical-chemical properties were not included in this literature flow diagram. The data quality evaluation of physical-chemical properties studies can be found in the supplemental document, (U.S. EPA 2019c) and the extracted data are presented in Table 1-1.

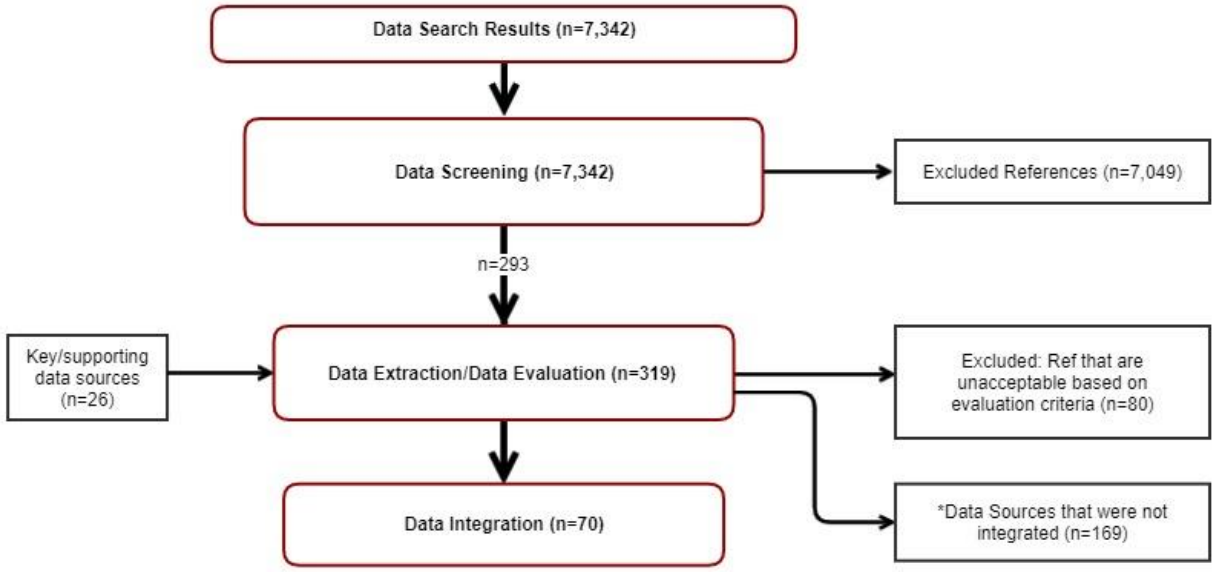


Figure 1-6. Literature Flow Diagram for Engineering Releases and Occupational Exposure

*The quality of data in these sources (n=201) were acceptable for risk assessment 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.

Note: Literature search results for environmental release and occupational exposure yielded 7,342 data sources. Of these data sources, 316 were determined 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 32 relevant data sources that bypassed the data screening step and were evaluated and extracted in accordance with *Appendix D: Data Quality Criteria for Occupational Exposure and Release Data of the Application of Systematic Review for TSCA Risk Evaluations* document (U.S. EPA 2018c). Of the 348 sources from which data were extracted and evaluated, 90 sources only contained data that were rated as unacceptable based on serious flaws detected during the evaluation. Of the 258 sources forwarded for data integration, data from 57 sources were integrated, and 201 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).

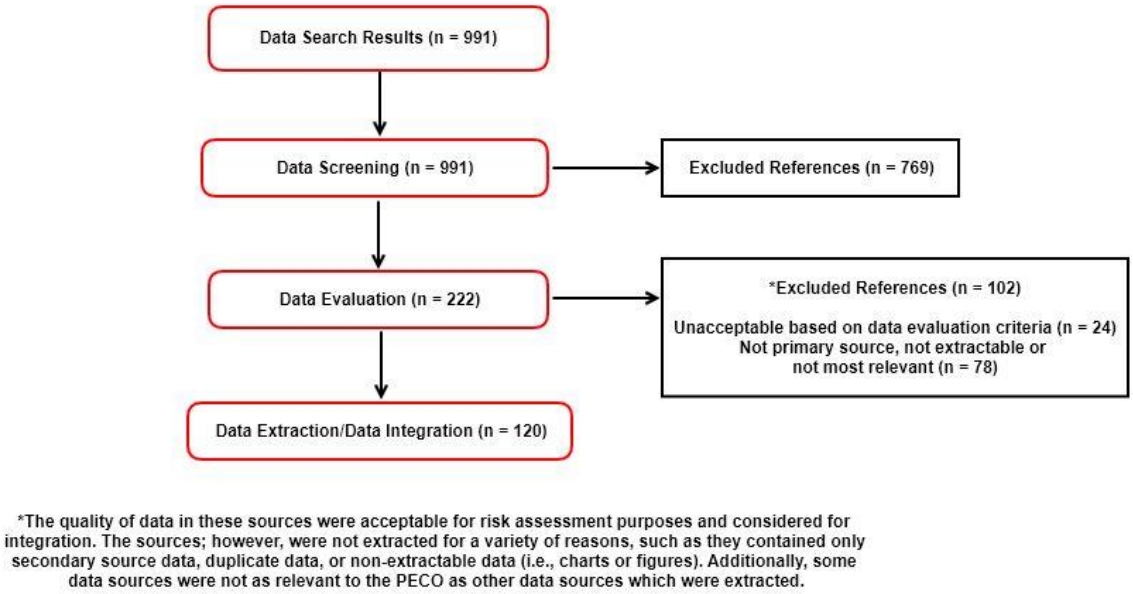


Figure 1-7. Literature Flow Diagram for Consumer and Environmental Exposure Data Sources

Note: EPA conducted a literature search to determine relevant data sources for assessing exposures for perchloroethylene within the scope of the risk evaluation. This search identified 991 data sources including relevant supplemental documents. Of these, 769 were excluded during the screening of the title, abstract, and/or full text and 222 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, 120 references were forwarded for further extraction and data integration. EPA has not developed data quality criteria for all types of exposure information, some of which may be relevant when estimating consumer exposures. This is the case for absorption and permeability data and some product-specific data such as density and weight fraction often reported in Safety Data Sheets. As appropriate, EPA evaluated and summarized these data to determine their utility with supporting the risk evaluation.

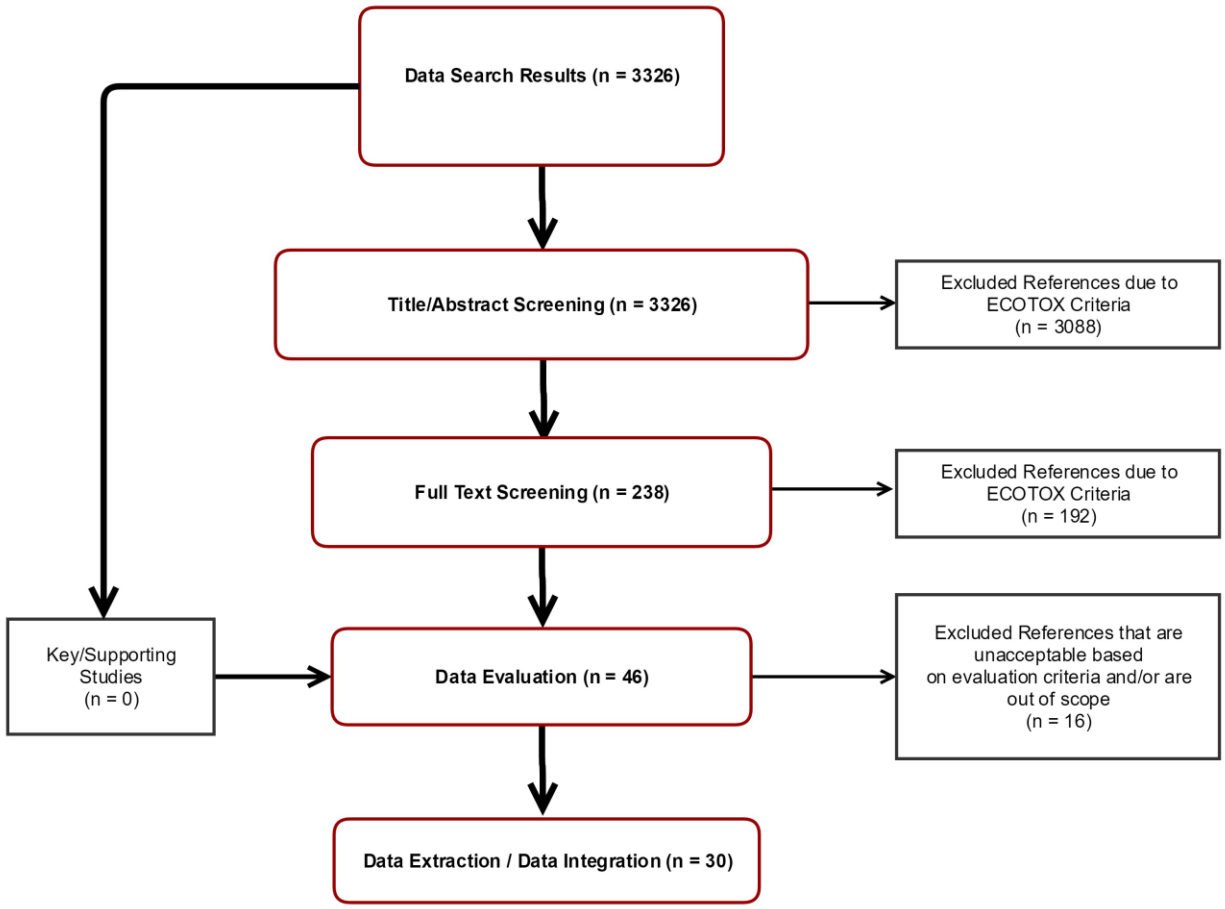


Figure 1-8. Literature Flow Diagram for Environmental Hazard Data Sources

Note: The environmental hazard data sources were identified through literature searches and screening strategies using the ECOTOX Standing Operating Procedures. Additional details about the process can be found in the Strategy for Conducting Literature Searches for *PCE: Supplemental File for the TSCA Scope Document*(U.S. EPA 2017i). During problem formulation, EPA made refinements to the conceptual models resulting in the elimination of the terrestrial exposure pathway. Thus, environmental hazard data sources on terrestrial organisms were considered out of scope and excluded from data quality evaluation.

The literature search process for environmental hazard data found 3326 citations for PCE. At the title and abstract screening phase, 3088 citations were excluded as off-topic using ECOTOXicology knowledgebase criteria. The remaining 238 citations underwent a more thorough full text screening using the same criteria to determine which citations should undergo data evaluation. For data evaluation, EPA developed data quality evaluation (DQE) criteria to evaluate the data under TSCA, based on a combination of EPA's ECOTOXicology knowledgebase (ECOTOX) criteria and the Criteria for Reporting and Evaluating ecotoxicity Data (CRED). There were 46 citations that went to data evaluation for PCE. EPA analyzed each of these studies using the DQE results to determine overall study quality. Thirty studies were considered acceptable and were rated high, medium, or low quality during this analysis. The extracted data from these 30 studies were used during data integration for PCE.

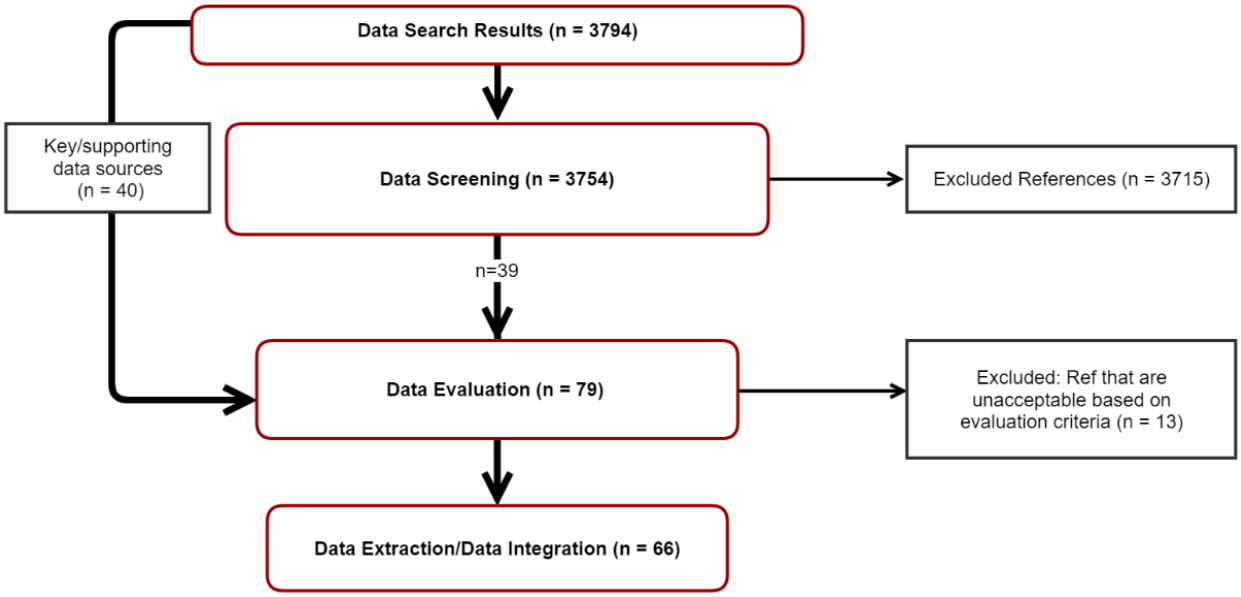


Figure 1-9. Literature Flow Diagram for Human Health Hazard Data Sources

Note: The literature search results for human health hazard of PCE yielded 3794 studies. This included 40 key and supporting studies identified from previous EPA assessments. Of the 3754 new studies screened for relevance, 3715 were excluded as off topic. The remaining 39 new studies together with the 40 key and supporting studies entered data evaluation. Thirteen studies were deemed unacceptable based on the evaluation criteria for human health hazard data sources and the remaining 66 studies were carried forward to data extraction/data integration. Additional details can be found in the *PCE Bibliography: Supplemental File for the TSCA Scope Document*, (U.S. EPA 2017e).

1.5.2 Data Evaluation

During the data evaluation stage, the EPA assesses the quality of the methods and reporting of results of the individual studies identified during problem formulation using the evaluation strategies described in *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA 2018b). The EPA evaluated the quality of the on-topic PCE study reports identified in *Perchloroethylene (CASRN 127-18-4) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA 2017e), and gave all studies an overall high, medium, low or unacceptable confidence rating during data evaluation.

The results of the data quality evaluations for key studies are summarized in Section 2.1(Fate and Transport), Section 2.2 (Releases to the Environment), Section 2.3 (Environmental Exposures), Section 2.4 (Human Exposures), Section 3 (Environmental Hazards) and Section 3.2 (Human Health Hazards). Supplemental files (5.3.68Appendix B) also provide details of the data evaluations including individual metric scores and the overall study score for each data source.

1.5.3 Data Integration

Data integration includes analysis, synthesis and integration of information for the risk evaluation. During data integration, the EPA considers quality, consistency, relevancy, coherence and biological plausibility to make final conclusions regarding the weight of the scientific evidence. As stated in *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA 2018b), data integration involves transparently discussing the significant issues, strengths, and limitations as well as the uncertainties of the reasonably available information and

the major points of interpretation (U.S. EPA 2018e). 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 (U.S. EPA 2017h).

EPA used previous assessments (see Table 1-3) to identify key and supporting information and then analyzed and synthesized available evidence regarding PCE's chemical properties, environmental fate and transport properties and its potential for exposure and hazard. EPA's analysis also considered recent data sources that were not considered in the previous assessments (1.5.1) as well as reasonably available information on potentially exposed or susceptible subpopulations.

The exposures and hazards sections describe EPA's analysis of the influential information (i.e., key and supporting data) that were found acceptable based on the data quality reviews as well as discussion of other scientific knowledge using the approach described in Section 1.5.1. The exposure section also describes whether aggregate or sentinel exposures to a chemical substance were considered under the conditions of use within the scope of the risk evaluation, and the basis for that consideration.

2 EXPOSURES

2.1 Fate and Transport

Environmental fate includes both 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 with other species in the environment. Hence, knowledge of the environmental fate of the chemical informs the determination of the specific exposure pathways and potential human and environmental receptors EPA has considered during risk evaluation.

2.1.1 Fate and Transport Approach and Methodology

Fate data including biotic and abiotic degradation rates, removal during wastewater treatment, volatilization from lakes and rivers, and organic carbon:water partition coefficient (log K_{OC}) were used when describing the fate of PCE. 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 2018b). Table 2-1 provides environmental fate data that EPA considered while assessing the fate of PCE. This data was updated after problem formulation with information identified through systematic literature review. Additional study summaries are in the supplemental document, *Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Extraction Tables for Environmental Fate and Transport Studies* (U.S. EPA 2020h), and complete information on data quality evaluations for all identified fate data are available in the supplemental document, *Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation for Environmental Fate and Transport Studies* (U.S. EPA 2020j). Environmental fate properties not adequately reported in the literature were estimated using Estimation Programs Interface (EPI) Suite™ models, as described in Appendix C.

Table 2-1. Environmental Fate Characteristics of PCE

Property or Endpoint	Value [a]	References	Data Quality Rating
Indirect photodegradation	Atmospheric lifetime = 80-251 days, equivalent to half-life = 55-174 days (estimated for removal by reaction with hydroxyl radical, •OH)	(Cupitt 1987)	High
Hydrolysis half-life	8.8 months	(Dilling et al. 1975)	High
	> Years	(Jeffers et al. 1989)	High
Aerobic Biodegradation	86-87% in 28 days	(Tabak et al. 1981)	High
	74% in batch-fed reactor	(Long et al. 1993)	High
	0% in continuous-flow system	(Bouwer and McCarty 1982)	High
	0% in 175 days	(Bouwer et al. 1981)	Low

Property or Endpoint	Value [a]	References	Data Quality Rating
	Loss of PCE in some studies may be due to volatilization	(Namkung and Rittmann 1987; Wakeham et al. 1983)	Medium, Medium
Anaerobic Biodegradation	100% in 37 days	(Cabirol et al. 1996)	High
	Approx. 38% in 30 days	(Wood et al. 1981)	High
	44%-68% in 112 days	(Bouwer et al. 1981)	High
Bioconcentration factor (BCF)	25.8-77.1 (fish)	(Kawasaki 1980)	High
	49 (fish)	(Barrows et al. 1980)	High
	39.7 (fish)	(Dow Chem 1973)	High
	312 and 118 (marine algae)	(Wang et al. 1996)	High
Bioaccumulation factor (BAF)	46 (estimated)[b]	(ECB 2005); (U.S. EPA 2012a)	High
Organic carbon:water partition coefficient (log K_{oc})	2.95 (estimated)[b]	(U.S. EPA 2012a)	High

[a] Measured unless otherwise noted.
[b] Information was estimated using EPI Suite™ (U.S. EPA 2012a)

2.1.2 Summary of Fate and Transport

The EPI Suite™ module that estimates chemical removal in sewage treatment plants ("STP" module) was run using default settings to evaluate the potential for PCE to be removed from wastewater. The STP module estimates that a total of 88% of PCE in wastewater will be removed, 82% by volatilization and 6% by adsorption to sludge organic matter. Based on the mixed aerobic biodegradation data reported for PCE (ranging from rapid to negligible biodegradation in aerobic environments; see Table 2-1) the overall removal of PCE in wastewater treatment plants is expected to range from 88% to complete. PCE has moderate potential to sorb to sludge organic matter and thus is expected to be present in biosolids (processed sludge). When biosolids are land applied, PCE will volatilize from solid and liquid phases during and after spraying, although some PCE may partition from biosolids into soil and groundwater.

In soil and aquifers, PCE has moderate potential to sorb to soil or sediment organic matter and may be transported to ground water. Anaerobic biodegradation, which is reported to be rapid to very slow depending on local conditions and microbial populations (WHO 2006a; ECB 2005), may be a significant degradation mechanism in soil and groundwater but. In anaerobic environments, PCE biodegradation products include potentially hazardous substances including trichloroethylene, cis-1,2 dichloroethene and vinyl chloride (de Bruin et al. 1992).

Based on its Henry's Law constant (0.0177 atm-m^3/mole) and vapor pressure (18.5 mmHg at 20°C), PCE can be expected to volatilize from surface water to air and from soil to air. The EPI Suite™ model that predicts volatilization for surface water ("Volatilization" module) estimated the PCE volatilization half-life from a model river to be 1.4 hours, and the volatilization half-life from a model lake to be 123 hours (5.1 days). In the vapor phase, PCE can be slowly transformed by reaction with hydroxyl and other radicals with half-lives of months or greater, and long-range transport may occur. In the atmosphere, PCE is expected to slowly degrade via indirect photolysis (half-life ≥ 80 days). Given its slow photodegradation, PCE is expected to undergo long-range atmospheric transport.

With measured bioconcentration factors of 312 or lower and estimated bioaccumulation factor of 46, the bioaccumulation potential of PCE is low.

Overall, PCE has moderate potential to accumulate is wastewater biosolids, soil, and sediment, and has low potential to biota and is expected to largely volatilize to the atmosphere where it may undergo long-range transport and slowly degrade via indirect photolysis. The fate of PCE in the environment is summarized in Figure 2-1.

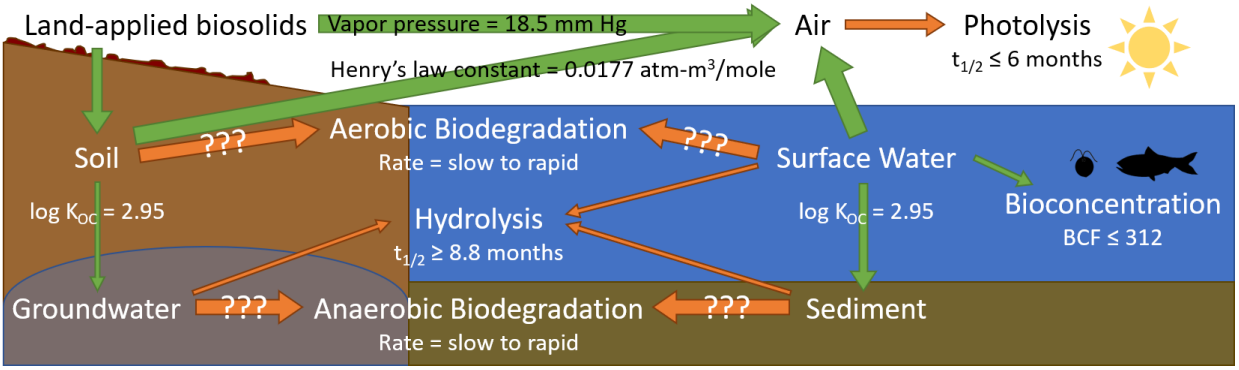


Figure 2-1. Diagram demonstrating the transport, partitioning, and degradation of PCE in the environment

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 and anaerobic biodegradation arrows indicate uncertainty regarding how quickly PCE 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 PCE biodegradation rates in aerobic and anaerobic environments ranged from slow to rapid (see Table 2-1). For comparison, the EPI Suite™ module that predicts biodegradation rates ("BIOWIN" module) was run using default settings to estimate

biodegradation rates of PCE. The BIOWIN models for aerobic environments (BIOWIN 1-6) estimate that PCE will not rapidly biodegrade in aerobic environments. The BIOWIN model of anaerobic biodegradation (BIOWIN 7) predicts that PCE will biodegrade under anaerobic conditions. Overall, PCE biodegradation rates in the environment may vary based on factors including level of oxygenation, microorganisms present, and microorganisms' previous exposure and adaptation to PCE. This uncertainty in biodegradation rates was considered in the assessment of persistence in aerobic and anaerobic environments and estimates of removal from wastewater.

2.2 Releases to the Environment

2.2.1 Environmental Discharges of Wastewater

EPA categorized the conditions of use (COUs) listed in Table 1-4 into 22 Occupational Exposure Scenarios (OES). For each OES, a daily wastewater discharge was estimated based on annual releases, release days, and the number of facilities (Figure 2-2). In this section, EPA describes its approach and methodology for estimating daily wastewater discharges, and for each OES, provides a summary of release days, number of facilities, and daily wastewater discharges. For detailed facility level results, see the "Water Release Assessment" section for each OES in the: *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

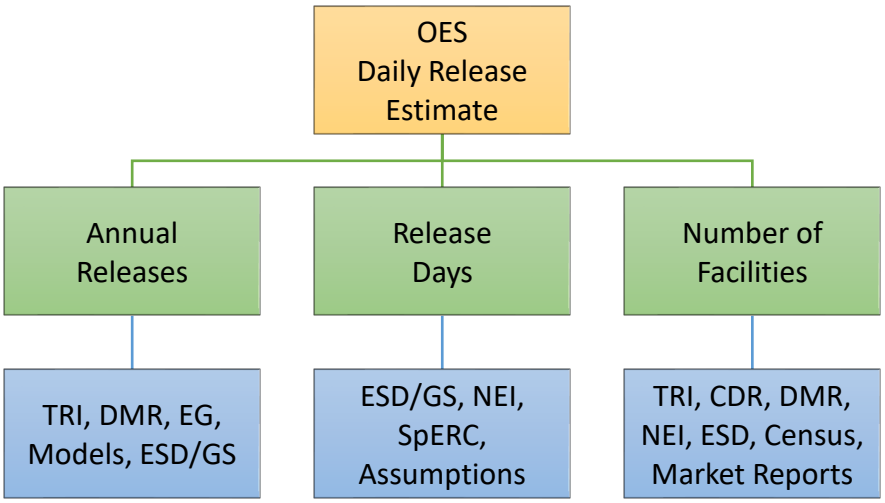


Figure 2-2. An overview of EPA's Approach to Estimate Daily Wastewater Discharges[5].

2.2.1.1 Results for Daily Wastewater Discharge Estimates

EPA combined its estimates for annual releases, release days, and number of facilities to estimate a range for daily wastewater discharges for each OES. A summary of these ranges across facilities is presented in Table 2-2. Summary of EPA's Daily Wastewater Discharge Estimates

[5] TRI = Toxics Release Inventory; DMR = Discharge Monitoring Report; NEI = National Emissions Inventory; CDR = Chemical Data Reporting; EG = Effluent Guidelines; ESD = Emission Scenario Document; GS = Generic Scenarios; SpERC = Specific Environmental Release Category

for Each OES. For some OES, EPA was not able to estimate or did not expect water releases. For example:

- **OES Aerosol Degreasing and Aerosol Lubricants:** Wastewater discharges containing PCE were not expected due to its volatility; releases from this OES are expected to be to air.
- **OES Wipe Cleaning and Metal/Stone Polishes:** Wastewater discharges containing PCE were not expected due to its volatility and the nature of the wipe cleaning and polishing process; releases from this OES are expected to be to air (volatilization) or with shop rags to landfill/incineration.
- **OES Other Spot Cleaning/Spot Removers (Including Carpet Cleaning):** EPA did not identify data to estimate wastewater discharges for this OES.
- **OES Laboratory Chemicals:** EPA did not identify data to estimate wastewater discharges for this OES.

Table 2-2. Summary of EPA's Daily Wastewater Discharge Estimates for Each OES[6]

Occupational Exposure Scenario (OES)	Release Media/ Treatment Facility Type[a]	Number of Sites with Wastewater Discharges[b]	Estimated Daily Release Range Across Sites (kg/site-day)[c]		Overall Confidence	Corresponding Section in the Supplemental Engineering Report (U.S. EPA 2019a)
			Minimum[d]	Maximum		
Manufacturing	Surface Water	1	1.7E-03		M	Section 2.1.4
	Non-POTW WWT	1	4.1E-02		M	
	Surface Water or POTW[e]	4	8.9E-05	0.1	M	
Repackaging	Surface Water	3	9.1E-05	4.8E-03	M	Section 2.2.4
	Non-POTW WWT	1	1.1		M	
Processing as a Reactant	Surface Water	18	1.2E-05	1.3	M	Section 2.3.4
	POTW	1	0.1		M	
Incorporation into Formulation, Mixture, or Reaction Product	Surface Water	1	1.7E-03		M	Section 2.4.4
	POTW	1	1.5E-03		M	
	Non-POTW WWT	1	5.3		M	
Batch Open-Top Vapor Degreasing[f]	Surface Water	16	9.0E-07	7.1E-02	M	Section 2.5.4
	POTW	1	3.5E-04		M	

[6] 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.

Occupational Exposure Scenario (OES)	Release Media/ Treatment Facility Type[a]	Number of Sites with Wastewater Discharges[b]	Estimated Daily Release Range Across Sites (kg/site-day)[c]		Overall Confidence	Corresponding Section in the Supplemental Engineering Report (U.S. EPA 2019a)
			Minimum[d]	Maximum		
Batch Closed-Loop Vapor Degreasing	Included with release estimates for Batch Open Top Vapor Degreasing[f].					Section 2.6.4
Conveyorized Vapor Degreasing	Included with release estimates for Batch Open Top Vapor Degreasing[f].					Section 2.7.4
Web Vapor Degreasing	Included with release estimates for Batch Open Top Vapor Degreasing[f].					Section 2.8.4
Cold Cleaning	Included with release estimates for Batch Open Top Vapor Degreasing[f].					Section 2.9.4
Aerosol Degreasing and Aerosol Lubricants	EPA does not expect wastewater discharges containing PCE from these sites.				H	Section 2.10.4
Dry Cleaning and Spot Cleaning (commercial)	POTW	12,822	5.6E-04	1.7E-03	M	Section 2.11.4
Dry Cleaning and Spot Cleaning (industrial)	Surface Water	2	4.5E-05	2.1E-04	M	Section 2.11.4
Adhesives, Sealants, Paints, and Coatings	POTW	41	2.0	370	M	Section 2.12.4
Maskant For Chemical Milling	Surface Water	3	5.9E-06	8.6E-04	M	Section 2.13.4
	POTW	2	2.6E-03	1.1E-02	M	
Industrial Processing Aid	Surface Water	12	3.0E-04	8.6E-02	M	Section 2.14.4

Occupational Exposure Scenario (OES)	Release Media/ Treatment Facility Type[a]	Number of Sites with Wastewater Discharges[b]	Estimated Daily Release Range Across Sites (kg/site-day)[c]		Overall Confidence	Corresponding Section in the Supplemental Engineering Report (U.S. EPA 2019a)
			Minimum[d]	Maximum		
	POTW	2[g]	8.8E-02	0.4	M	
Metalworking Fluids	Included with release estimates for Batch Open Top Vapor Degreasing[f].					Section 2.15.4
Wipe Cleaning and Metal/Stone Polishes	EPA does not expect wastewater discharges containing PCE from these sites.				H	Section 2.16.4
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	EPA did not identify data to estimate wastewater discharges for this OES.					Section 2.17.4
Other Industrial Uses	Surface Water	7	1.1E-06	0.3	M	Section 2.18.4
Other Commercial Uses	Surface Water	7	1.3E-05	2.9E-03	M	Section 2.19.4
Laboratory Chemicals	EPA did not identify data to estimate wastewater discharges for this OES.					Section 2.20.4
Waste Handling, Disposal, Treatment, and Recycling	Surface Water	5	5.9E-05	3.8E-03	M	Section 2.21.4
	POTW	4	3.6E-07	0.3	M	
	Non-POTW WWT	4	5.4E-03	1.4	M	
Other Department of Defense Uses	EPA did not identify data to estimate wastewater discharges for this OES.					Section 2.22.4

[a] The daily discharge estimates presented in this table represent both direct discharges to surface water and indirect discharges to POTW and non-POTW WWT. Removal efficiencies at POTWs and non-POTW WWT are taking into account in the environmental exposure assessment.

[b] For most conditions of use, only a subset of the sites use are expected to discharge wastewater containing PCE. Other sites may dispose of PCE-containing wastes through other means such as via landfill or incineration.
[c] Except for commercial dry cleaning estimates; the minimum and maximum daily discharge estimates are based on site-specific discharges (i.e., the minimum corresponds to the site with the lowest discharge and the maximum corresponds to the site with the highest discharge). Minimum daily discharge at any given site may be higher than the minimum presented, and the maximum daily discharge may be lower than the value presented.
[d] The minimum presented represents the minimum of the sites that have wastewater discharges, it does not include sites that dispose of PCE through other media which would result in a minimum of zero for most OES.
[e] Discharges from these sites may be to either surface water or POTW but not both for a given site.
[f] EPA does not have enough information to distinguish whether these sites use PCE in OTVDs, closed-loop degreasers, conveyorized degreasers, web degreasers, cold cleaners, or metalworking fluids. Therefore, the daily release estimates may include sites that perform any of these activities.
[g] These two sites reported both direct and indirect discharges.

2.2.1.2 Approach and Methodology

2.2.1.2.1 Wastewater Discharge Estimates

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 2017k) and 2016 Discharge Monitoring Report (DMR) (U.S. EPA 2016a) 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 NAICS code, and manufactures, processes, or uses the chemical in quantities greater than a certain threshold (25,000 pounds for manufacturers and processors of PCE and 10,000 pounds for users of PCE). Due to these limitations, some sites that manufacture, process, or use PCE 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) to query all PCE 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 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 PCE may not be included in the DMR dataset.

Facilities reporting discharges 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 discharge and mapped each facility to a potential condition of use associated with occupational exposure scenarios (OES, see Table 2-12). 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.

EPA's preference was to use TRI or DMR data to assess wastewater discharges; however, due to the reporting requirements for each dataset (described above in this section), these data may not be available for all conditions of use or for all sites within a condition of use. In such cases, EPA estimated wastewater discharges using release data from literature, relevant emission scenario documents (ESD) or generic scenarios (GS), existing EPA/OPPT models, and/or relevant Effluent Guidelines (EG). EG are national regulatory standards set forth by EPA for wastewater discharges to surface water and municipal sewage treatment plants.

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 ESDs, GSs, existing models, or EGs were used EPA estimated a daily release and calculated the annual release by multiplying the daily release by the number of release days per year.

2.2.1.2.2 Estimates of Number of Facilities

Where available, EPA used 2016 CDR (U.S. EPA 2016d), 2016 TRI (U.S. EPA 2017k), 2016 Discharge Monitoring Report (DMR) (U.S. EPA 2016a) and 2014 National Emissions Inventory (NEI) (U.S. EPA 2018a) data to provide a basis to estimate the number of sites using PCE within a condition of use. Generally, information for reporting sites in CDR and NEI was sufficient to accurately characterize each reporting sites condition of use. However, information for determining the condition of use for reporting sites in TRI and DMR is typically more limited.

In TRI, sites submitting a Form R indicate whether they perform a variety of activities related to the chemical including, but not limited to: produce the chemical; import the chemical; use the chemical as a reactant; use the chemical as a chemical processing aid; and ancillary or other use. In TRI, sites submitting Form A are not required to designate an activity. For both Form R and Form A, TRI sites are also required to report the primary North American Industry Classification System (NAICS) code for their site. For each TRI site, EPA used the reported primary NAICS code and activity indicators to determine the condition of use at the site. For instances where EPA could not definitively determine the condition of use because: 1) the report NAICS codes could include multiple conditions of use; 2) the site report multiple activities; and/or 3) the site did not report activities due to submitting a Form A, EPA had to make an assumption on the condition of use to avoid double counting the site. For these sites, EPA supplemented the NAICS code and activity information with the following information to determine a "most likely" or "primary" condition of use:

1. Information on known uses of the chemical and market data identifying the most prevalent conditions of use of the chemical.
2. Information obtained from public comments and/or industry meetings with EPA that provided specific information on the site.

In DMR, the only information reported on condition of use is each site's Standard Industrial Classification (SIC) code. EPA could not determine each reporting site's condition of use based on SIC code alone; therefore, EPA supplemented the SIC code information with the same supplementary information used for the TRI sites (market data, public comments, and industry meetings).

Where the number of sites could not be determined using CDR/TRI/DMR/NEI or where CDR/TRI/DMR/NEI data were determined to not capture the entirety of sites within a condition of use, EPA supplemented the available data with U.S. economic data using the following method:

1. Identify the NAICS codes for the industry sectors associated with these uses.
2. Estimate total number of sites using the U.S. Census' Statistics of US Businesses (SUSB) (SUSB Data) data on total establishments by 6-digit NAICS.
3. Use market penetration data to estimate the percentage of establishments likely to be using PCE instead of other chemicals.
4. Combine the data generated in Steps 1 through 3 to produce an estimate of the number of sites using PCE in each 6-digit NAICS code, and sum across all applicable NAICS codes for the condition of use to arrive at a total estimate of the number of sites within the condition of use.

Table 2-3 summarizes the number of facilities estimates for each OES. Based on reasonably available data, EPA does not expect all sites within a condition of use will have wastewater discharges containing PCE; therefore, the number of facilities estimates in Table 2-3 may be greater than the number of sites presented in release summary in Table 2-2.

Table 2-3. Summary of EPA's Estimates for the Number of Facilities for Each OES

Occupational Exposure Scenario (OES)	Number of Facilities	Notes
Manufacturing	8	Based on CDR reporting
Repackaging	51	Based on TRI and DMR reporting
Processing as a Reactant	117	Based on TRI and DMR reporting
Incorporation into Formulation, Mixture, or Reaction Product	39	Based on TRI and DMR reporting
Batch Open-Top Vapor Degreasing	398 to 4,942	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Batch Closed-Loop Vapor Degreasing	13,912 to 25,546	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Conveyorized Vapor Degreasing	395 to 568	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Web Degreasing	395 to 568	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Cold Cleaning	17	Based on NEI reporting
Aerosol Degreasing and Aerosol Lubricants	75,938	Based on Census data and a market penetration of 29.6% based on California Air Resources Board (CARB) survey of automotive maintenance and repair facilities
Dry Cleaning and Spot Cleaning	12,822 (commercial) 12 (industrial)	Commercial estimate based on Census data and a market penetration of 60% based on information from the Dry Cleaning and Laundry Institute and the National Cleaners Association Industrial estimate based on U.S. EPA (2006b) economics report
Adhesives, Sealants, Paints, and Coatings	60	Based on NEI reporting
Maskant for Chemical Milling	71	Based on stakeholder information from AC Products (2017)
Industrial Processing Aid	98	Based on TRI and DMR reporting
Metalworking Fluids	-	No information identified to estimate number of facilities
Wipe Cleaning and Metal/Stone Polishes	-	No information identified to estimate number of facilities
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	-	No information identified to estimate number of facilities
Other Industrial Uses	130	Based on TRI and DMR reporting
Other Commercial Uses	-	No information identified to estimate number of facilities
Laboratory Chemicals	-	No information identified to estimate number of facilities
Waste Handling, Disposal, Treatment, and Recycling	94	Based on TRI and DMR reporting
Other Department of Defense Uses	-	No information identified to estimate number of facilities

2.2.1.2.3 Estimates of Release Days

EPA referenced ESDs, NEI data, SpERCs, or needed to make assumptions when estimating release days for each OES. A summary along with a brief explanation is presented in Table 2-4 below.

Table 2-4. Summary of EPA's Estimates for Release Days for Each OES

Occupational Exposure Scenario (OES)	Release Days	Notes
Manufacturing	350	Assumes operation seven days/week and 50 weeks/yr with two weeks down for shutdown activities
Repackaging	250	Assumed 5 days per week and 50 weeks per year
Processing as a Reactant	350	Assumes operation seven days/week and 50 weeks/yr with two weeks down for shutdown activities
Incorporation into Formulation, Mixture, or Reaction Product	300	SpERC for the formulation and (re)packing of substances and mixtures (European Solvents Industry 2019)
Batch Open-Top Vapor Degreasing	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Batch Closed-Loop Vapor Degreasing	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Conveyorized Vapor Degreasing	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Web Degreasing	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Cold Cleaning	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Aerosol Degreasing and Aerosol Lubricants	-	Wastewater discharges not expected from this OES
Dry Cleaning and Spot Cleaning	250 to 312	Assumes facilities may operate five days/week and 50 weeks/yr at the low-end up to six days/week and 52 weeks/yr at the high-end
Adhesives, Sealants, Paints, and Coatings	250	Assumed 5 days per week and 50 weeks per year
Maskant for Chemical Milling	172 to 208	Based on NEI reporting
Industrial Processing Aid	300	SpERC for the manufacture of a substance (which includes use as a process chemical or extraction agent) (European Solvents Industry 2012)
Metalworking Fluids	260	2017 Draft ESD on the Use of Vapor Degreasers (OECD 2017a)
Wipe Cleaning and Metal/Stone Polishes	-	Wastewater discharges not expected from this OES
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	-	No information identified to estimate wastewater discharges from this OES

Occupational Exposure Scenario (OES)	Release Days	Notes
Other Industrial Uses	250	Assumed 5 days per week and 50 weeks per year
Other Commercial Uses	250	Assumed 5 days per week and 50 weeks per year
Laboratory Chemicals	-	No information identified to estimate wastewater discharges from this OES
Waste Handling, Disposal, Treatment, and Recycling	250	Assumed 5 days per week and 50 weeks per year
Other Department of Defense Uses	-	No information identified to estimate wastewater discharges from this OES

2.2.1.3 Assumptions, Key Sources of Uncertainty, and Overall Confidence for Environmental Releases

Table 2-5 provides a summary of the assumptions, key sources of uncertainty, and EPA's overall confidence in its release estimates for each of the OES assessed.

Table 2-5. Summary of Assumptions, Uncertainty, and Overall Confidence in Release Estimates by OES

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
Manufacturing	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI for four sites. TRI data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of "medium". The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in the Daily Discharge Estimates:*** EPA assumed 350 days/yr of operation (7 days/week, 50 weeks/yr with two weeks for turnaround) and averaged the annual discharges over the operating days. There is some uncertainty that all sites manufacturing PCE will operate for this duration as some sites may operate less than 7 days/wk or may have turnarounds greater than or less than the assumed 2 weeks/yr. Therefore, the average daily discharges may be higher if sites operate for fewer than 350 days/yr or lower if they operate for greater than 350 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day due to changes in process conditions (e.g., total wastewater flow) such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge.

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	Strengths in Discharges Assessed Using Effluent Guidelines: The discharges estimated using the EG are within an order of magnitude of the discharges reported by sites in TRI. The exception to this is the Solvents & Chemicals site which had a much lower production volume than the averaged assessed at all other sites. ***Uncertainties in Discharges Assessed Using Effluent Guidelines:*** Water discharges from the remaining four sites were estimated using the maximum daily and monthly discharge limits in the OCPSF (Organic Chemicals, Plastics and Synthetic Fibers) EG and the estimated volume of wastewater produced per pound of PCE production from the SpERC developed by the European Solvent Industry Group for the manufacture of a substance. The estimates assume the sites operate at the limits set by the EG; actual releases may be lower for sites operating below the limits or higher for sites not in compliance with the OCPSF EG. Furthermore, the production volumes used to estimate discharges for three of the four sites are based on the average production volume. Each site may manufacture volumes greater than or less than the average resulting in higher or lower discharge volumes, respectively. ***Uncertainties in the Number of Sites Estimate:*** Information to determine the activity at two of the assessed sites as manufacture or import was not publicly available. It is possible these two sites are importers and not manufacturers; thus, eliminating the wastewater discharges from manufacturing at these sites (note: the sites may have other wastewater discharges of PCE depending on the conditions of use at the site). ***Overall Confidence Rating:*** Based on the data quality score and the uncertainties in the daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates for the four sites in the 2016 TRI. Based on the uncertainties in using effluent guidelines and the number of sites, EPA has a medium confidence in the wastewater discharge estimates for the four sites assessed using the OCPSF EG.
Repackaging	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing repackaging activities rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 250 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites repackaging PCE will repackage PCE for this duration as some sites may not repackage PCE every day while others may operate more than 5 days/week and 50 weeks/yr. Therefore, the average daily discharges may be higher if sites repackage for fewer than 250 days/yr or lower if they repackage for greater than 250 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Processing as a Reactant	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are processing PCE as a reactant rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged;

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 350 days/yr of operation (7 days/week, 50 weeks/yr with two weeks for turnaround) and averaged the annual discharges over the operating days. There is some uncertainty that all sites processing PCE as a reactant will operate for this duration as some sites may operate less than 7 days/wk, have turnarounds greater than or less than the assumed 2 weeks/yr, or not manufacture products that use PCE as a reactant every day. Therefore, the average daily discharges may be higher if sites operate for fewer than 350 days/yr or lower if they operate for greater than 350 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Incorporation into Formulation, Mixture, or Reaction Product	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing formulation activities rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 300 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites formulating PCE-

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	based products will operate for this duration as some sites may not make products that contain PCE every day while others may operate more than 300 days/yr based on product demand and process needs. Therefore, the average daily discharges may be higher if sites operate for fewer than 300 days/yr or lower if they operate for greater than 300 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Batch Open-Top Vapor Degreasing	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, EPA does not expect all sites using PCE in OTVD to be captured in the databases. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT; however, the sites may be required to comply with an EG depending on the industry in which the OTVD is being used. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are using PCE in OTVD rather than a different condition of use (including other vapor degreasing and cold cleaning operations and use of PCE in metalworking fluids). If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 260 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites using PCE in OTVDs will operate for this duration as some sites may use degreasing equipment more or less frequently than 260 days/yr depending on process demands. Therefore, the average daily discharges may be higher if sites operate for fewer than 260 days/yr or lower if they operate for greater than

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	260 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Batch Closed-Loop Vapor Degreasing	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Conveyorized Vapor Degreasing	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Web Degreasing	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Cold Cleaning	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Aerosol Degreasing and Aerosol Lubricants	EPA assessed no wastewater discharges for this OES. There is some uncertainty as to whether and how much PCE may deposit on shop floors. However, due to the volatility of PCE, EPA expects PCE to evaporate from any such deposit prior to it being discharged; thus, limiting any potential discharges to surface water, POTW, or non-POTW WWT from this source. Based on this information, EPA has a high confidence in the release assessment.
Dry Cleaning and Spot Cleaning	***Data Quality Ratings:*** Wastewater discharges from industrial launderers are assessed using reported discharges from the 2016 DMR. DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. The "low" scores are a result of the information available in DMR. For example, DMR does not include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Limitations to Release Data for Industrial Launderer:*** DMR does not contain data for 4 of the 12 industrial launderer sites. These four sites may not be in DMR because they may have no water discharges or because they discharge to sewer rather than surface water (sewer discharges not reported in DMR). ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to DMR only report annual discharges; to assess daily discharges, EPA assumed annual days of operation and averaged the annual discharges over the operating days. There is some uncertainty that all industrial launderers using PCE will operate for this duration as site-specific demands may result in higher or lower operating days. Therefore, the average daily discharges may be higher if sites operate for fewer than the operating days or lower if they operate for greater than the operating days. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. Based on this information, EPA has a medium confidence in the wastewater discharge estimates at industrial launderers.

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	Strengths of the Release Model for Small Commercial Dry Cleaners: Wastewater discharges from small commercial dry cleaners is assessed using the Solvent Release in Water Discharge from Dry Cleaning Machines Model. The model is based on the *EPA/OPPT Water Saturation Loss Model*, which assumes that water contacted with the chemical becomes saturated with the chemical and remains saturated at the time of disposal. The primary difference between this model and the *EPA/OPPT Water Saturation Model* is this model calculates the amount of produced wastewater using data (and distributions, where available) obtained from literature for the volume of water produced water per pound of clothes cleaned, load size, and loads per day. Using these parameters and distributions the model is able to capture variability in the amount of produced wastewater at dry cleaners. ***Uncertainties in the Release Model for Small Commercial Dry Cleaners:*** There is some uncertainty on how sites will dispose of water containing-PCE and some states may regulate the disposal; therefore, not all sites are expected to discharge wastewater to POTW. ***Overall Confidence Rating:*** Based on the data quality score, the limitations to the release data, and the uncertainties in the daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates at industrial launderers. Based on the strengths and uncertainties of the model, EPA has a medium level of confidence in the wastewater discharge estimates at small commercial dry cleaners.
Adhesives, Sealants, Paints, and Coatings	***Uncertainties in the Release Model:*** Wastewater discharges from adhesive, sealant, coating, and paint applications are assessed using loss fractions from ESDs and the EPA/OPPT Automobile OEM (Original Equipment Manufactuer) Coating Overspray Loss Model. These approaches represent release estimates for the solids (i.e., non-volatile) portions of the coatings or adhesives and do not account for potential evaporation of volatiles from the mist prior to entering wastewater. Therefore, these estimates likely overestimate actual wastewater discharges of PCE due to volatilization (PCE vapor pressure is 18.5 mmHg at 25°C). This evaporation is difficult to estimate and is not considered in this assessment. ***Uncertainties in Number of Sites Estimate:*** There is further uncertainty that the number of sites obtained from the 2014 NEI represent the total number of sites using adhesives or coatings containing PCE. NEI data only covers specific industries which may not capture the entirety of industries using these products. NEI also does not include operations that are classified as area sources because area sources are reported at the county level and do not include site-specific information. It is uncertain the extent that sites not captured in this assessment discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. ***Overall Confidence Rating:*** Based on the uncertainties in the release model and number of sites, EPA has a medium confidence in the wastewater discharge estimates.

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
Maskant for Chemical Milling	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** The discharges in TRI and DMR do not include 44 of the expected 71 sites that use PCE-based maskants. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT; however, the sites may be required to comply with the Metal Finishing EG. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing maskant operations rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA used site-specific reported operating time from the 2014 NEI, where available, or assumed 172 days/yr of operation (based on the average operating time from the 2014 NEI) and averaged the annual discharges over the operating days. There is some uncertainty that all sites using PCE-based maskants will operate for this duration as, based on process needs, some sites may perform masking activities more or less frequently than the average days/yr from NEI or use other maskants not containing PCE for certain operations. Therefore, the average daily discharges may be higher if sites operate for fewer than the estimated operating days or lower if they operate for greater than the estimated operating days. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Industrial Processing Aid	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are using PCE as a processing aid rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 300 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites using PCE as a processing aid will operate for this duration as some sites may use PCE processing aids more or less frequently than 300 days/yr based on process needs. Therefore, the average daily discharges may be higher if sites operate for fewer than 300 days/yr or lower if they operate for greater than 300 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Metalworking Fluids	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Wipe Cleaning and Metal/Stone Polishes	EPA assessed no wastewater discharges for this OES. There is some uncertainty as to whether and how much PCE may drip from the rag/cloth or the substrate surface onto shop floors or ground (for outdoor applications). However, due to the volatility of PCE, EPA expects PCE to evaporate from any such deposit prior to it being discharged; thus, limiting any potential discharges to surface water, POTW, or non-POTW WWT from this source. Based on this information, EPA has a high confidence in the release assessment.

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	No information identified to estimate wastewater discharges from this OES.
Other Industrial Uses	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing other industrial uses rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 250 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites using PCE for other industrial uses will operate for this duration as some sites may use PCE more or less frequently than 250 days/yr based on process needs. Therefore, the average daily discharges may be higher if sites operate for fewer than 250 days/yr or lower if they operate for greater than 250 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Other Commercial Uses	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 DMR. DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the DMR data were scored high for representativeness of

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in DMR. For example, DMR does not include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for DMR, these sites are not expected to capture the entirety of water releases from this OES. It is uncertain the extent that sites not captured in DMR discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing other commercial uses rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to DMR only report annual discharges; to assess daily discharges, EPA assumed 250 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites using PCE in other commercial uses will operate for this duration as some sites may use PCE more or less frequently than 250 days/yr based on process needs. Therefore, the average daily discharges may be higher if sites operate for fewer than 250 days/yr or lower if they operate for greater than 250 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Laboratory Chemicals	No information identified to estimate wastewater discharges from this OES.
Waste Handling, Disposal, Treatment, and Recycling	***Data Quality Ratings:*** Wastewater discharges are assessed using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" data quality rating through EPA's systematic review process. Specifically, the TRI and DMR data were scored high for representativeness of geographic scope, applicability, and temporal representativeness but scored low for methodology, accessibility/clarity, and variability/uncertainty resulting in an overall quality of medium. The "low" scores are a result of the information available in each data source. For example, neither TRI nor DMR include: data on how each reporter estimated their releases (methodology); metadata (e.g., release frequency, process/unit operation that is the source of the release) other than the media

Occupational Exposure Scenario (OES)	Assumptions, Uncertainty, and Overall Confidence in Release Estimates
	of release (accessibility/clarity); or address variability/uncertainty in the reported estimates. ***Uncertainties in Number of Sites Estimate:*** Due to reporting requirements for TRI and DMR, the number of sites in this OES may be underestimated. It is uncertain the extent that sites not captured in these databases discharge wastewater containing PCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of PCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all of the sites assessed in this section are performing waste treatment, disposal, and recycling activities rather than a different condition of use. If the sites were categorized under a different OES, the annual wastewater discharges for each site would remain unchanged; however, average daily discharges may change depending on the number of operating days expected for the OES. ***Uncertainties in the Daily Discharge Estimates:*** Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 250 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites disposing/treating/recycling wastes containing PCE will operate for this duration as some sites may receive/treat PCE-containing wastes more or less frequently than 250 days/yr based on customer demands. Therefore, the average daily discharges may be higher if sites operate for fewer than 250 days/yr or lower if they operate for greater than 250 days/yr. Furthermore, PCE concentrations in wastewater discharges at each site may vary from day-to-day such that on any given day the actual daily discharges may be higher or lower than the estimated average daily discharge. ***Overall Confidence Rating:*** Based on the data quality score, and the uncertainties in the number of sites and daily discharge estimates, EPA has a medium confidence in the wastewater discharge estimates.
Other Department of Defense Uses	No information identified to estimate wastewater discharges from this OES.

2.3 Environmental Exposures Overview

The manufacturing, processing, use and disposal of PCE can result in releases to the environment. In this section, EPA presents what approach and methodology was used to evaluate PCE exposures to aquatic organisms via surface water. The environmental exposure characterization focuses on aquatic releases of PCE from facilities that use, manufacture, or process PCE under industrial and/or commercial conditions of use subject to TSCA regulations.

To characterize environmental exposure, EPA identified and reviewed national scale monitoring data. Measured surface water concentrations were obtained from EPA's Water Quality Exchange (WQX) using the online 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 Survey (USGS), National Water Information System (NWIS), and other federal, state, and tribal sources. A full systematic review

of reasonably available surface water literature was also conducted to identify other peer-reviewed or grey literature[7] sources of measured surface water concentrations in the US. Point estimate exposures were derived from both measured and predicted concentrations of PCE in surface water in the United States. Predicted surface water concentrations were modeled for facility releases in the EPA Lifecycle Release Analysis conducted for reporting year 2016, as determined from EPA's Toxics Release Inventory (TRI), Discharge Monitoring Reports (DMR; through EPA's Water Pollutant Loading Tool), and EPA's Chemical Data Reporting (CDR).

The aquatic modeling was conducted with EPA's Exposure and Fate Assessment Screening Tool, version 2014 (E-FAST 2014) (U.S. EPA 2014b), using reported annual release/loading amounts (kg/yr) and estimates of the number of days per year that the annual load is released. As appropriate, two scenarios were modeled per release: release of the annual load over an estimated maximum number of operating days per year and over only 20 days per year. Twenty days of release was modeled as the low-end release frequency at which possible ecologic chronic risk could be determined. Additionally, the Probabilistic Dilution Model (PDM), a module of E-FAST 2014 was run to estimate 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 watershed level (HUC-8 and HUC-12; Hydrologic Unit Codes) was conducted to compare the measured and predicted surface water concentrations and investigate if the facility releases may be associated with the observed concentrations in surface water. Hydrologic Unit Codes (HUCs) are a geographically hierarchical tiered approach to organizing stream networks across the United States from regions to sub water sheds 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 they were expected to give a representative geographic size range over which predicted surface water concentrations would be relevant to measured concentrations.

2.3.1 Aquatic Exposure Modeling Approach

Surface water concentrations resulting from wastewater releases of PCE from facilities that use, manufacture, or process PCE related to TSCA conditions of use were modeled using EPA's Exposure and Fate Assessment Screening Tool, Version 2014 (U.S. EPA 2014b). 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 high-end exposure estimates. Other assumptions and uncertainties in the model, including ways it may be underestimating or overestimating exposure, are discussed in the Sections 4.3.1. 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

[7] Grey 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. (U.S. EPA 2018c)

tool, as well as a description of required inputs and the methodology to obtaining and using inputs specific to this assessment is described below. To obtain more detailed information on the E-FAST 2014 tool from the user guide/background document (U.S. EPA 2014b), as well as to download the tool, 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.

2.3.1.1 Exposure and Fate Assessment Screening (E-FAST) Tool 2014 Inputs

Individual model inputs and accompanying considerations for the surface water modeling for E-Fast 2014 (U.S. EPA 2014b) are discussed in the following sections.

2.3.1.1.1 Chemical release to wastewater (WWR)

Annual wastewater loading estimates (kg/site/year or lb/site/year) were obtained from TRI, the Water Pollutant Loading Tool, or CDR in the year 2016, as discussed in the lifecycle assessment in Section 2.2.1.1. To model these releases within E-FAST 2014 (U.S. EPA 2014b), the annual release is converted to a daily release using an estimated days of release per year. Below is an example calculation:

WWR (kg/day) = Annual loading (kg/site/year) * Days released per year (days/year) (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).

2.3.1.1.2 Release Days (days/year)

The number of days per year 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 (U.S. EPA 2014b). The two scenarios modeled are a maximum release frequency (200 to 365 days) based on estimates specific to the facility's condition of use and a low-end release frequency of 20 days of release per year. The 20-day chronic risk 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 occur at receiving WWTPs which typically operate every day of the year.

2.3.1.1.3 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 2.1.2, Summary of Fate and Transport, the WWT% for PCE was estimated as 80% using the "STP" module within The EPI Suite™, which was run using default settings to evaluate the potential for PCE to volatilize to air or adsorb to sludge during wastewater treatment. However, E-FAST does not consider volatilization of PCE

therefore the removal percentage of 80% was slightly lower than what EPI suites estimated at 88%. EPA took a more conservative approach in the estimated removal of PCE using the E-FAST model. The WWT% of 80% 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. Direct discharging facilities that release PCE to surface water is not treated prior to discharge, therefore EPA does not account for removal of PCE. If not enough release information was available to determine if the release was direct or indirect, then E-FAST 2014 (U.S. EPA 2014b) was run with and without the WWT%. These releases are typically those identified through the OCSPF EGL data source and are from facilities that are not in DMR or TRI.

2.3.1.1.4 Facility or Industry Sector

The required site-specific stream flow or dilution factor information is contained in the E-FAST 2014 database (U.S. EPA 2014b), which is accessed by querying a facility National Pollutant Discharge Elimination System (NPDES) number, name, or reach code. For facilities that directly discharge to surface water (i.e., "direct dischargers"), the NPDES 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 waste-water treatment plant (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 of receiving facilities, the NPDES was obtained using EPA's Envirofacts search tool (https://www3.epa.gov/enviro/facts/multisystem.html, (U.S. EPA 2019d)). If a facility NPDES was not available in the E-FAST-2014 database (U.S. EPA 2014b), the release was modeled using water body data for a surrogate NPDES (preferred) or an industry sector, as described below.

2.3.3.1.4.1 Surrogate NPDES

In cases where the site-specific NPDES was not available in the E-FAST 2014 database (U.S. EPA 2014b), the preferred alternative was to select the NPDES for a nearby facility that discharges to the same waterbody. Nearby facilities were identified using the Chemical Safety Mapper within IGEMS and/or search of the E-FAST 2014 database (U.S. EPA 2014b) by reach code.

2.3.3.1.4.2 Industry Sector (SIC Code Option)

If the NPDES 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 (U.S. EPA 2014b). This option uses the 10^{th} and 50^{th} percentile receiving 7Q10 stream flows for dischargers in a given industry sector, as defined by the Standard Industrial Classification (SIC) codes of the industry. The industrial sectors for each condition of use category can be found in 5.3.68Appendix D.

2.3.1.2 E-FAST 2014 Equations

2.3.1.2.1 Surface Water Concentrations

EPA used E-FAST 2014 (U.S. EPA 2014b) 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 (U.S. EPA 2014b) 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 µg/L)
WWR	=	Chemical release to wastewater (kg/day)
WWT	=	Removal from wastewater treatment (%)
SF	=	Estimated flow of the receiving stream (MLD, Million Liters per Day)
CF1	=	Conversion factor (10^9 µg/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 (U.S. EPA 2014b) 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 (U.S. EPA 2014b) 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 µg/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 used for the water body (typically between 1 and 200)
CF1	=	Conversion factor (10^9 µg/kg)
CF2	=	Conversion factor (10^6 L/day/MLD)

2.3.1.2.2 Days of COC Exceedance

The Probabilistic Dilution Model (PDM) portion of E-FAST 2014 (U.S. EPA 2014b) was also run for free-flowing water bodies, which predicts the number of days per year a chemical's concentration of concern (COC) in an ambient water body will be exceeded. 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 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 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 per year (see required inputs below).

2.3.1.3 E-FAST 2014 Outputs

There are two main results generated from E-FAST (U.S. EPA 2014b) 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 both the 7Q10 and harmonic mean stream flows. The 7Q10 stream flow is the lowest consecutive 7-day average flow during any 10-year period. The harmonic mean stream flow, a less conservative value, is the inverse mean of reciprocal daily arithmetic mean flow values. 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 (U.S. EPA 2014b) (discussed below). Estimates from this calculation method are reported for the 10^{th} percentile harmonic mean and 10^{th} percentile 7Q10 stream flows.

2.3.2 Surface Water Monitoring Data Gathering Approach

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

2.3.2.1 Method for Systematic Review of Surface Water Monitoring Data

EPA conducted a review of published literature to identify studies reporting concentrations of PCE in surface water associated with background levels of contamination or potential releases from facilities that manufacture, process, use and/or dispose of PCE in the United States. Studies clearly associated with releases from Superfund sites, improper disposal methods, and landfills were considered off-PECO and excluded from data evaluation and extraction. The systematic review process is described in detail in Section 1.5. A total of 26 surface water studies were extracted and the results are summarized in Section 2.3.4.2.3. A total of 3 U.S. surface water studies were extracted and the results are summarized in Section 2.3.4.2.3

2.3.2.2 Method for Obtaining Surface Water Monitoring Data from WQX/WQP

The primary source for the occurrence of PCE in surface water is monitoring data retrieved from the Water Quality Portal (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 PCE 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 1960s 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, (Nwqmc 2017))

2.3.2.2.1 Data Retrieval from WQP

Surface water data for PCE were downloaded from the WQP (Nwqmc 2017) on October 3, 2018. The WQP can be searched through three different search options: Location Parameters, Site Parameters, and Sampling Parameters. The PCE data were queried through the Sampling Parameters search using the Characteristics parameter (selected "Tetrachloroethene (NWIS, STORET)") and Date Range parameter (selected "01-01-2008 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. An example of WQP search option is shown below in Figure 2-3.

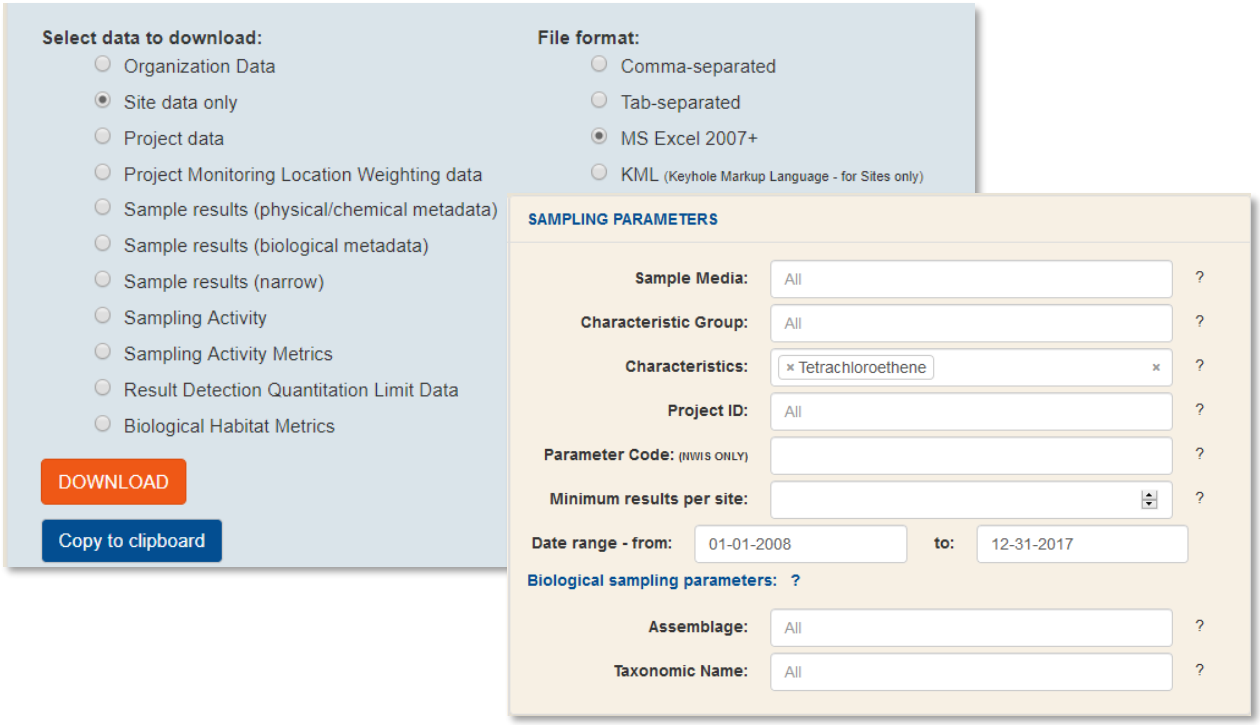


Figure 2-3. WQP Search Option. Surface water data were obtained from the WQP by querying the Sampling Parameters search option for the characteristic (STORET data), Parameter Code (NWIS data), and date range parameter.

2.3.2.2.2 Data Filtering and Cleansing

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 were only for the media of interest (i.e., surface water), were not quality control samples (i.e., field blanks), were of high analytical quality (i.e., no quality control issues, sample contamination, or estimated values), and were not associated with contaminated sites (i.e., Superfund).

The following filtering to obtain the final dataset, the 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 0.3 µg/L).

2.3.3 Geospatial Analysis Approach

Using 2016 data, the measured surface water concentrations from the WQP and predicted concentrations from the modeled facility releases were mapped in ArcGIS to conduct a watershed analysis at the HUC 8 and HUC 12 level. The purpose of the analysis was to identify if any 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 to possible exclude these monitoring sites from the analysis. A U.S. scale map was developed to provide a spatial representation of the measured and predicted concentrations. HUCs with co-located monitoring stations and facility releases were identified and examined further. Maps were developed on a U.S. scale to provide a spatial display of the concentrations, as well as at the HUC scale to focus on co-located monitoring stations and facility releases.

2.3.3.1 Geographic Coordinates

The location of the monitoring stations was determined from the geographic coordinates (latitude and longitude) provided in WQP. Releases from facilities were located based on the geographic coordinates for the NPDES, TRI, and/or FRS of the mapped facility, as provided by FRS. For indirect dischargers, the location of the receiving facility was mapped if known. If not known, the location of the indirect discharger was mapped. Superfund sites in 2016 were identified and mapped using geographic coordinates of the "front door", as reported in the Superfund Enterprise Management System (SEMS) database in Envirofacts,(U.S. EPA 2014d).

2.3.4 Environmental Exposure Results

In the section below, EPA summarizes what was identified in the evaluation of PCE in surface water. To determine what potential PCE occurrence there is in surface water, EPA evaluated both measured and modeled data using various approaches and methods. In the evaluation of PCE there are certain limitations that need to be accounted for when interpreting PCE exposure in the environment.

2.3.4.1 Aquatic Environmental Exposures

2.3.4.1.1 Predicted Surface Water Concentrations: E-FAST 2014 Modeling

A summary of the surface water concentration estimates modeled using E-FAST 2014 (U.S. EPA 2014b), based on the lifecycle release analysis for the year 2016, is summarized by OES category in Table 2-6 through Table 2-8. For the maximum release scenario (200-365 days of release/year), surface water concentrations under 7Q10 flow conditions ranged from 9.6E-09 to 135 ppb (Table 2-6). For the 20 days of release/year scenario for direct dischargers, surface water concentrations under 7Q10 flow conditions ranged from 4.0E-06 to 397 ppb (Table 2-7). For comparison purposes, indirect releases to non-POTW WWTPs were also modeled for the 20 days of release/year scenario, resulting in surface water concentrations of 1.0E-02 to 2034 ppb (Table 2-8). On a per facility basis, the 20 day release scenario yielded higher surface water concentrations than the maximum days of release scenario.

Reported loadings were used to model surface water concentrations with E-FAST 2014 (U.S. EPA 2014b). E-FAST was run using no further removal for wastewater treatment, this is appropriate for direct release DMR data because DMRs are "submitted from facilities that have NPDES permitted outfalls (which in most cases are discharges to surface waters)" (https://echo.epa.gov/trends/loading-tool/resources/faq), and the top indirect dischargers were themselves wastewater treatment facilities, reporting post-treatment release to surface water. TRI reporting facilities must identify the name of water body (or receiving POTW) into which the TRI chemical is being discharged.(https://www.epa.gov/toxics-release-inventory-tri-program/descriptions-tri-data-terms-text-version, (U.S. EPA 2020m)) data may be transferred through pipes or sewers to POTWs (18/24 top releasers identified as release to surface water,

others were assumed to be surface water releases, using SIC code) National Pollutant Discharge Elimination System (NPDES) permit codes were used to identify reach and flow characteristics for discharges. If a NPDES code was not identified, the most applicable SIC (Standard Industrial Classification) code was used. Surface water estimates were generated assuming an acute scenario of a single day release, and chronic scenarios of 20 and 250 days of release. Wastewater treatment plants and water pollution control plants were only assessed for chronic scenarios (20 and 250 days of release).

Table 2-6 Summary of Surface Water Concentrations by OES for Maximum Days of Release Scenario

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (µg/L)	
		Min	Max
Processing as a Reactant	18	2.9E-05	5.0
OTVD	17	3.4E-06	5.9
Industrial Processing Aid	14	2.4E-05	11
Waste Handling, Disposal, Treatment, and Recycling	13	9.6E-09	34
Manufacturing	10	8.0E-06	18
Other Industrial Uses	8	1.7E-03	31
Other Commercial Uses	7	1.2E-03	3.9E-01
Chemical Maskant	5	5.3E-04	2.8E-01
Import/Repackaging	4	4.0E-07	28
Incorporation into Formulation	4	2.6E-04	135
Dry Cleaning (industrial only)	2	2.2E-02	1.1E-01
Commercial Dry Cleaning Sites	1	3.6E-02	3.6E-02
Overall	**103**	**9.6E-09**	**135**

1. Maximum and central annual release amounts were available for four facilities/sites (Axiall Corporation, Greenchem, Solvents & Chemicals, and Commercial Dry Cleaning Sites). This summary table only compiles the high-end release amount.

Table 2-7 Summary of Surface Water Concentrations by OES for 20 Days of Release Scenario for Direct Releaser Facilities

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (µg/L)	
		Min	Max
Processing as a Reactant	17	7.2E-04	100
OTVD	16	1.3E-03	77
Industrial Processing Aid	12	6.6E-01	170
Other Industrial Uses	8	2.1E-02	397
Other Commercial Uses	7	2.1E-02	4.6

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (µg/L)	
		Min	Max
Manufacturing	5	1.2E-04	99
Waste Handling, Disposal, Treatment, and Recycling	5	6.4E-01	6.0
Chemical Maskant	3	4.6E-03	1.3
Import/Repackaging	3	4.0E-06	2.1E-02
Dry Cleaning (industrial only)	2	3.9E-01	1.7
Overall	**78**	**4.0E-06**	**397**

Table 2-8 Summary of Surface Water Concentrations by OES for 20 Days of Release Scenario for Indirect Releaser Facilities

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (µg/L)	
		Min	Max
Import/Repackaging	1	359	359
Incorporation into Formulation	2	1.0E-02	2034
Manufacturing	1	5.6E-02	5.6E-02
Waste Handling, Disposal, Treatment, and Recycling	4	1.7	436
Overall	**8**	**1.0E-02**	**2034**

2.3.4.1.2 Characterization of Modeled Releases

As discussed in Section 2.2.1.1, releases of PCE were determined from three data sources (TRI, DMRs, and CDR) for the 2016 calendar year, and assigned to 16 TSCA condition of use COU categories. Overall, modeling was conducted on 94 unique active releasing facilities plus one industry with sites nationwide (12,822 commercial dry cleaning sites). As some facilities may be in more than one OES category, and multiple facilities had both direct and indirect releases, a total of 103 facilities releases were modeled for both the maximum days of release and 20 days of release scenarios, as appropriate. The 94 active releasers were located in 28 states; states with the highest number of facilities (5 to 14 each) were TX, LA, IL, CO, CA, NY, and OH. The remaining 21 states had 1 to 4 facilities each. Figure 2-4 gives a graphical representation of the number of active releasers were for each state.

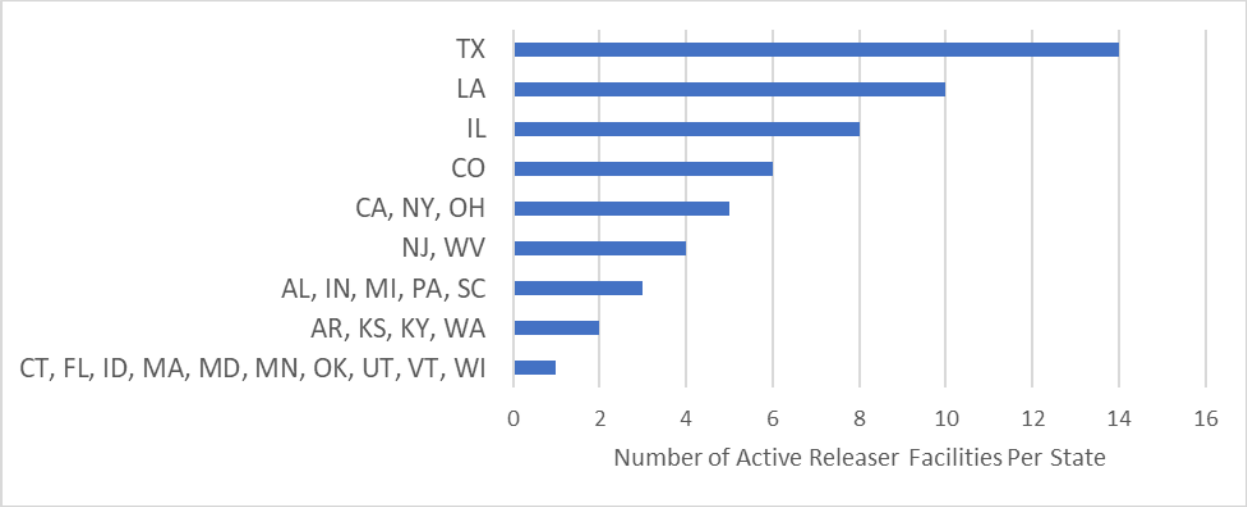


Figure 2-4. Distribution of Active Facility Releases Modeled

The location of the actual releases, when accounting for indirect dischargers, occurred in 27 states (all states as the active releaser, except CT). With respect to watersheds, the releases occurred across 66 HUC-8 areas and 82 HUC-12 areas. Over three quarters of the HUCs with facilities contained only 1 release location (76% for HUC-8 and 93% for HUC-12). The remaining HUCS contained 2 to 5 release locations each.

Direct and indirect dischargers accounted for 76% and 24% of the total releases modeled, respectively. Site-specific waterbody flow/dilution data (identified via NPDES) were available in E-FAST 2014 (U.S. EPA 2014b) for the majority of the releases (51%); surrogate site-specific waterbody flow/dilution data were identified for 6% of the cases; and the remaining cases (43%) were run using a representative industry sector SIC code. For releases modeled with a NPDES (including a surrogate NPDES), surface water concentrations were calculated for free-flowing water bodies in 81% of the cases, and still water bodies for the remaining cases (19%). Figure 2-5 gives a graphical representation of the modeled releases described above.

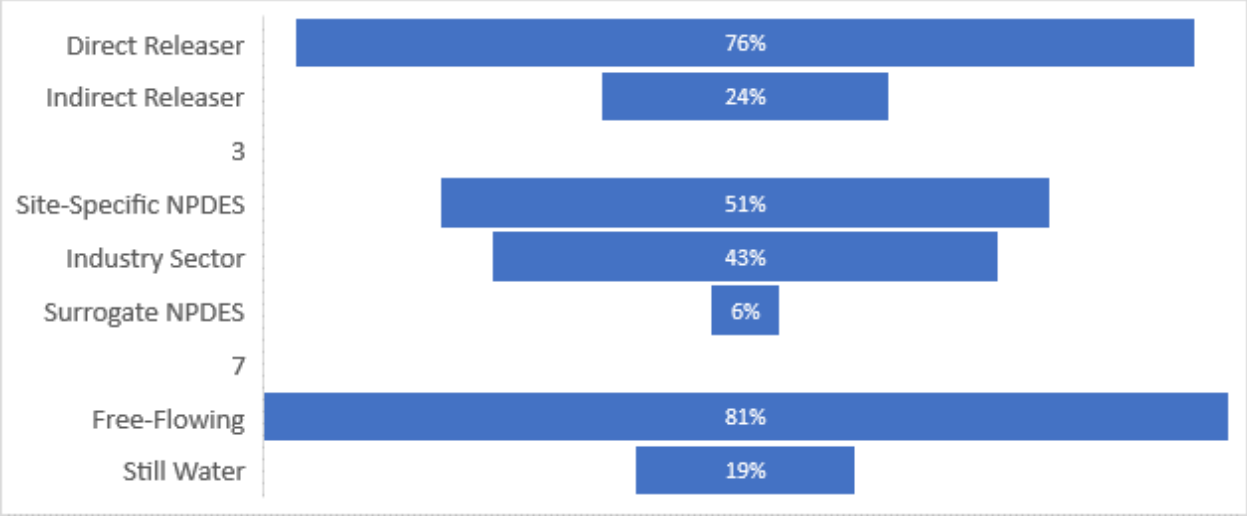


Figure 2-5. Modeled Release Characteristics (Percent Occurrence)

The predicted surface water concentrations for 65 modeled releases exceeded the lowest COC, and the PDM days of exceedance for 41 modeled releases was 20 days or more. In general, facilities with exceedances were facilities that had higher annual release amounts. Many releases, but not all, were modeled using surrogate stream flows based on the industry sector. Concentrations calculated using surrogate stream flows could be refined with the use of site-specific data.

For indirect releasers, Lord Corp in Saegertown, PA (OES: Incorporation into Formulation), had the highest surface water concentrations (both maximum days of release and 20 days of release scenarios). The annual release at this facility was the highest of all active releasers, and generally was an order of magnitude higher than all other releases. Stream flows for the receiving facility (EQ DETROIT INC, as determined from TRI) was not available in E-FAST (U.S. EPA 2014b) and therefore the indirect release was modeled using a surrogate industry sector (SIC Code Option).

For direct releasers, GM Components Holdings LLC in Lockport, NY (OES: OTVD), had the highest surface water concentrations (both maximum days of release and 20 days of release scenarios). This facility had an annual release amount significantly lower than Lord Corp in Saegertown, PA described above, but was modeled using site-specific stream flow data for a free-flowing waterbody. A detailed summary table by facility is provided in the supplemental file "Risk Evaluation for PCE Data Extraction for Consumer and Aquatic Exposure Monitoring Studies".

2.3.4.2 Monitored Surface Water Concentrations

2.3.4.2.1 Measured Surface Water Concentrations from WQX/WQP

A summary of the WQX data obtained from the WQP is provided in Table 2-9 below for years 2013-2017. Per year, the cleansed datasets evaluated contained between 171 and 512 surface water samples collected from 89 to 193 unique monitoring stations. Detection frequencies were low, ranging from 5.5E-01 to 7.6%. Concentrations ranged from not detected (ND; <2.6E-02 to 5) to 9.2E-02 µg/L in 2013, ND (<2.2E-02 to 5) to 1.6 µg/L in 2014, ND (<3.4E-02 to 1.8) to

3.2E-02 µg/L in 2015, ND (<2.8E-02 to 5) to 5.2E-02 µg/L in 2016, and ND (<3.6E-02 to 5) to 6.2E-01 µg/L in 2017. The temporal trend based on the average and maximum concentrations of all samples is graphically presented in Figure 2-6**.** A peak was observed in 2014, 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 2-9. Measured Concentrations of PCE in Surface Water Obtained from the Water Quality Portal: 2013-2017[8]

		Concentration in All Samples (µg/L)			Concentrations (µg/L) in Only Samples Above the Detection Limit		
Year	**Detection Frequency**	**No. of Samples (No. of Unique Stations)**	**Range[9]**	**Average ± Standard Deviation (SD)**	**No. of Samples (No. of Unique Stations)**	**Range**	**Average ± SD[10]**
2013	0.5%	366 (172)	ND (2.6E-02 to 5) to 9.2E-02	2.3E-01 ± 5.8E-01	2 (2)	7.2E-02 to 9.2E-02	8.2E-02 ± 1.4E-02
2014	7.6%	512 (193)	ND (2.2E-02 to 5) to 1.6	1.9E-01 ± 5.0E-01	39 (19)	1.1E-02 to 1.6	2.0E-01 ± 3.5E-01
2015	1.7%	347 (166)	ND (3.4E-02 to 1.8) to 3.2E-02	2.0E-01 ± 1.7E-01	6 (2)	1.7E-02 to 3.2E-02	2.5E-02 ± 6.0E-03
2016	3.5%	201 (91)	ND (2.8E-02 to 5) to 5.2E-02	2.9E-01 ± 7.6E-01	7 (4)	1.4E-02 to 5.2E-01	2.9E-02 ± 1.3E-02
2017	5.9%	171 (89)	ND (3.6E-02 to 5) to 6.2E-01	3.4E-01 ± 7.5E-01	10 (5)	1.8E-02 to 6.2E-01	2.4E-01 ± 2.6E-01
All 5 Years	4.0%	1597 (454)	ND (2.2E-02 to 5) to 1.6	2.3E-01 ± 5.5E-01	64 (27)	1.1E-01 to 1.6	1.7E-01 ± 2.9E-01

[8] Data were downloaded from the Water Quality Portal ((Nwqmc 2017), www.waterqualitydata.us) on 10/3/2018 by selecting "Tetrachloroethene (NWIS, STORET)" for the Characteristic. Results were reviewed and filtered to obtain a cleansed dataset (i.e., samples/sites were eliminated if identified as estimated, quality control, media type other than surface water, Superfund, landfill, failed laboratory quality control, etc.).

[9] ND = Not Detected. Reported detection limits varied between samples, as shown in parenthesis.

[10] 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 ½ the average of the reported detection limits in all samples (average = 0.3 µg/L).

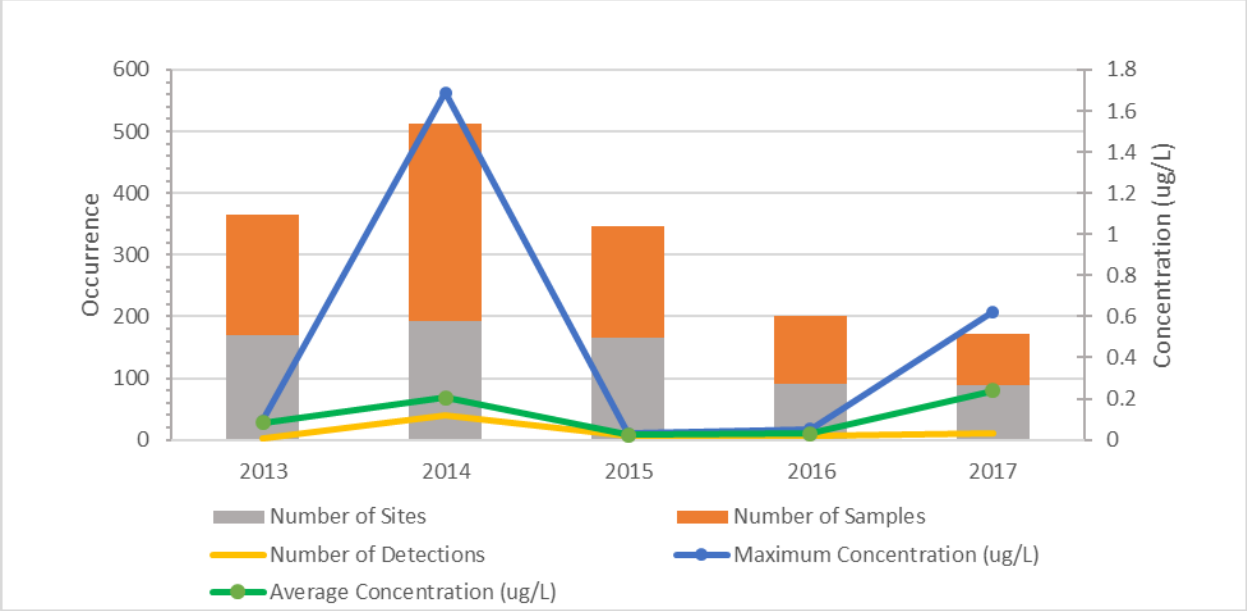


Figure 2-6. Temporal WQX Sampling and Surface Water Concentration Trends: 2013 - 2017

The quantitative ecological assessment used the 2016 data set only. For the 2016 data, only 7 samples from 4 monitoring sites (all in Tennessee) had PCE concentrations above the detection limit. The concentrations ranged from 1.4E-02 to 5.2E-02 µg/L, which are below the lowest COC of 1.4 µg/L.

Only one sample in the 2013-2017 dataset (Sample ID nwisnc.01.01400387) had a concentration that exceeded the lowest COC of 1.4 µg/L. This sample was collected in 2014 from Marsh Creek near New Hope, NC (Site ID USGS-0208732885) and had a concentration of 1.6 µg/L. The sample site was not co-located with any 2016 active releaser facility.

2.3.4.2.2 Characterization of WQP Data

The original dataset downloaded contained 7,661 samples for years 2013 through 2017. Following the filtering and cleansing procedure, only 21% of the samples remained (n = 1,604). The majority of the samples (94%) were excluded because they were an off-topic media (i.e., groundwater, artificial, bulk deposition, leachate, municipal waste, or stormwater) or location type (i.e., landfill, subsurface, spring, or well). A smaller number of samples were excluded because they were quality control samples (~2%), estimated values (~1%), or had other quality control issues (<1%). Samples associated with one Superfund site (Palermo Wellfield Superfund Site) were also excluded.

For the 2016 cleansed dataset (n = 201 samples), observations were made in 19 states/territories (AZ, IN, KS, LA, MD, MI, MN, MO, NJ, NM, NY, OH, OR, PA, PR, TN, TX, UT, and WI) at 91 unique monitoring sites, with 1 to 6 samples collected per sampling site. On a watershed level, observations were made in 47 HUC-8 areas and 68 HUC-12 areas. The majority of HUCs had only one monitoring site (68% for HUC-8; 78% for HUC-12). Up to 9 sites were present in a

HUC-8 and up to 4 sites in a HUC-12. A list of individual HUCs, including the number of monitoring sites and samples in each HUC, is provided in 5.3.68Appendix D, Table_Apx D-2 for HUC-8 and Table_Apx D-3 for HUC-12

An analysis of the 2016 cleansed dataset was also conducted to determine if any monitoring station may be associated with Superfund sites that could be contributing to PCE releases, and thus would not fall under the scope of this TSCA evaluation. For samples with concentrations above the detection limit, there are four monitoring stations within 5 miles of a Superfund site. However, there is no hydrologic connectivity as all four are located in a HUC that is adjacent to the superfund site and not in the same HUC itself. For monitoring stations that were also co-located in the same HUC as a facility, a search was also conducted for Superfund sites within 1 mile. There are two co-located monitoring stations within one mile of a superfund site: USGS-04092750 and USGS-04095090. While USGS-04092750 is found in the same HUC as a facility it is on a separate portion of the stream network from the facility. The other station USGS-04095090, is however immediately downstream of a superfund site and is closer to it (at 0.24 miles) than it is to the upstream facility (at 2.3 miles). Concentrations at this site were not-detect (sampled in 2015-2017). No monitoring data from WQP was excluded based on proximity to a Superfund site through this Superfund analysis.

2.3.4.2.3 Measured Concentrations of PCE from Published Literature

EPA's review of published literature yielded only a minimal amount of surface water monitoring data for PCE in the U.S.; a summary of the individual studies is provided in Table 2-2-10.. Only three studies were identified (USGS 2006), (USGS 2003), and (Singh et al. 1983)), which encompassed 416 surface water samples collected from rivers and oceans between 1979 and 2001. The reported concentrations of PCE ranged from below the detection limit (1.0E-04 to 0.2) to 5.5 µg/L, with reported central tendency values ranging from <0.2 to 0.7 µg/L. The overall detection frequency is a maximum of approximately 12%. The maximum concentration was collected during a large nationwide survey of surface water for drinking water sources (rivers and reservoirs) between 1999 and 2000 (USGS 2006)), in which PCE was only detected in 3 of 375 samples. The next highest reported concentration was only 2.8E-03 µg/L, from a sample collected in the Eastern Pacific Ocean in 1979-1981 (Singh et al. 1983).

Table 2-2-10. Levels of PCE in U.S. Surface Water from Published Literature

Country	Site Information	Date Sampled	N (Detection Frequency)	Concentration (µg/L)		HERO/ Source	Data Quality Score
				Range	Central Tendency (±SD)		
United States	Anchorage, AK; Chester Creek (6 urban sampling sites)	1998-2001	11 (0)	All ND (<0.2)		3975042	Medium
United States	Nation-wide; Surface water for drinking	1999-2000	375 (8.0E-03)	ND (<0.2)–5.5	NR	3975046	Medium

Country	Site Information	Date Sampled	N (Detection Frequency)	Concentration (µg/L)		HERO/ Source	Data Quality Score
				Range	Central Tendency (±SD)		
	water sources (rivers and reservoirs)						
United States	Eastern Pacific Ocean (California, US to Valparaiso, Chile)	1979-1981	30 (0.9)	ND (<1.0E-04) – 2.8E-03	Mean: 0.7 (7.0E-04); Median: 4.0E-04	29192	Medium

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

2.3.4.2.4 Geospatial Analysis Comparing Predicted and Measured Surface Water Concentrations

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 can be found in Section 4, Figure 4-1 and Figure 4-2 (east and west US, respectively) for the maximum days of release scenario, and in Figure 4-3 and Figure 4-4 (east and west US, respectively) for the 20-days of release scenario. Overall, there are 33 U.S. states/territories with either a measured concentration or a predicted concentration; at the watershed level, there are 109 HUC-8 areas and 149 HUC-12 areas with either measured or predicted concentrations. Appendix D Table_Apx D-2 and Table_Apx D-3 provides a list of states/territories with facility releases (as mapped) and/or monitoring sites.

2.3.4.2.5 Co-location of PCE Releasing Facilities and Monitoring Stations

The co-occurrence of PCE releasing facilities and monitoring stations in a HUC is shown in Figure 2-7 (Little Arkansas and Rush-vermillion) and Figure 2-8 (Little Calument-Galien and Lower Grand). There are four HUC-8 areas that have both measured and predicted concentrations. 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. The collocated facilities and monitoring stations are briefly described below and summarized in

Table 2-11.

A. HUC 11030012 (Little Arkansas in Kansas) has one facility with modeled 7Q10 surface water concentrations ranging from 4.4E-02 to 6.6E-01 ppb, and 7 monitoring stations all with concentrations less than the reported detection limit (<0.1 ppb). The monitoring stations are over 20 miles downstream of the facility or are neither up nor downstream of the facility.

B. HUC 07040001 (Rush-Vermillion in Minnesota) has one facility with modeled 7Q10 surface water concentrations ranging from 2.8E-03 to 5.6E-02 ppb, and 1 monitoring station with a non-detect concentration (<0.1 ppb) that is located approximately 20 miles downstream of the facility.

C. HUC 04040001 (Little Calumet-Galien in Indiana) has one receiving facility with concentrations ranging from 0.1 to 1.7 ppb, and two monitoring stations with non-detect concentrations (<0.1 ppb). The monitoring stations are either over 2 miles downstream of the facility, or neither up nor downstream of the facility. It should be noted however, that a modeled receiving facility (East Chicago Municipal Sewage Treatment Plant; FRS 110006645531) is located just outside of the HUC on the south side. Monitoring site USGS-04092750 is located on a canal/ditch north of the facility; based on NHD water flows south from the monitoring site toward the facility.

D. HUC 04050006 (Lower Grand in Michigan), has one receiving facility with concentrations ranging from 0.1 to 1.0 ppb, and one monitoring station with non-detect concentrations (<0.1 ppb).

Figure 2-7. Colocation of PCE Releasing Facilities and WQX Monitoring Stations at the HUC 8 and HUC 12 Level

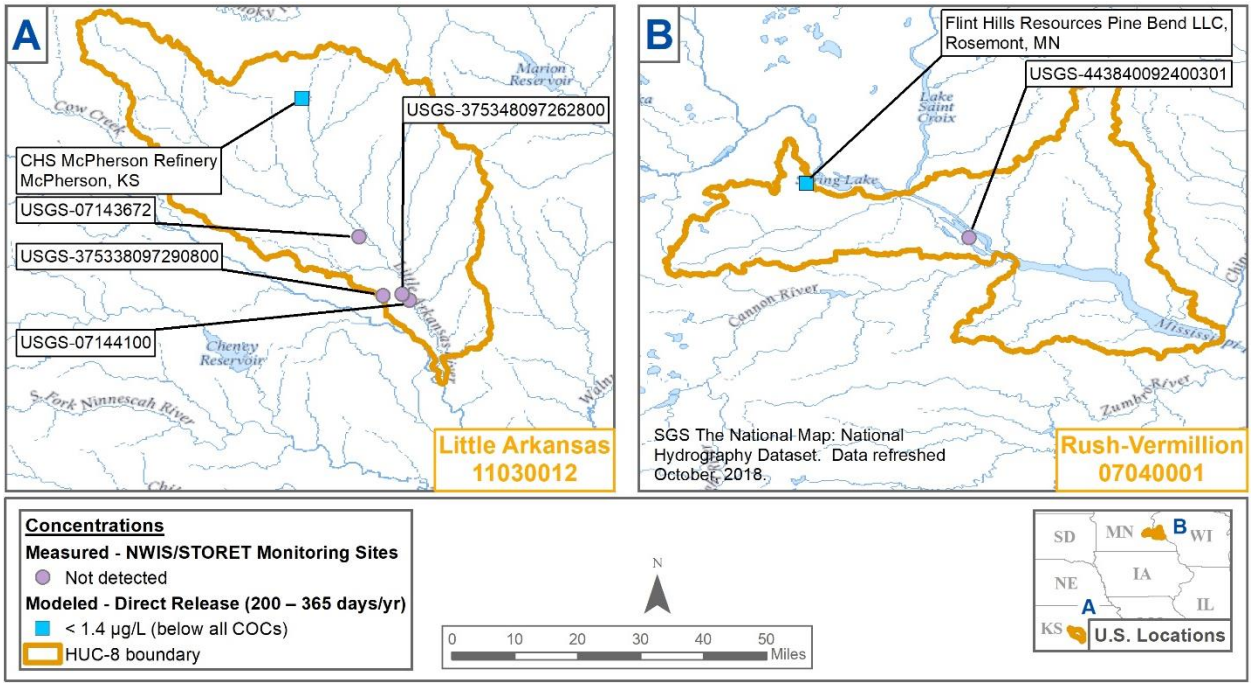
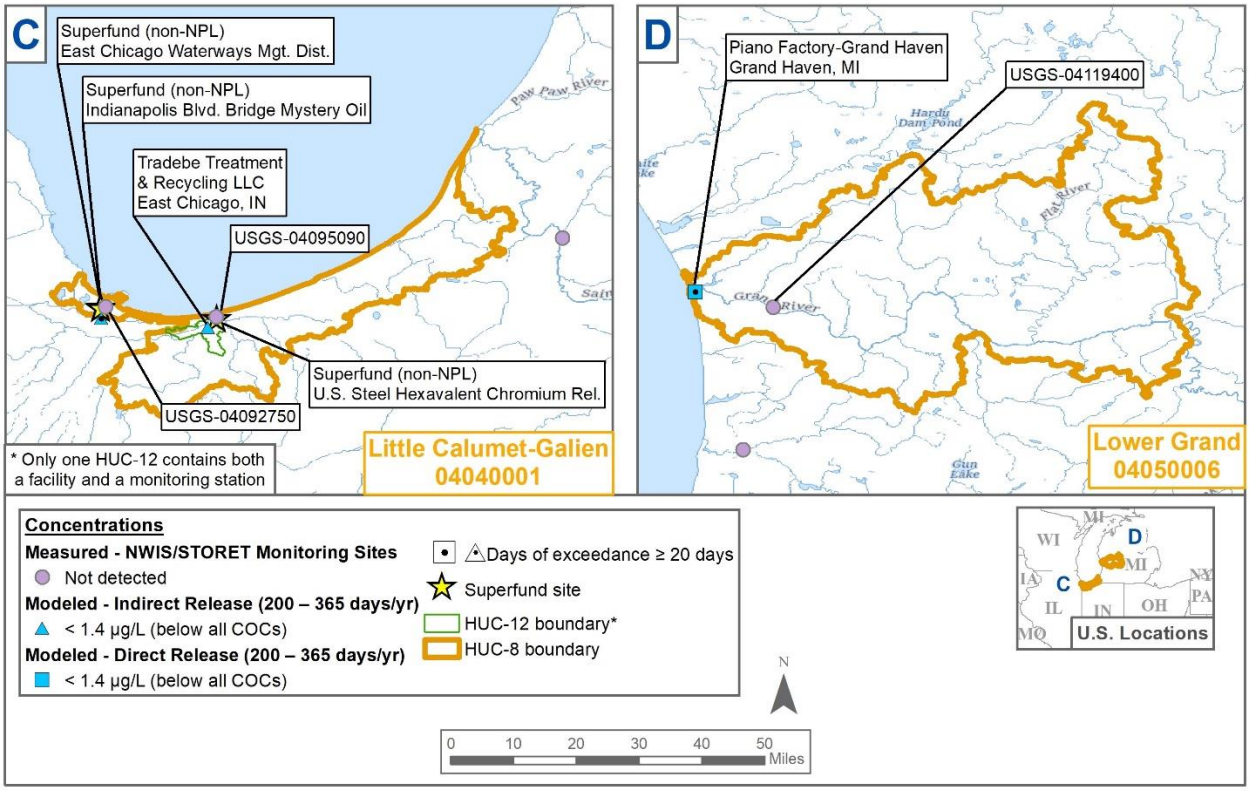


Figure 2-8. Colocation of PCE Releasing Facilities and WQX Monitoring Stations at the HUC 8 and HUC 12 Level



2488 **Table 2-11.** Co-Location of Facility Releases and Monitoring Sites within HUC 8 and HUC 12 Boundaries (Year 2016)

Map	HUC 8	Facilities in HUC		Monitoring Sites in HUC			
		Site (Name, Location, FRS)	**Modeled 7Q10 Surface Water Concentrations [a] (µg/L)**	**Monitoring Site ID**	**No. of Samples**	**Measured Surface Water Concentrations (µg/L)**	**Location Relative to Facility[b] (Miles)**
A	11030012 Little Arkansas	CHS McPherson Refinery McPherson, KS (FRS 110015862440)	300 days: 4.4E-02 20 days: 0.6	USGS-07143672	4	<0.1 (all)	Downstream/23
				USGS-07144100	4	<0.1 (all)	Downstream/34
				USGS-375338097290800	2	<0.1 (all)	Downstream/33
				USGS-375348097262800	2	<0.1 (all)	Downstream/33
				USGS-375338097290800	2	<0.1 (all)	Neither/42
B	07040001 Rush-Vermillion	Flint Hills Resources Pine Bend LLC *Rosemount, MN* (FRS 110000424611)	350 days:2.8E-03 20 days: 5.6E-02	USGS-443840092400301	1	<0.1	Downstream/20
C	04040001 Little Calumet-Galien	Tradebe Treatment & Recycling LLC East Chicago, IN (FRS 110000397874) Receiving Facility (modeled site): Advanced Waste Services of Indiana LLC/Covanta Environmental Solutions LLC Portage, IN	250 days: 0.1 20 days: **1.7[a]**	USGS-04095090[c]	1	<0.1	Downstream/2.3
				USGS-04092750[d]	4	<0.1 (all)	Neither/14

Map	HUC 8	Facilities in HUC		Monitoring Sites in HUC			
		Site (Name, Location, FRS)	Modeled 7Q10 Surface Water Concentrations [a] (µg/L)	Monitoring Site ID	No. of Samples	Measured Surface Water Concentrations (µg/L)	Location Relative to Facility[b] (Miles)
		(FRS 110020159852)					
D	04050006 Lower Grand	Piano Factory-Grand Haven Grand Haven, MI (FRS 110006739832)	260 days: 0.1***** 20 days: 1.0	USGS-04119400	4	<0.1 (all)	Upstream/10

[a] Concentrations above the COC of 1.4 µg/L are shown in bold. Concentrations leading to modeled days of exceedance ≥20 days are indicated by an asterisks (*).

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

[c] The HUC 8 co-located facility and monitoring station are also in the same HUC 12 (040400010509; Willow Creek-Burns Ditch).

[d] The East Chicago Municipal Sewage Treatment Plant (FRS 110006645531), which receives wastewater from Safety Kleen Systems, Inc. in East Chicago, IN is not located in the HUC, but is located just south of the HUC, near monitoring site USGS-04092750. This monitoring site is located on a canal/ditch, and according to NHD, the water flows south from the monitoring site toward the facility.

2.3.4.3 Biomonitoring Data

EPA identified blood biomonitoring measurements from multiple sources. The most comprehensive source is the National Health and Nutrition Examination Survey (NHANES) conducted by CDC's National Center for Health Statistics (NCHS). The survey is "a complex, stratified, multistage, probability-cluster design survey" designed to collect data on the health and nutrition of a representative sample of the US population. NHANES measured PCE in whole blood of males and females ages 12+ years. In the Fourth Report on Human Exposure to Environmental Chemicals (CDC 2017), statistics were reported for the 50th, 75th, 90th, and 95th percentiles for 2-year cycles starting in 2001 through 2008. Sample sizes ranged from 978 (2001-2002) to 2,940 (2005-2006). The concentrations in all samples were less than the limit of detection (0.048 ng/mL) at the 50th percentile for all years. At the 95th percentile, concentrations ranged from 9.4E-02 µg/L (2007-2008) to 1.9E-01 µg/L (2001-2002).

For 1999-2004 (n=2577), the mean sample concentration was 8.1E-02 µg/L, and the median sample concentration was 3.4E-02 µg/L. This study also reported regression statistics, coefficients, and trends over time for each chemical reported. Another source (Sexton et al. 2005), measured concentrations of PCE in whole blood from 150 children from two poor, minority neighborhoods in Minneapolis, Minnesota in four periods during 2000-2001. These samples were collected as part of the School Health Initiative: Environment, Learning, Disease (SHIELD) study. PCE was detected in 37 to 63% of the samples, with concentrations ranging from 2.0E-02 – 3.0E-02 ng/mL (10th percentile) to 0.1-0.8 ng/mL (99th percentile). The limit of detection was 2.2E-02 ng/mL. The SHIELD study also collected 2-day, integrated personal air samples. Blood samples were also collected as part of the National Human Exposure Assessment Survey (NHEXAS) Phase I conducted by EPA (Clayton et al. 1999). Samples were collected from 147 people in six states (IL, IN, OH, MI, MN, and WI) in 1995-1997. PCE was detected in 37% of the samples, with a mean of 0.2 ng/mL, a 50th percentile of 5.0E-02 ng/mL, and a 90th percentile of 0.1 ng/mL. NHEXAS Phase I also collected indoor air and personal air samples. PCE concentrations in blood were similar between the NHANES, SHIELD, and NHEXAS surveys conducted between 1995 and 2016.

In addition to blood samples, NHANES also collected urine samples for the PCE metabolite N-Acetyl-S-(trichlorovinyl)-L-cysteine. Samples were collected for males and females ages 6+ years. Statistics were reported for both uncorrected urine concentrations and creatine corrected urine concentrations. Data were reported for the survey years 2011-2012, and all samples measured (n=2,464-2,466) were below the detection limit of 3.0 µg/L. The NHANES urine metabolite data for PCE was also used in a 2015 study analyzing the reported data to develop means and other descriptive statistics (Jain, 2015). In that paper, the urinary metabolite TCVMA was reported in measurements of male (n=203) and female children (n=214) in 2011 and 2012. The mean concentration for male children was reported as 6.9 ng/mL and 6.4 ng/mL for female children. The 95% confidence interval around the mean was reported as 5.8 to 8.4 ng/mL for male children and 5.2 to 8.0 ng/mL for female children

Breath samples were also collected as part of the Total Exposure Assessment Methodology (TEAM) Study (Wallace 1987), which also collected concurrent personal inhalation monitoring samples and outdoor air samples. In Phase II and III of the study conducted between 1981 and

1984, samples were collected from adults conducting normal daily activities in industrial/chemical manufacturing and /or petroleum refining regions of the US, including Elizabeth and Bayonne, NJ, Los Angeles, CA, and Contra Costa, CA (n= 660). Arithmetic means ranged from 8.3 to 13 µg/m^3, with detection in 58 to 100% of samples.

2.3.4.4 Assumptions and Key Sources of Uncertainty for Environmental Exposures

The WQP Tools contains data from USGS-NWIS and STORET databases, and is one of the largest environmental monitoring databases in the U.S. (Nwqmc 2017); 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 of all of these shortcomings, 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 PCE 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 very few instances (*i.e.*, less than 0.01 percent) where measured PCE levels in the ambient environment exceeded the identified concentrations of concern for water or organisms (1.4 ppb). It is also important to note that only a few USGS-NWIS and STORET monitoring stations aligned with the watersheds of the PCE 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. To better characterize instream concentrations of PCE in the environment and provide for more robust confirmation of our modeled results, we would support the collection of collocated instream measurements with known discharging facilities.

The DMR, TRI and CDR databases represent comprehensive sources of environmental release data for the US; however, there are limitations and assumptions involved. These data are self-reported by facilities and subject to minimum reporting thresholds; therefore, they may not capture releases from smaller facilities (*i.e.*, environmental releases may be underestimated). Some of the reported information may be inaccurate because it reflects approximations rather than actual emissions or release data. TRI is based on mass balances and emission factors, whereas DMR is based on representative pollutant monitoring data at facility outfalls (mg/L) and corresponding wastewater discharge (million gallons per day). The assumed maximum days per year of release from each facility is uncertain and may in some cases lead to underestimation of daily release rates.

Use of release information from facility data used 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.

A major limitation associated with use of the E-FAST 2014 (U.S. EPA 2014b) model relates to the assumptions made regarding missing information that was required for model input, such as site-specific streamflow data. When site-specific or surrogate site-specific stream flow data were not available, flow data based on a representative industry sector was used in the assessment. This includes cases where a receiving facility for an indirect release could not be determined. Additionally, the data currently available in E-FAST 2014 (U.S. EPA 2014b) are 15 to 30 years old. Although stream conditions do change over time, changes in the flow values are not expected to be drastic. More recent flow data are available through the National Hydrological Dataset (NHD). It is important to note however, that these limitations are unlikely to change the stated conclusions of this assessment because they are based on a series of conservative assumptions that likely overestimate exposure potential.

With respect to the geospatial analysis, a 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.

2.3.4.4.1 Confidence in Aquatic Exposure Scenarios

Confidence ratings for aquatic exposure scenarios are informed by uncertainties surrounding inputs and approaches used in modeling surface water concentrations. In Section 2.2.1.1, confidence ratings are assigned to these estimated daily releases (kg/site-day) on a per occupational exposure scenario (OES) basis and primarily reflect moderate confidence (one OES shows high confidence for this estimate). As these release estimates serve as the key inputs into the exposure mode and are therefore a key component of the overall aquatic exposure scenario confidence.

Other considerations that impact confidence in the aquatic exposure scenarios include the model used E-FAST 2014, (U.S. EPA 2014b) and its associated default and user-selected values and related uncertainties. As described in Section 4.1.2, there are uncertainties related to the ability of E-FAST 2014 (U.S. EPA 2014b) to incorporate downstream fate and transport; the likely number of release days from given discharging facilities; and, in some cases (i.e., when the NPDES for the discharging facility cannot be found within the E-FAST database), the applied stream flow distribution.

There are monitoring data available in surface water that reflect both near-facility and ambient (i.e., background) exposure levels in this media in the United States. Samples characterizing background levels in surface water ranged from non-detect (ND) to 310 µg/L, from both literature and the Water Quality Portal database.

2.4 Human Exposures

EPA evaluated acute and chronic exposures to workers by dermal and inhalation routes and occupational non-users (ONUs) by inhalation routes in association with PCE use in industrial and commercial applications. EPA also evaluated acute exposures to consumers by dermal and inhalation routes in association with PCE use in consumer applications. The assessed conditions of use are described above 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, subcategories for intermediate uses in industrial gas manufacturing, basic organic chemical manufacturing, and petroleum refineries may generally have similar worker activities, and EPA does not have data to distinguish whether workers are exposed differently for these subcategories. Therefore, EPA has grouped these intermediate 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 2-12 below.

Table 2-12 Crosswalk of Subcategories of Use Listed in the Problem Formulation Document to Exposure Scenarios Assessed in the Risk Evaluation

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
Manufacture	Domestic manufacture	Domestic manufacture	Section 2.4.1.6– Manufacturing	Manufacturing	N/A
	Import	Import	Section 2.4.1.7 – Repackaging[c]	Repackaging	N/A
Processing	Processing as a reactant/ intermediate	Intermediate in industrial gas manufacturing	Section 2.4.1.8 – Processing as a Reactant	Processing as Reactant/ Intermediate	N/A
		Intermediate in basic organic chemical manufacturing			
		Intermediate in petroleum refineries			
		Residual or byproduct reused as a reactant [d]			
	Incorporated into formulation mixture or reaction product	Cleaning and degreasing products	Section 2.4.1.9 – Incorporation into Formulation, Mixture, or Reactant Product	Incorporation into Formulation - Aerosol Packing; Incorporation into Formulation - Degreasing Solvent; Incorporation into Formulation - Dry Cleaning Solvent; Incorporation into Formulation - Miscellaneous	N/A
		Adhesive and sealant products			
		Paint and coating products			
		Other chemical products and preparations			

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
	Processing – Incorporated into articles	Plastic and rubber products	After further review, EPA determined that PCE is not incorporated into plastic articles but rather is used as a degreasing solvent at plastic manufacture sites; therefore, no exposure scenario was developed for incorporation into articles. Use of PCE as a degreasing solvent at plastic manufacture sites is assessed with other degreasing scenarios in Sections 2.4.1.10 through 2.4.1.13	N/A	N/A
	Repackaging[c]	Solvent for cleaning or degreasing	Section 2.4.1.7– Repackaging	Repackaging	N/A
		Intermediate			
	Recycling	Recycling	Section 2.4.1.26– Waste Handling, Disposal, Treatment, and Recycling	Disposal/Recycling	N/A

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
Distribution in commerce	Distribution	Distribution	Activities related to distribution (e.g., loading, unloading) are considered throughout the life cycle, rather than using a single distribution scenario.	N/A	N/A
Industrial use	Solvents (for cleaning or degreasing)	Solvents and/or Degreasers (cold, aerosol spray or vapor degreaser; not specified in comment)	See sections for specified degreasing and cleaning operations.	See sections for specified degreasing and cleaning operations.	N/A
		Batch vapor degreaser (e.g., open-top, closed-loop)	Section 2.4.1.10– Batch Open-Top Vapor Degreasing; Section 2.4.1.11– Batch Closed-Loop Vapor Degreasing	Open-top Vapor Degreasing; Closed Loop Vapor Degreasing	
		In-line vapor degreaser (e.g., conveyorized, web cleaner)	Section 2.4.1.12– Conveyorized Vapor Degreasing; Section 2.4.1.13– Web Degreasing	Conveyorized Vapor Degreasing; Web Degreasing	
		Cold cleaner	Section 2.4.1.14– Cold Cleaning	Cold Cleaning	
		Aerosol spray degreaser/cleaner	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants	Aerosol Degreasing/ Lubricants	

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
		Dry cleaning solvent	Section 2.4.1.16– Dry Cleaning and Spot Cleaning	Post-2006 Dry Cleaning (including spot cleaning); 4th/5th Gen Only Dry Cleaning (including spot cleaning)	
		Spot cleaner			
	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants; Section 2.4.1.20– Metalworking Fluids	Aerosol Degreasing/ Lubricants; Metalworking Fluid	N/A
	Adhesives and sealants	Solvent-based adhesives and sealants	Section 2.4.1.17– Adhesive, Sealants, Paints, and Coatings	Adhesives	N/A
	Paints and coatings including paint and coating removers	Solvent-based paints and coatings, including for chemical milling	Section 2.4.1.17 – Adhesive, Sealants, Paints, and Coatings; Section 2.4.1.18– Maskant for Chemical Milling	Paints/Coatings; Chemical Maskant	N/A

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
	Processing aids, not otherwise listed	Pesticide, fertilizer and other agricultural chemical manufacturing	Section 2.4.1.19– Industrial Processing Aid	Industrial Processing Aid	N/A
	Processing aids, specific to petroleum production	Catalyst regeneration in petrochemical manufacturing	Section 2.4.1.19– Industrial Processing Aid	Industrial Processing Aid	N/A
	Other uses	Textile processing	Section 2.4.1.22– Other Spot Cleaning/Spot Removers (Including Carpet Cleaning); Section 2.4.1.23– Other Industrial Uses	Other Spot Cleaning/Spot Removers (Including Carpet Cleaning); Other Industrial Uses	N/A
		Wood furniture manufacturing	Section 2.4.1.23– Other Industrial Uses	Other Industrial Uses	
		Laboratory chemicals	Section 2.4.1.25– Laboratory Chemicals	N/A – qualitative assessment	
		Foundry applications	Section 2.4.1.23– Other Industrial Uses	Other Industrial Uses	

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
Commercial/consumer use	Cleaning and furniture care products	Cleaners and degreasers (other)	Section 2.4.1.21– Wipe Cleaning and Metal/Stone Polishes; Section 2.4.1.22– Other Spot Cleaning/Spot Removers (Including Carpet Cleaning); Section 2.4.1.24 – Other Commercial Uses	Wipe Cleaning and Metal/Stone Polishes; Other Spot Cleaning/Spot Removers (Including Carpet Cleaning); Other Commercial Uses - Mold Release	Section 2.4.2.3.1-Aerosol Degreasers (includes: marine cleaner, degreaser, coil cleaner, electric motor cleaner, parts cleaner, cable cleaner, stainless steel polish, electrical/energized cleaner, wire and ignition demoisturants, electric motor cleaner; brake cleaners)
		Dry cleaning solvent	Section 2.4.1.16– Dry Cleaning and Spot Cleaning	Post-2006 Dry Cleaning (including spot cleaning);	Section 2.4.2.4-Dry Cleaned Articles

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
		Spot cleaner		4th/5th Gen Only Dry Cleaning (including spot cleaning)	Combined under Aerosol Cleaner
		Automotive care products (e.g., engine degreaser and brake cleaner)	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants	Aerosol Degreasing/ Lubricants	Section 2.4.2.3.1- Brake Cleaner
					Section 2.4.2.3.2- Parts Cleaner
		Aerosol cleaner			Section 2.4.2.3.3- Vandalism Mark & Stain Remover, Mold Cleaner, Weld Splatter Protectant
		Non-aerosol cleaner	Section 2.4.1.21– Wipe Cleaning and Metal/Stone Polishes	Wipe Cleaning and Metal/Stone Polishes	Section 2.4.2.3.4- Marble and Stone Polish (liquid)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants; Section 2.4.1.20 – Metalworking Fluids	Aerosol Degreasing/ Lubricants; Metalworking Fluid	Section 2.4.2.3.5- Cutting Fluid
					Section 2.4.2.3.6- Spray Lubricant and Penetrating Oil
	Adhesives and sealant chemicals	Adhesives for arts and crafts	Not assessed in occupational settings – consumer use only	N/A	Section 2.4.2.3.7- Adhesives (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant)
					Section 2.4.2.3.8 - Livestock Grooming Adhesive
		Light repair adhesives	Section 2.4.1.17– Adhesive, Sealants, Paints, and Coatings	Adhesives	Section 2.4.2.3.9- Column Adhesive, Caulk and Sealant

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
	Paints and coatings	Solvent-based paints and coatings	Section 2.4.1.17– Adhesive, Sealants, Paints, and Coatings	Paints/Coatings	Section 2.4.2.3.10-Outdoor watershield (liquid)
					Section 2.4.2.3.11-Coatings and primers (aerosol)
					Section 2.4.2.3.12-Rust Primer and Sealant (liquid)
					Section 2.4.2.3.13-Metallic Overglaze
	Other Uses	Carpet cleaning	Section 2.4.1.22– Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	Not found as consumer product
		Laboratory chemicals	Section 2.4.1.25– Laboratory Chemicals	N/A – qualitative assessment	Not assessed in consumer setting – occupational use only

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
		Metal (e.g., stainless steel) and stone polishes	Section 2.4.1.21 - Wipe Cleaning and Metal/Stone Polishes	Wipe Cleaning and Metal/Stone Polishes	Section 2.4.2.3.14-Marble and Stone Polish (wax)
		Inks and ink removal products	Section 2.4.1.24 – Other Commercial Uses	Other Commercial Uses - Printing	Ink removal combined under Aerosol Cleaner (vandalism and stain remover); use in printing inks discussed as "other use"
		Welding[e]	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants[b]	Aerosol Degreasing/ Lubricants	Combined under Aerosol Cleaner (weld splatter protectant)
		Photographic film	Section 2.4.1.24– Other Commercial Uses	Other Commercial Uses - Photographic Film	Not found as consumer product
		Mold cleaning, release and protectant products	Section 2.4.1.24 – Other Commercial Uses	Other Commercial Uses - Mold Release	Combined under Aerosol Cleaner (mold cleaner)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario	Associated Condition of Use in Risk Calculator	Consumer Exposure Scenario
Disposal[f]	Disposal	Industrial pre-treatment	Section 2.4.1.26 - Waste Handling, Disposal, Treatment and Recycling	Process Solvent Recycling and Worker Handling of Wastes	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			
		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 PCE in industrial and/or commercial settings.

[b] These subcategories reflect more specific uses of PCE.

[c] The repackaging scenario covers only those sites that purchase PCE or PCE containing products from domestic and/or foreign suppliers and repackage the PCE from bulk containers into smaller containers for resale. Sites that import and directly process/use PCE are assessed in the relevant condition of use. Sites that import and either directly ship to a customer site for processing or use or warehouse the imported PCE and then ship to customers without repackaging are assumed to have no exposures or releases and only the processing/use of PCE at the customer sites are assessed in the relevant conditions of use.

[d] EPA assessed PCE as a reactant where it was produced as a byproduct from EDC manufacture and reused as a reactant.
[e] Identified welding products were anti-spatter aerosol products; therefore, the assessment is included with the assessment of other aerosol products.
[f] Each of the conditions of use of PCE may generate waste streams of the chemical that are collected and transported to third-party sites for disposal, treatment, or recycling. Industrial sites that treat, dispose, or directly discharge onsite wastes that they themselves generate are assessed in each condition of use assessment. This section only assesses wastes of PCE that are generated during a condition of use and sent to a third-party site for treatment, disposal, or recycling.

2.4.1 Occupational Exposures

The following subsections describe EPA's approach to assessing occupational exposures and results for each condition of use assessed. For additional details on development of approaches and results refer to the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

2.4.1.1 Approach to Workers and Occupational Non-Users

As described in the *Problem Formulation of the Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro)*(U.S. EPA 2018d), for each condition of use, EPA endeavors to distinguish exposures for workers and occupational non-users (ONUs). Normally, a primary difference between workers and ONUs is that workers may handle PCE and have direct contact with the chemical, while ONUs are working in the general vicinity of workers but do not handle PCE and do not have direct contact with PCE being handled by the workers. The size of the area that ONUs may work can vary across each OES and across facilities within the same OES and will depend on the facility configuration, building and room sizes, presence of vapor barrier, and worker activity pattern. For example, an ONU can be a production employee whose workstation is located on the factory floor where a degreasing unit is installed. Absence of any vapor barrier (e.g., walls) between the degreaser and the rest of the factory, this "area" can be an entire factory floor. Alternately, the area can be in a specific room of a building where a chemical is handled (e.g., a room in a dry cleaning shop where the dry cleaning machine is installed and where dry cleaned loads are unloaded, pressed, and finished). Where possible, for each condition of use, EPA identified job types and categories for workers and ONUs.

EPA evaluated inhalation exposures to workers and ONUs, and dermal exposures to workers. EPA did not assess dermal exposures to ONUs as EPA does not expect ONUs to have routine dermal exposures in the course of their work. Depending on the condition of use, ONUs may have incidental dermal exposures due to surface contamination. However, data (e.g., frequency and amount of liquid on the skin after contact) were not identified to assess this exposure.

2.4.1.2 Number of Workers and Occupational Non-Users Approach and Methodology

Where available, EPA used CDR data to provide a basis to estimate the number of workers and ONUs. EPA supplemented the available CDR data using available market data; NAICS and SIC code data from TRI, DMR, and NEI sites identified for each condition of use (for number of sites estimates see Section 2.2.1.2.2); and analyzing Bureau of Labor Statistics (BLS) and U.S. Census data using the methodology described in the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). Where market penetration data and site-specific NAICS/SIC codes from TRI/DMR/NEI were not available, EPA estimated the number of workers using data from GSs and ESDs. For additional details on development of estimates of number of workers refer to Appendix A in the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

Table 2-13 presents the confidence rating of data that EPA used to estimate number of sites and workers.

Table 2-13. Data Evaluation of Sources Containing Number of Worker Estimates

Source Reference	Data Type	Data Quality Rating	Condition(s) of Use
(U.S. EPA 2016d)	Number of Workers	High	Manufacturing

(U.S. BLS 2016)	Number of Workers	High	Manufacturing; Repackaging; Processing as a Reactant; Incorporation into Formulation, Mixture, or Reaction Product; Cold Cleaning; Aerosol Degreasing and Aerosol Lubricants; Dry Cleaning and Spot Cleaning; Adhesives, Sealants, Paints, and Coatings; Chemical Maskants; Industrial Processing Aid; Other Industrial Uses; Laboratory Chemicals; Waste Handling, Disposal, Treatment, and Recycling
(U. S. Census Bureau 2015)	Number of Workers	High	
(OECD 2017a)	Number of Workers	N/A – ESD	OTVD, Closed-Loop Vapor Degreasing, Conveyorized Vapor Degreasing, Web Degreasing
(OECD 2011)	Number of Workers	N/A – ESD	Metalworking Fluids
(OECD 2017b)	Number of Workers	N/A – ESD	Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)
(U.S. EPA 1994a)	Number of Workers	N/A – GS	
(CARB 2000)	Market Penetration Data	High	Aerosol Degreasing and Aerosol Lubricants
(DLI/NCA 2017)	Market Penetration Data	High	Dry Cleaning

2.4.1.3 Inhalation Exposures Approach and Methodology

To assess inhalation exposure, EPA reviewed exposure monitoring data identified through the systematic review process (described in Section 1.5) and monitoring data provided to EPA by other government agencies (e.g., OSHA and DOD) and mapped them to specific conditions of use. Monitoring data used in the occupational exposure assessment include data collected by government agencies such as OSHA and NIOSH, and data found in published literature. For each exposure scenario and worker job category ("worker" or "occupational non-user"), where available, EPA provided results representative of *central tendency* and *high-end* exposure levels. For datasets with six or more data points, central tendency and high-end exposures were estimated using the 50th and 95th percentile value from the observed dataset, respectively. For datasets with three to five data points, the central tendency and high-end exposures were estimated using the median and maximum values. For datasets with two data points, the midpoint and the maximum value were presented. Finally, datasets with only one data point were presented as-is. For datasets including exposure data that were reported as below the limit of detection (LOD), EPA estimated the exposure concentrations for these data, following guidance in

EPA's *Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b)[11]. A dataset comprises the combined exposure monitoring data from all studies applicable to that condition of use.

For exposure assessment, personal breathing zone (PBZ) monitoring data were used to determine the time-weighted average (TWA) exposure concentration. The lone exception to this is exposures from mold release products (assessed in "Other Commercial Uses") where the assessment was made with area monitoring data as PBZ data were not available. TWA exposure concentrations are then used to calculate the Acute Concentration (AC) used for estimating acute risks (i.e., risks associated from a single day or 24-hr of exposure); Average Daily Concentrations (ADC) used for estimating chronic, non-cancer risks; and Lifetime Average Daily Concentration (LADC) used for estimating chronic cancer risks. AC, ADC, and LADC are calculated using the approach and equations described in Appendix B and C of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). Table 2-14 presents the confidence rating of monitoring data that EPA used to assess occupational exposures. EPA evaluated monitoring data using the evaluation strategies laid out in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA 2018b). All exposure monitoring data used in the assessment have a "high" or "medium" confidence rating.

EPA also presented TWA concentrations based on shorter averaging times (e.g., 15-min, 30-min, 1-hr, and 4-hr) in addition to full-shift (either 8- or 12-hour) TWAs for several conditions of use. Short-term TWAs are only presented where data were available to do so. EPA's primary concern for this assessment were full-shift exposures; therefore, no effort was made to estimate shorter-term exposure values where data were not reasonably available. AC, ADC, and LADC values are only calculated based on the full-shift (8- or 12-hr TWAs) as full-shift data represent the closest approximation to a worker's exposure for a full day (i.e., 24-hr), assuming no exposure once the worker leaves the job site. The full-shift exposure results can then be averaged over 24 hours, working years, or lifetime years to estimate AC, ADC, and LADC, respectively. Short-term data may not be representative of a full day's exposure, thus, underestimating AC, ADC, and LADC results.

For several conditions of use, EPA modeled exposure in occupational settings. The models were used to either supplement existing exposure monitoring data or to provide exposure estimates where measured data are unavailable. The use of modeling to supplement existing exposure monitoring data was primarily used to aid EPA's understanding of the monitoring data's representativeness of actual exposures within the condition of use. For example, where model results and monitoring data are similar, it helps corroborate the representativeness of the data to actual exposures. When determining unreasonable risks for scenarios with both monitoring data and modeling, EPA generally uses monitoring data results over modeling unless the data quality score for the monitoring data is low, or there were limited number of data points for the scenario such that the representativeness of the data is limited. Where measured monitoring data and models were not available, EPA estimated exposures using values from GSs and ESDs. A summary of approaches and EPA's overall confidence in the exposure estimates are provided in Table 2-14.

[11] Using the $\frac{LOD}{\sqrt{2}}$ if the geometric standard deviation of the data is less than 3.0 and $\frac{LOD}{2}$ if the geometric standard deviation is 3.0 or greater.

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Source Reference	Data Type	Data Quality Rating	Condition of Use
(HSIA 2018a)	PBZ Monitoring	High	Manufacturing; Processing as a Reactant
(Dow Chem 1984)	PBZ Monitoring	Medium	Repackaging
(Orris and Daniels 1981)	PBZ Monitoring	High	Incorporation into Formulation, Mixture, or Reaction Product (Aerosol Packing Only)
(Gorman et al. 1984)	PBZ Monitoring	Medium	OTVD
(Ruhe 1982)	PBZ Monitoring	Medium	OTVD
(NIOSH 2002b)	PBZ Monitoring	High	OTVD
(NIOSH 2002d)	PBZ Monitoring	High	OTVD
(NIOSH 2002a)	PBZ Monitoring	High	OTVD; Closed-Loop Vapor Degreasing
(NIOSH 2002c)	PBZ Monitoring	High	Closed-Loop Vapor Degreasing; Cold Cleaning
(Vulcan 1994)	PBZ Monitoring	High	Cold Cleaning
(U.S. DOD and Environmental Health Readiness System - Industrial 2018)	PBZ Monitoring	High	Aerosol Degreasing and Aerosol Lubricants; Dry Cleaning and Spot Cleaning; Adhesives, Sealants, Paints, and Coatings (Paints and Coatings Only); Chemical Maskant; Other DoD Uses
(Cosgrove and Hygiene 1994)	PBZ Monitoring	High	Aerosol Degreasing and Aerosol Lubricants
(Vulcan 1992)	PBZ Monitoring	High	Aerosol Degreasing and Aerosol Lubricants
(Vulcan 1993)	PBZ Monitoring	High	Aerosol Degreasing and Aerosol Lubricants
(OSHA 2017)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(NIOSH 1995)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning

Source Reference	Data Type	Data Quality Rating	Condition of Use
(Burroughs 1999a)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(Burroughs 1999b)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(Burroughs 1999b)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(Burroughs 2000)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(NIOSH 2000)	PBZ Monitoring	High	Dry Cleaning and Spot Cleaning
(Gromiec et al. 2002)	PBZ Monitoring	Medium	Adhesives, Sealants, Paints, and Coatings (Adhesives Only)
(Chrostek and Levine 1981)	PBZ Monitoring	High	Adhesives, Sealants, Paints, and Coatings (Paints and Coatings Only)
(Stephenson and Albrecht 1986)	PBZ Monitoring	High	Adhesives, Sealants, Paints, and Coatings (Paints and Coatings Only)
(Hanley 1993)	PBZ Monitoring	Medium	Adhesives, Sealants, Paints, and Coatings (Paints and Coatings Only)
(Ford Motor 1981)	PBZ Monitoring	Medium	Adhesives, Sealants, Paints, and Coatings (Paints and Coatings Only)
(Hervin et al. 1977)	PBZ Monitoring	High	Chemical Maskant
(Dow Chem 1983b)	PBZ Monitoring	Medium	Industrial Processing Aid
(Dow Chem 1983a)	PBZ Monitoring	Medium	Industrial Processing Aid
(Dow Chem 1982)	PBZ Monitoring	Medium	Industrial Processing Aid
(Dow Chem 1979)	PBZ Monitoring	Medium	Industrial Processing Aid
(Gunter and Lybarger 1979)	PBZ Monitoring	High	Wipe Cleaning and Metal/Stone Polishes
(Moody et al. 1983)	PBZ Monitoring	High	Wipe Cleaning and Metal/Stone Polishes
(Burton and Monestersky 1996)	PBZ Monitoring	High	Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

Source Reference	Data Type	Data Quality Rating	Condition of Use
(Gold et al. 2008)	Area Monitoring	High	Other Commercial Uses (Mold Release Only)
(NIOSH 1980)	PBZ Monitoring	Medium	Other Commercial Uses (Printing Only)
(Apol 1981)	PBZ Monitoring	High	Other Commercial Uses (Printing Only)
(Love 1982)	PBZ Monitoring	High	Other Commercial Uses (Printing Only)
(Ruhe 1983)	PBZ Monitoring	High	Other Commercial Uses (Printing Only)
(Gunter et al. 1984)	PBZ Monitoring	High	Other Commercial Uses (Printing Only)
(Burotn 1994)	PBZ Monitoring	Medium	Other Commercial Uses (Printing Only)
(Moseley 1980)	PBZ Monitoring	Medium	Other Commercial Uses (Photographic Film Only)
(Stefaniak et al. 2000)	PBZ Monitoring	High	Other Commercial Uses (Photocopying Only)

Table 2-15. A Summary of Approaches and Overall Confidence for Exposures Estimates for Each OES

Note: Where EPA was not able to estimate ONU inhalation exposure from monitoring data or models, this was assumed equivalent to the central tendency experienced by workers for the corresponding OES; dermal exposure for ONUs was not evaluated because they are not expected to be in direct contact with PCE and data to model incidental exposures were not available.

Occupational Exposure Scenario (OES)	Inhalation Exposure									Dermal Exposure Modeling[b]	
	Monitoring					Modeling		Overall Confidence			
	Monitoring Data	# Data Points[a]	Data Quality Rating	Worker	ONU	Worker	ONU	Worker	ONU	Worker	ONU
Manufacturing	✓	152[c]	H	✓	×	×	×	H	L	✓	-
Repackaging	✓	10	M	✓	×	×	×	M	L	✓	-
Processing as a Reactant	✓	152[d]	H	✓	×	×	×	H	L	✓	-

Occupational Exposure Scenario (OES)	Inhalation Exposure									Dermal Exposure Modeling[b]	
	Monitoring					Modeling		Overall Confidence			
	Monitoring Data	# Data Points[a]	Data Quality Rating	Worker	ONU	Worker	ONU	Worker	ONU	Worker	ONU
Incorporation into Formulation, Mixture, or Reaction Product (Aerosol Packing Only)	✓	5	H	✓	×	×	×	H	L	✓	-
Incorporation into Formulation, Mixture, or Reaction Product (Non-Aerosol Packing Only)	×	-	-	×	×	✓	×	M	L	✓	-
Batch Open-Top Vapor Degreasing	✓	75	M to H	✓	✓	×	×	M to H	M to H	✓	-
Batch Closed-Loop Vapor Degreasing	✓	15	H	✓	✓	×	×	H	H	✓	-
Conveyorized Vapor Degreasing	×	-	-	×	×	✓	✓	M	M	✓	-
Web Degreasing	×	-	-	×	×	✓	✓	M	M	✓	-
Cold Cleaning	✓	29	H	✓	×	✓	✓	M to H	M to H	✓	-
Aerosol Degreasing and Aerosol Lubricants	✓	130	H	✓	×	✓	✓	H	H	✓	-
Dry Cleaning and Spot Cleaning	✓	140[e]	H	✓	✓	✓	✓	H	H	✓	-
Adhesives, Sealants, Paints, and Coatings	✓	28[f]	M; M to H[g]	✓	×	×	×	M	L	✓	-
Maskant For Chemical Milling	✓	24	H	✓	×	×	×	M to H	L	✓	-
Industrial Processing Aid	✓	89	M	✓	×	×	×	M	L	✓	-

Occupational Exposure Scenario (OES)	Inhalation Exposure									Dermal Exposure Modeling[b]	
	Monitoring					Modeling		Overall Confidence			
	Monitoring Data	# Data Points[a]	Data Quality Rating	Worker	ONU	Worker	ONU	Worker	ONU	Worker	ONU
Metalworking Fluids[h]	✗	-	-	✗	✗	✗	✗	M	L	✓	-
Wipe Cleaning and Metal/Stone Polishes	✓	10	H	✓	✓	✗	✗	M to H	M to H	✓	-
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	✓	3	H	✓	✓	✗	✗	M	M	✓	-
Other Industrial Uses	✗	-	-	✗	✗	✓	✗	M	L	✓	-
Other Commercial Uses	✓	92[i]	M to H; H; M; H[j]	✓	✗	✗	✗	M to H; M	L	✓	-
Laboratory Chemicals	EPA did not identify data to assess this OES.									✓	-
Waste Handling, Disposal, Treatment, and Recycling	✗	-	-	✗	✗	✓	✗	M	L	✓	-
Other Department of Defense Uses	✓	2[k]	H	✓	✗	✗	✗	H	L	✓	-

[a] This number only includes full-shift (8-hr and 12-hr TWAs) and does not include short-term samples (i.e., 15-min, 30-min, 60-min, or 4-hr TWAs).
[b] EPA has a medium level of confidence in its dermal exposure estimates which are based on high-end/central tendency parameters and commercial/industrial settings.
[c] This count includes 75 8-hr TWA data points and 77 12-hr TWA data points.
[d] The data for this OES are the same monitoring data from PCE manufacturing sites used as surrogate for sites processing PCE as a reactant.
[e] This count includes 22 data points for the post-2006 NESHAP mix of machine generations and 118 data points for fourth and fifth generation machines only. See Section 2.4.1.16 for further discussion of the two data sets.
[f] This count includes 13 data points for adhesives/sealants and 15 data points for paints/coatings.
[g] For adhesives/sealants the data quality is M; for paints/coatings the data quality is M to H.
[h] Exposure to metalworking fluids were assessed using estimates from an ESD.
[i] This includes 23 data points for printing applications, 3 data points for photocopying, 62 data points for photographic film applications, and 4 for mold release products.
[j] For printing applications the data quality is M to H; for photocopying the data quality is H; for photographic film applications the data quality is M; for mold release products the data quality is H.
[k] This count includes one data point for oil analysis uses at DoD sites and one data point for water pipe repair uses at DoD sites.

2.4.1.4 Consideration of Engineering Controls and Personal Protective Equipment

OSHA and NIOSH recommend 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 (e.g., source enclosure, local exhaust ventilation systems), followed by administrative controls (e.g. do not open machine doors when running), or changes in work practices (e.g., maintenance plan to check equipment to insure no leaks) to reduce exposure potential. Administrative controls are policies and procedures instituted and overseen by the employer to limit 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.

OSHA's Respiratory Protection Standard (29 CFR § 1910.134) requires employers to address workplace hazards by implementing engineering control measures and, if these are not feasible, provide respirators that are applicable and suitable for the purpose intended. Respirator selection provisions are provided in § 1910.134(d) and require that appropriate respirators are selected based on the respiratory hazard(s) to which the worker will be exposed and workplace and user factors that affect respirator performance and reliability. Assigned protection factors (APFs) are provided in Table 1 under § 1910.134(d)(3)(i)(A) (see below in Table 2-16) and refer to the 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.

If respirators are necessary in atmospheres that are not immediately dangerous to life or health, workers must use NIOSH-certified air-purifying respirators or NIOSH-approved supplied-air respirators with the appropriate APF. Respirators that meet these criteria may include air-purifying respirators with organic vapor cartridges. Respirators must meet or exceed the required level of protection listed in Table 2-16. Based on the APF, inhalation exposures may be reduced by a factor of 5 to 10,000, if respirators are properly worn and fitted.

Table 2-16. Assigned Protection Factors for Respirators in OSHA Standard 29 CFR 1910.134

Type of Respirator	Quarter Mask	Half Mask	Full Facepiece	Helmet/ Hood	Loose-fitting Facepiece
1. Air-Purifying Respirator	5	10	50		
2. Power Air-Purifying Respirator (PAPR)		50	1,000	25/1,000	25
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 (e.g., open/closed circuit)			10,000	10,000	

Source: 29 CFR § 1910.134(d)(3)(i)(A)

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 2001b). Results of the survey include the number and percent of establishments and employees using respirators within 12 months prior to the survey. For additional information, please also refer to [Memorandum_NIOSH_BLS Respirator Usage in Private Sector Firms, Docket: TBD].

The plausibility of regular respirator use by workers was considered on an OES-specific basis. See Table 4-3 for determinations of whether respirator use was assumed for each OES during risk characterization.

2.4.1.5 Dermal Exposure Assessment Approach

Dermal exposure data was not readily available for the conditions of use in the assessment. Because PCE is a volatile liquid that readily evaporates from the skin, EPA estimated dermal exposures using the *Dermal Exposure to Volatile Liquids Model*. This model determines a dermal potential dose rate based on an assumed amount of liquid on skin during one contact event per day and the steady-state fractional absorption for PCE based on a theoretical framework provided by Kasting (2006). The amount of liquid on the skin is adjusted by the weight fraction of PCE in the liquid to which the worker is exposed. Specific details of the dermal exposure assessment can be found in Section 2.4.1.29 and equations and sample calculations for estimate dermal exposures can be found in Appendix K of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

2.4.1.6 Manufacturing

Worker Activities

During manufacturing, workers are potentially exposed while connecting and disconnecting hoses and transfer lines to containers and packaging to be loaded with PCE product (e.g., railcars, tank trucks,

totes, drums, bottles) and intermediate storage vessels (e.g., storage tanks, pressure vessels). Workers near loading racks and container filling stations are potentially exposed to fugitive emissions from equipment leaks and displaced vapor as containers are filled. These activities are potential sources of worker exposure through dermal contact with liquid and inhalation of vapors.

ONUs include employees that work at the site where PCE is manufactured, but they do not directly handle the chemical and therefore are assumed to have lower inhalation exposures, and are not assumed to have dermal exposures. ONUs for manufacturing include supervisors, managers, and tradesmen that may be in the manufacturing area but do not perform tasks that result in the same level of exposures as manufacturing workers.

Number of Workers and Occupational Non-Users

To determine the number of workers, EPA used the average of the ranges reported in the 2016 CDR for four sites where data were available and worker and ONUs estimates from the BLS analysis for the other four sites (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). For the BLS analysis EPA used the NAICS code 325199—All Other Basic Organic Chemical Manufacturing to estimate workers and ONUs. CDR data do not differentiate between workers and ONUs; therefore, EPA assumed the ratio of workers to ONUs would be similar as determined in the BLS data where approximately 68% of the exposed personnel are workers and 32% are ONUs (U.S. BLS 2016). This resulted in approximately 640 workers and 300 ONUs (see Table 2-17).

Table 2-17. Estimated Number of Workers Potentially Exposed to PCE During Manufacturing

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
8	80	38	640	300	940

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

Table 2-18 summarizes 15-min, 30-min, 8-hr, and 12-hr TWA exposure results for manufacturing. The high-ends are the 95th percentile of the respective data sets and the central tendencies are the 50th percentile. EPA assessed exposures using data submitted for three companies by the Halogenated Solvent Industry Alliance (HSIA) (HSIA 2018a). It should be noted that approximately 65% of the 8-hr TWA exposure data, 73% of the 12-hr TWA exposure data, 24% of the 15-min TWA exposure data, and 55% of the 30-min TWA exposure data were below the limit of detection (LOD). To estimate exposure concentrations for these data, EPA followed the *Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b) as discussed in Section 2.4.1.3. The geometric standard deviation for the 8-hr TWA data, 12-hr TWA data, and 15-min TWA were all above 3.0; therefore, EPA used the $\frac{LOD}{2}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b). The geometric standard deviation for the 30-min TWA was below 3.0; therefore, EPA used the $\frac{LOD}{\sqrt{2}}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b). Because over 50% of the data are below the LOD for the 8-hr, 12-hr, and 30-min TWA data, calculating statistics from this data does present the potential

to introduce biases into the results. Estimation of exposure values for results below the LOD may over- or under-estimate actual exposure thus skewing the calculated statistics higher or lower, respectively. The overall directional bias of the exposure assessment, accounting for both the overestimate and underestimate, is not known.

It should also be noted that 18 8-hr TWA exposure data points and 5 30-min TWA data points from Company C were not included in the results as they were reported as being below the detection limit, but the company did not provide the value of the LOD. Therefore, EPA could not estimate a value for these data using the guidelines described above. Data were not available to estimate ONU exposures; EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-18. Summary of Inhalation Monitoring Data for the Manufacture of PCE

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	3.3E-02	2.6	75[b]	3.3E-02	High
Acute Exposure Concentration (AC) based on 8-hr TWA	1.1E-02	0.9		1.1E-02	
Average Daily Concentration (ADC) based on 8-hr TWA	7.4E-03	0.6		7.4E-03	
Lifetime Average Daily Concentration (LADC) based on 8-hr TWA	2.9E-03	0.3		2.9E-03	
12-hr TWA Exposure Concentration	2.1E-02	0.2	77	2.1E-02	
Acute Exposure Concentration (AC) based on 12-hr TWA	1.0E-02	0.1		1.0E-02	
Average Daily Concentration (ADC) based on 12-hr TWA	7.0E-03	7.3E-02		7.0E-03	
Lifetime Average Daily Concentration (LADC) based on 12-hr TWA	2.8E-03	3.7E-02		2.8E-03	
15-min TWA Exposure Concentration	2.0	15	161	2.0	
30-min TWA Exposure Concentration	0.7	12	38[c]	0.7	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

[b] Data does not include 18 data points that were reported as being below the detection limit, but for which the company did not provide the LOD for use in estimating an exposure value.

[c] Data does not include five data points that were reported as being below the detection limit, but for which the company did not provide the LOD for use in estimating an exposure value.

Sources: (HSIA 2018a)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data collected at workplaces directly applicable to this condition of use, and the data were determined to have a "high" confidence rating through EPA's systematic review process. Specifically, the data were determined to be highly representative in geographic scope and reflective of current operations. The source also provides metadata including sample type and sample duration.

The data includes exposure concentrations for a variety of worker tasks at each of the three manufacturing facilities from which the data were obtained. It is not known whether these data points would also be representative of the worker exposure level at other domestic manufacturing facilities. Despite this uncertainty, EPA has a high level of confidence in the assessed worker exposures based on the strength of the monitoring data.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.7 Repackaging

Worker Activities

During repackaging, workers are potentially exposed while connecting and disconnecting hoses and transfer lines to containers and packaging to be unloaded (e.g., railcars, tank trucks, totes), intermediate storage vessels (e.g., storage tanks, pressure vessels), and final packaging containers (e.g., drums, bottles). Workers near loading racks and container filling stations are potentially exposed to fugitive emissions from equipment leaks and displaced vapor as containers are filled. These activities are potential sources of worker exposure through dermal contact with liquid and inhalation of vapors.

ONUs include employees that work at the site where PCE is repackaged, but they do not directly handle the chemical and are therefore expected to have lower inhalation exposures and are not expected to have dermal exposures. 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.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during repackaging of PCE using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). This resulted in approximately 210 workers and 75 ONUs potentially exposed during repackaging of PCE (see Table 2-19).

Table 2-19. Estimated Number of Workers Potentially Exposed to PCE During Repackaging

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
51	4	1	210	75	280

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA assessed inhalation exposures during import/repackaging using identified monitoring data. Table 2-20 summarizes 15-min, 30-min, and 8-hr TWA results obtained from data submitted to EPA by Dow Chemical under TSCA (Dow Chem 1984). For the 8-hr TWA results the 95th percentile and 50th

percentiles are presented as the high-end and central tendency exposure values, respectively. For the 15-min TWA, only two data points were available; therefore, EPA presents two scenarios: 1) using the maximum as a "higher value"; and 2) using the midpoint as a "midpoint value". For the 30-min TWA, only five data points were available; therefore, the maximum is presented as the high-end and the median is presented as the central tendency. It should be noted that two of the 30-min TWA samples measured below the LOD (Dow Chem 1984). To estimate exposure concentrations for these data, EPA followed the *Guidelines for Statistical Analysis of Occupational Exposure Data* (1994) as discussed in Section 2.4.1.3. The geometric standard deviation for was above 3.0; therefore, EPA used the $\frac{LOD}{2}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b). Data were not available to estimate ONU exposures; EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-20. Summary of Inhalation Monitoring Data for Repackaging

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	0.4	0.8	10	0.4	Medium
Acute Exposure Concentration (AC)	0.1	0.3		0.1	
Average Daily Concentration (ADC)	9.9E-02	0.2		9.9E-02	
Lifetime Average Daily Concentration (LADC)	3.9E-02	9.6E-02		3.9E-02	
15-min TWA Exposure Concentration[b]	0.9	1.6	2	0.9	
30-min TWA Exposure Concentration	8.0E-02	5.7	5	8.0E-02	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] Due to only two data points identified, EPA presents two scenarios: 1) using the higher of the two values; and 2) using the midpoint of the two values.
Sources: (Dow Chem 1984)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data collected at one repackaging facility. The data were determined to have a "medium" confidence rating through EPA's systematic review process. However, the data may not be representative of exposures across other repackaging facilities (e.g., those repackaging from and into different container sizes than the used in the identified data). Based on reasonably information above, EPA has a medium level of confidence in the assessed worker exposure.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.8 Processing as a Reactant

Worker Activities

At industrial facilities, workers are potentially exposed when unloading PCE from transport containers into intermediate storage tanks and process vessels. Workers may be exposed via inhalation of vapor or via dermal contact with liquids while connecting and disconnecting hoses and transfer lines. Once PCE is unloaded into process vessels, it is consumed as a chemical intermediate.

ONUs are employees who work at the facilities that process and use PCE, but who do not directly handle the material. ONUs may also be exposed to PCE 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.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during processing of PCE as a reactant using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015)as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). This resulted in approximately 4,200 workers and 1,900 ONUs potentially exposed during processing of PCE as a reactant (see Table 2-21).

Table 2-21. Estimated Number of Workers Potentially Exposed to PCE During Processing as a Reactant

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed ONUs[a]	Total Exposed[a]
117	36	17	4,200	1,900	6,100

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation monitoring data to assess exposures during processing PCE as a reactant. EPA assumes that potential sources of exposure at sites using PCE as a reactant are similar to sites manufacturing raw PCE. Therefore, EPA assessed inhalation exposures during processing PCE as a reactant using monitoring data from manufacturing sites as a surrogate for sites processing PCE as a reactant. The results from the surrogate inhalation monitoring data are provided in Table 2-22.

Table 2-22. Summary of Inhalation Monitoring Results for Processing PCE as a Reactant[a]

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[b]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	3.3E-02	2.6	75[c]	3.3E-02	High
Acute Exposure Concentration (AC) based on 8-hr TWA	1.1E-02	0.9		1.1E-02	
Average Daily Concentration (ADC) based on 8-hr TWA	7.4E-03	0.6		7.4E-03	
Lifetime Average Daily Concentration (LADC) based on 8-hr TWA	2.9E-03	0.3		2.9E-03	
12-hr TWA Exposure Concentration	2.1E-02	0.2	77	2.1E-02	
Acute Exposure Concentration (AC) based on 12-hr TWA	1.0E-02	0.1		1.0E-02	
Average Daily Concentration (ADC) based on 12-hr TWA	7.0E-03	7.3E-02		7.0E-03	
Lifetime Average Daily Concentration (LADC) based on 12-hr TWA	2.8E-03	3.7E-02		2.8E-03	
15-min TWA Exposure Concentration	2.0	15	161	2.0	
30-min TWA Exposure Concentration	0.7	12	38[d]	0.7	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] These results are based on monitoring data from PCE manufacturing used as surrogate for sites processing PCE as a reactant.

[b] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

[c] Data does not include 18 data points that were reported as being below the detection limit, but for which the company did not provide the LOD for use in estimating an exposure value.

[d] Data does not include five data points that were reported as being below the detection limit, but for which the company did not provide the LOD for use in estimating an exposure value.

Sources: (HSIA 2018a)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data collected at facilities manufacturing PCE as a surrogate for facilities processing PCE as reactant. The data were determined to have a "high" confidence rating through EPA's systematic review process. Although these data are not directly applicable to processing of PCE as a reactant, EPA expects a high degree of overlap of worker tasks at both manufacturing sites and sites processing PCE as a reactant. Based on this expectation and the strength of the monitoring data, EPA has a medium to high level of confidence in the assessed worker exposures.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.9 Incorporation into Formulation, Mixture, or Reactant Product

Worker Activities

At formulation facilities, workers are potentially exposed when unloading PCE into mixing vessels, taking QC samples, and packaging formulated products into containers and tank trucks. The exact activities and associated level of exposure will differ depending on the degree of automation, presence of engineering controls, and use of PPE at each facility.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during formulation of PCE-containing products using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d)for number of sites estimate). This resulted in approximately 800 workers and 310 ONUs potentially exposed during formulation of PCE-containing products (see Table 2-23).

Table 2-23. Estimated Number of Workers Potentially Exposed to PCE During Formulation

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
39	21	8	800	310	1,100

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data related to the aerosol packing of PCE-containing products (Orris and Daniels 1981). However, no monitoring data was identified for other formulation sites and it is unlikely aerosol packing is representative of other formulation sites where workers are exposed during unloading of bulk containers (i.e., tank trucks and rail cars) and loading of formulated products into smaller containers (e.g., drums). Therefore, EPA used the monitoring data to assess exposures at aerosol packing facilities and the *EPA/OAQPS AP-42 Loading Model*, *EPA/OPPT Mass Balance Model* and Monte Carlo analysis to assess exposures at other non-aerosol packing facilities. Details of the model design and parameters is provided in Appendix F of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

Table 2-24 summarizes 8-hr TWA PBZ monitoring data for aerosol packing formulation sites. Due to the limited number of data points (five), EPA used the maximum value as the high-end and the 50th percentile as the central tendency. Data were not available to estimate short-term or ONU exposures; EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-24. Summary of Inhalation Exposure Monitoring Data for Aerosol Packing Formulation Sites

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	8.3	13	5	8.3	High
Acute Exposure Concentration (AC)	2.8	4.4		2.8	
Average Daily Concentration (ADC)	1.9	3.0		1.9	
Lifetime Average Daily Concentration (LADC)	0.8	1.5		0.8	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
Sources: (Orris and Daniels 1981)

The modeling approach used to assess exposures at non-aerosol packing formulation sites estimates exposures to workers loading formulated PCE-based products into 55-gallon drums. Inhalation exposure to chemical vapor during loading is a function of physical properties of PCE, various EPA default constants, and other model parameters. While physical properties are fixed for a substance, some model parameters, such as weight fraction of PCE in the product, ventilation rate, mixing factor, and vapor saturation factor, are expected to vary from one facility to another. This approach addresses variability for these parameters using a Monte Carlo analysis.

The modeling approach requires an input on the number of containers loaded per day which is determined based on the throughput of PCE at each site and the weight fraction of PCE in the product. To determine these values EPA divided each site identified in Section 2.2.1.2.2 into one of the following categories: 1) sites formulating degreasing solvents; 2) sites formulating dry cleaning solvents, and 3) sites formulating "miscellaneous" PCE-containing products, including coatings, adhesives, metalworking fluids, and other niche use PCE-based products. The three categories were selected based on available market data from HSIA (2008), where the first two categories (degreasing and dry cleaning formulation) had market information indicating the percentage of the production volume used in those types of products. The HSIA (2008) market data did not include detailed production volume data for the third group so EPA could not divide the PCE production volume amongst the product types to calculate per site throughputs. Therefore, EPA assessed as a single category.

Table 2-25 summarizes model results for workers at non-aerosol packing formulation sites with the 50th percentile presented as the central tendency and the 95th percentile presented as the high-end. Data were not available to incorporate ONU exposures into the model. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-25. Summary of Exposure Modeling Results for Formulation of PCE-Based Products

Formulation Type	Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
		Central Tendency (ppm)	High-End (ppm)		
Degreasing Solvent	8-hr TWA Exposure Concentration	0.7	2.6	0.7	N/A – modeled data
	Acute Exposure Concentration (AC)	0.1	0.4	0.1	
	Average Daily Concentration (ADC)	1.6E-02	5.7E-02	1.6E-02	
	Lifetime Average Daily Concentration (LADC)	2.3E-03	8.4E-03	2.3E-03	
Dry Cleaning Solvent	8-hr TWA Exposure Concentration	4.0	14	4.0	
	Acute Exposure Concentration (AC)	0.6	2.1	0.6	
	Average Daily Concentration (ADC)	8.6E-02	0.3	8.6E-02	
	Lifetime Average Daily Concentration (LADC)	1.3E-02	4.5E-02	1.3E-02	
Miscellaneous	8-hr TWA Exposure Concentration	0.4	1.4	0.4	
	Acute Exposure Concentration (AC)	5.9E-02	0.2	5.9E-02	
	Average Daily Concentration (ADC)	8.6E-03	3.1E-02	8.6E-03	
	Lifetime Average Daily Concentration (LADC)	1.3E-03	4.5E-03	1.3E-03	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers at aerosol packing formulation sites is assessed using PCE personal breathing zone monitoring data collected at workplaces directly applicable to this condition of use, and the data were

determined to have a "high" confidence rating through EPA's systematic review process. Specifically, the data were determined to be highly reliable, representative in geographic scope and reflective of current operations. The source also provides metadata including sample type and sample duration. The data includes exposure at a single aerosol packing facility. It is not known whether these data points would also be representative of the worker exposure level at other similar facilities. Despite this uncertainty, EPA has a high level of confidence in the assessed worker exposures based on the strength of the monitoring data.

The *EPA/OAQPS AP-42 Loading Model* and *EPA/OPPT Mass Balance Model* are used to estimate worker exposures for non-aerosol packing facilities. The model uses a Monte Carlo analysis to incorporate variability in the model input parameters. EPA believes the model exposures are likely to be representative of worker exposure associated with loading 55-gallon drums. However, it assumes all products are loaded into drums and does not consider the potential for loading of products into smaller containers instead of or in addition to drums.

The model also does not consider worker exposure from unloading raw PCE from bulk containers (i.e. tank trucks or railcars). Although EPA can estimate exposures during this unloading activity using the *Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model*, it is unclear if the same workers will perform both unloading and loading activities in the same day. Therefore, it may not be accurate to combine estimates from each model to estimate a total exposure. In the case where a worker is both unloading bulk containers and loading products into drums on the same day, the overall error from not including exposures during unloading in the results is expected to be small as the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model estimates an 8-hr TWA exposure of 0.01 ppm for tank truck unloading and an 8-hr TWA of 0.04 ppm for railcar unloading whereas the model for drum loading estimates 8-hr TWAs ranging from 0.60 to 14.1 ppm.

Furthermore, loading activities may be only a small part of the worker's day. The model does not account for other potential sources of exposure at industrial facilities, such as sampling, equipment cleaning, and other process activities that can contribute to a worker's overall 8-hr daily exposure. These model uncertainties could result in an underestimate of the worker 8-hr exposure. Based on reasonably available information above, EPA has a medium level of confidence in the assessed worker exposure.

Exposure to ONUs at both aerosol packing and non-aerosol packing facilities is assessed using the worker central tendency exposure values from the respective facility types. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.10 Batch Open-Top Vapor Degreasing

Worker Activities

When operating OTVD, workers manually load or unload fabricated parts directly into or out of the vapor cleaning zone. Worker exposure can occur from solvent dragout or vapor displacement when the substrates enter or exit the equipment, respectively (Kanegsberg and Kanegsberg 2011). The amount of time a worker spends at the vapor degreaser can vary depending on the number of workloads needed to be cleaned. Reports from NIOSH at three sites using OTVDs found degreaser operators may spend 0.5 to 2 hours per day at the degreaser (NIOSH 2002a, b, d).

Worker exposure is also possible while charging new solvent or disposing spent solvent. The frequency of solvent charging can vary greatly from site-to-site and is dependent on the type, size, and amount of parts cleaned in the degreaser. NIOSH investigations found that one site added a 55-gallon drum of new solvent to the degreaser unit everyone to two weeks; another site added one 55-gallon drum per month; and another site added two 55-gallon drums per month to its large degreaser and three 55 gallon drums per year to its small degreaser (NIOSH 2002a, b, d).

EPA defined ONU as an employee who does not regularly handle PCE or operate the degreaser but performs work in the area around the degreaser.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE in OTVDs using the Draft ESD on the Use of Vapor Degreasers (OECD 2017a). The ESD estimates seven workers and four ONUs per site (OECD 2017a). EPA multiplied these values by the number of sites estimated in the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). This resulted in approximately 2,800 workers and 1,600 ONUs using the number of sites estimated from the 95th percentile use-rate and 35,000 workers and 20,000 ONUs using the number of sites estimated from the 50th percentile use-rate. Table 2-26 summarizes these results. Note: These are bounding estimates and may overestimate actual number of workers.

Table 2-26. Estimated Number of Workers Potentially Exposed to PCE During Use in Open-Top Vapor Degreasing

Use-Rate Scenario	Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
95th Percentile	398	7	4	2,800	1,600	4,400
50th Percentile	4,942	7	4	35,000	20,000	54,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

Table 2-27 summarizes the 8-hr TWA monitoring data, 4-hr TWA monitoring data, and 15-minute TWA monitoring data for the use of PCE in OTVDs. The high-end and central tendency values for the 8-hr TWA data represent the 95th and 50th percentile, respectively. Due to the limited number of data points (three samples), the 4-hr TWA high-end is the maximum value and the central tendency is the 50th percentile. There is only a single 15-min TWA sample.

EPA recognizes that worker job titles and activities may vary significantly from site to site; therefore, EPA typically identified samples as worker samples unless it was explicitly clear from the job title (e.g., inspectors) and the description of activities in the report that the employee was not operating the degreaser during the sampling period. Samples from employees determined not to be operating the degreasing equipment were designated as ONU samples.

EPA identified inhalation exposure monitoring data from NIOSH investigations at five sites using PCE as a degreasing solvent in OTVDs. Due to the large variety in shop types that may use PCE as a vapor degreasing solvent, there is some uncertainty in how representative these data are of a "typical" shop.

Table 2-27. Summary of Worker Inhalation Exposure Monitoring Data for Open-Top Vapor Degreasing

Exposure Concentration Type	Worker Exposures		Number of Worker Samples	Occupational Non-User Exposures		Number of ONU Samples	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	2.1	32	63	0.6	5.2	12	Medium to High
Acute Exposure Concentration (AC)	0.7	11		0.2	1.7		
Average Daily Concentration (ADC)	0.5	7.3		0.1	1.2		
Lifetime Average Daily Concentration (LADC)	0.2	3.8		5.5E-02	0.6		
15-min TWA Exposure Concentration	17		1	No 4-hr or 15-minute data identified for ONUs			
4-hr TWA Exposure Concentration	1.3	1.6	3				

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
Source: (NIOSH 2002a, b, d; Gorman et al. 1984; Ruhe 1982)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from several different sources, with confidence rating of the data ranging from "medium" to "high", as determined through EPA's systematic review process. Due to the large variation amongst sites that operate OTVDs, there is some uncertainty in how representative the monitoring data of typical shops. Despite this uncertainty, EPA has a medium to high level of confidence in the assessed exposure for this condition of use, based on the strength of the monitoring data.

2.4.1.11 Batch Closed-Loop Vapor Degreasing

Worker Activities

For closed-loop vapor degreasing, worker activities can include placing or removing parts from the basket, as well as general equipment maintenance. Workers can be exposed to residual vapor as the door to the degreaser chamber opens after the cleaning cycle is completed. The amount of time workers spend in the degreaser area can vary greatly by site. One NIOSH report (NIOSH 2002c) reported workers spent 1.5 to 2 hours per shift at the degreaser and another NIOSH report (NIOSH 2002a) indicating that workers spent over 90% of their day in the degreaser area. Similarly, addition of fresh solvent to the degreasing machine can vary significantly with one site indicating 50 gallons of PCE per month were added and another site indicating 10 to 20 gallons of PCE per year were added to the machine (NIOSH 2002a, c).

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE in closed-loop degreasing using the same methodology as described for OTVDs. This resulted in approximately 97,000 workers and 56,000 ONUs using the number of sites estimated from the 95th percentile use-rate and 180,000 workers and 100,000 ONUs using the number of sites estimated from the 50th percentile use-rate (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). Table 2-28 summarizes these results. Note: These are bounding estimates and may overestimate actual number of workers.

Table 2-28. Estimated Number of Workers Potentially Exposed to PCE During Use in Closed-Loop Vapor Degreasing

Use-Rate Scenario	Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
95th Percentile	13,912	7	4	97,000	56,000	150,000
50th Percentile	25,546	7	4	180,000	100,000	280,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE as a degreasing solvent in batch closed-loop vapor degreasers. Due to the large variety in shop types that may use PCE as a vapor degreasing solvent, it is unclear how representative these data are of a "typical" shop. EPA does not have a model for estimating exposures from closed-loop degreasers; therefore, the assessment is based on the identified monitoring data.

Worker samples were determined to be any sample taken on a person while performing the degreasing tasks. ONUs samples were determined to be any sample taken on a person in the same location as the degreaser but not performing the degreasing themselves.

Table 2-29 summarizes the 8-hr TWA and 4-hr TWA monitoring data for the use of PCE in closed-loop vapor degreasers. For workers, the 8-hr TWA high-end and central tendency are based on the 95th and 50th percentiles, respectively. Due to the limited data points for worker 4-hr TWAs, EPA used the maximum and median as the high-end and central tendency, respectively. For ONUs, only two data points were available; therefore, EPA presents two scenarios: 1) using the maximum as a "higher value," and 2) using the midpoint as a "midpoint value."

When comparing to monitoring data from OTVDs, the data show a decrease in worker exposure of 99.2% at the 95th percentile and 96.6% at the 50th percentile and a decrease in ONU exposure of 98.2% at the 95th percentile and 89.2% at the 50th percentile. This is generally consistent with data in literature which found that solvent purchases for closed-loop systems were reduced by 83% to over 98% as compared to OTVDs and air emissions were reduced from 95% to over 99% as compared to OTVDs (Durkee 2014; Newmoa 2001).

Table 2-29. Summary of Worker Inhalation Exposure Monitoring Data for Closed-Loop Vapor Degreasing

Exposure Concentration Type	Worker Exposures		Number of Worker Samples	Occupational Non-User Exposures[a]		Number of ONU Samples	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	7.2E-02	0.3	13	6.5E-02	9.6E-02	2	High
Acute Exposure Concentration (AC)	2.4E-02	8.4E-02		2.2E-02	3.2E-02		
Average Daily Concentration (ADC)	1.6E-02	5.8E-02		1.5E-02	2.2E-02		
Lifetime Average Daily Concentration (LADC)	6.6E-03	3.0E-02		5.9E-03	1.1E-02		
4-hr TWA Exposure Concentration	2.0E-02	8.6E-02	3	No 4-hr data identified for ONUs			

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] Due to only two data points identified, EPA presents two scenarios: 1) using the higher of the two values; and 2) using the midpoint of the two values.
Source: (NIOSH 2002a, c)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from two sources with the data determined to have a "high" confidence rating, as determined through EPA's systematic review process. The data show a decrease in exposure concentrations as compared to OTVD monitoring data that agrees with literature expectations. Based on the reasonably available information above, EPA has a high level of confidence in the assessed exposure for this condition of use.

2.4.1.12 Conveyorized Vapor Degreasing

Worker Activities

For conveyorized vapor degreasing, worker activities can include placing or removing parts from the basket, as well as general equipment maintenance. Depending on the level of enclosure and specific conveyor design, workers can be exposed to vapor emitted from the inlet and outlet of the conveyor portal.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE in conveyorized degreasing using the same methodology as described for OTVDs. This resulted in approximately 2,800 workers and 1,600 ONUs using the number of sites estimated from the 95th percentile use-rate and 4,000 workers and 2,300 ONUs using the number of sites estimated from the 50th percentile use-rate (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). Table 2-30 summarizes these results. Note: These are bounding estimates and may overestimate actual number of workers.

Table 2-30. Estimated Number of Workers Potentially Exposed to PCE During Use in Conveyorized Vapor Degreasing

Use-Rate Scenario	Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
95th Percentile	395	7	4	2,800	1,600	4,300
50th Percentile	568	7	4	4,000	2,300	6,200

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation exposure monitoring data related to the use of PCE in conveyorized degreasing. Therefore, EPA assessed inhalation exposures during conveyorized degreasing using the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model. Details of the model design and parameters is provided in Appendix G of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

The key parameter in the model is the emission rate from the degreaser. Emission rates were modeled using the reported unit emissions of PCE from the single conveyorized degreaser in the 2014 NEI (U.S. EPA 2018a). The model estimates exposures for both workers and ONUs. Workers estimates are based on concentrations in the near-field where the conveyorized degreasing work occurs, and ONU exposures are based on concentrations in the far-field away from the conveyorized degreaser. The results from the inhalation model are provided in Table 2-31. The high-end and central tendency are the 95th and 50th percentiles, respectively, calculated by the model.

Table 2-31. Summary of Exposure Modeling Results for Use of PCE in Conveyorized Vapor Degreasing

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures		Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)	Central Tendency (ppm)	High-End (ppm)	
8-hr TWA Exposure Concentration	78	186	41	126	N/A – modeled data
Acute Exposure Concentration (AC)	26	62	14	42	
Average Daily Concentration (ADC)	18	42	9.3	29	
Lifetime Average Daily Concentration (LADC)	6.7	17	3.5	12	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model. The model uses a Monte Carlo analysis, which incorporates variability in the model input parameters. Only a single emission rate data point was available for PCE conveyorized degreasing for use in the model and there is some uncertainty in how representative this data point is of a "typical" conveyorized degreaser. Based on the reasonably available information above, EPA has a medium level of confidence in the assessed exposure for this condition of use.

2.4.1.13 Web Degreasing

Worker Activities

Worker activities for web degreasing are expected to be similar to other degreasing uses and can include placing or removing parts from the degreasing machine, as well as general equipment maintenance. Depending on the level of enclosure and specific design, workers can be exposed to vapor emitted from the inlet and outlet of the conveyor portal.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE in web degreasing using the same methodology as described for OTVDs. This resulted in approximately 2,800 workers and 1,600 ONUs using the number of sites estimated from the 95th percentile use-rate and 4,000 workers and 2,300 ONUs using the number of sites estimated from the 50th percentile use-rate (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). Table 2-32 summarizes these results. Note: These are bounding estimates and may overestimate actual number of workers.

Table 2-32. Estimated Number of Workers Potentially Exposed to PCE During Use in Web Degreasing

Use-Rate Scenario	Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
95th Percentile	395	7	4	2,800	1,600	4,300
50th Percentile	568	7	4	4,000	2,300	6,200

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation exposure monitoring data related to the use of PCE in web degreasing. Therefore, EPA assessed inhalation exposures during web degreasing using the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model. Details of the model design and parameters is provided in Appendix G of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

The key parameter in the model is the emission rate from the degreaser. Emission rates were modeled using the reported unit emissions of PCE from web degreasers in the 2014 NEI (U.S. EPA 2018a). The model estimates exposures for both workers and ONUs. Workers estimates are based on concentrations in the near-field where the web degreasing work occurs, and ONU exposures are based on concentrations in the far-field away from the web degreaser. The results from the inhalation model are provided in Table 2-33. The high-end and central tendency are the 95th and 50th percentiles, respectively, calculated by the model.

Table 2-33. Summary of Exposure Modeling Results for Use of PCE in Web Degreasing

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures		Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)	Central Tendency (ppm)	High-End (ppm)	
8-hr TWA Exposure Concentration	0.6	1.8	0.3	1.2	N/A – modeled data
Acute Exposure Concentration (AC)	0.2	0.6	0.1	0.4	
Average Daily Concentration (ADC)	0.1	0.4	7.3E-02	0.3	
Lifetime Average Daily Concentration (LADC)	5.3E-02	0.2	2.7E-02	0.1	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model. The model uses a Monte Carlo analysis, which incorporates variability in the model input parameters. Due to the limited number of data points, there is some uncertainty on the representativeness of emission rates from the 2014 NEI (U.S. EPA 2018a) of "typical" web degreasers. Based on the reasonably available information above, EPA has a medium level of confidence in the assessed exposure for this condition of use.

2.4.1.14 Cold Cleaning

Worker Activities

The general worker activities for cold cleaning include placing the parts that require cleaning into a vessel. The vessel is usually something that will hold the parts but not the liquid solvent (i.e., a wire basket). The vessel is then lowered into the machine, where the parts could be sprayed, and then completely immersed in the solvent. After a short time, the vessel is removed from the solvent and allowed to drip/air dry. Depending on the industry and/or company, these operations may be performed manually (i.e., by hand) or mechanically. Sometimes parts require more extensive cleaning; in these cases, additional operations are performed including directly spraying solvent on the part, agitation of the solvent or parts, wipe cleaning and brushing (NIOSH 2001a; U.S. EPA 1997).

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE in cold cleaners using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the NAICS code reported by the site in the 2014 NEI (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate)(U.S. EPA 2018a). In the 2014 NEI (U.S. EPA 2018a), four sites reported NAICS code for which there was no Census data available. To estimate the number of workers/ONUs at these sites, EPA referenced the 2017 Emission Scenario Document (ESD) on the Use of Vapor Degreasers (OECD 2017a)[12]. There are approximately 710 workers and 420 ONUs potentially exposed during use of PCE in cold cleaning (see Table 2-34).

It should be noted that this number is expected to underestimate the total number of workers and ONUs exposed to PCE during cold cleaning as NEI data does not include cold cleaner operations that are classified as area sources. Area sources are reported at the county level and do not include site-specific information. Therefore, any sites operating a cold cleaning machine that is classified as an area source would not be included in the count of sites in the 2014 NEI. EPA does not have sufficient information to estimate the number of area sources that may operate cold cleaning machines.

[12] Although the ESD covers vapor degreasers not cold cleaners, the types of industries using cold cleaners are assumed to be similar to those using vapor degreasers. Therefore, the number of workers/ONUs are assumed to be similar.

Table 2-34. Estimated Number of Workers Potentially Exposed to PCE During Use in Cold Cleaning

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
17	42	25	710	420	1,100

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

Table 2-35 summarizes the 8-hr TWA and 4-hr TWA monitoring data for the use of PCE in cold cleaners. For the 8-hr TWA, the 95th percentile and 50th percentile of the identified exposure data are presented as the high-end and central tendency exposure values, respectively. Due to the limited number of data points for the 4-hr TWA, the maximum and 50th percentile (median) of the data are presented as the high-end and central tendency, respectively. The data were obtained from two sources: 1) a NIOSH In-Depth Survey Report (NIOSH 2002c); and 2) a study submitted to EPA by Vulcan Chemicals (1994) under TSCA.

Worker samples were determined to be any sample taken on a person while performing the cold cleaning tasks. ONUs samples were determined to be any sample taken on a person in the same location as the cold cleaning machine but not performing the cold cleaning themselves. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

Table 2-35. Summary of Worker Inhalation Exposure Monitoring Data for Use of PCE in Cold Cleaning

Exposure Concentration Type	Central Tendency (ppm)	High-End (ppm)	Number of Samples	Data Quality Rating of Air Concentration Data
8-hr TWA Exposure Concentration	1.4	4.1	29	High
Acute Exposure Concentration (AC)	0.5	1.4		
Average Daily Concentration (ADC)	0.3	0.9		
Lifetime Average Daily Concentration (LADC)	0.1	0.5		
4-hr TWA Exposure Concentration	2.9	4.3	5	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
Source: (NIOSH 2002c; Vulcan 1994)

Due to the large variety in shop types that may use PCE as a cold cleaning solvent, it is unclear how representative these data are of a "typical" shop. Therefore, EPA supplemented the identified monitoring data using the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model. Details of the model design and parameters is provided in Appendix G of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). The results from the model are provided in

Table 2-36. For model results, the high-end and central tendency are the 95th and 50th percentiles, respectively.

The key parameter in the model is the emission rate from the cold cleaning machine. Emission rates were modeled using a discrete distribution of reported cold cleaning machine unit emissions of PCE in the 2014 NEI (U.S. EPA 2018a). The model estimates exposures for both workers and ONUs. Workers estimates are based on concentrations in the near-field where the cold cleaning work occurs, and ONU exposures are based on concentrations in the far-field away from the cold cleaning machine.

The high-end results of the model are within the same order of magnitude as the high-end and central tendency found in the monitoring data. However, the central tendency estimated by the model is three orders of magnitude lower than the central tendency from the monitoring data. This may be due to the limited number of sites from which the monitoring data were taken whereas the model is meant to capture a broader range of scenarios.

Table 2-36. Summary of Exposure Modeling Results for Use of PCE in Cold Cleaning

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures		Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)	Central Tendency (ppm)	High-End (ppm)	
8-hr TWA Exposure Concentration	2.4E-03	1.5	1.2E-03	0.8	N/A – modeled data
Acute Exposure Concentration (AC)	8.0E-04	0.5	4.1E-04	0.3	
Average Daily Concentration (ADC)	5.5E-04	0.4	2.8E-04	0.2	
Lifetime Average Daily Concentration (LADC)	2.0E-04	0.1	1.1E-04	6.7E-02	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from two sources with the data determined to have a "high" confidence rating, as determined through EPA's systematic review process. The exposure data are supplemented with near-field/far-field exposure modeling using a Monte Carlo analysis, which incorporates variability in the model input parameters. The high-end model results generally agree with monitoring data high-end and central tendency. However, the central tendency model results are three orders of magnitude lower than the monitoring data. This may be due to uncertainty in the representativeness of the monitoring data of "typical" exposures from cold cleaning. Based on the reasonably available information above, EPA has a medium to high level of confidence in the assessed exposure for this condition of use.

2.4.1.15 Aerosol Degreasing and Aerosol Lubricants

Worker Activities

PCE-based aerosol products include degreasers for applications such as brake cleaning, engine degreasing, electric motor cleaners, cable cleaners, coil cleaners, and other metal product cleaning. Additional aerosol products include penetrating lubricants and oils, high pressure non-melt red greases, white lithium greases, silicone lubricants, chain and cable lubricants, vandal mark removers, mold cleaners, and weld anti-spatter protectants. EPA expects significant overlap in the industry sectors that use aerosol-based products; therefore, these uses are assessed together.

One example of a commercial setting with aerosol degreasing operations is repair shops, where service items are cleaned to remove any contaminants that would otherwise compromise the service item's operation. Internal components may be cleaned in place or removed from the service item, cleaned, and then re-installed once dry (U.S. EPA 2014a).

Workers at these facilities are expected to be exposed through dermal contact with and inhalation of mists during application of the aerosol product to the service item. ONUs are expected to have lower inhalation exposures and are not expected to have dermal exposures.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed to aerosol degreasers and aerosol lubricants containing PCE using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). Based on the market penetration of 29.6% and data from the BLS and U.S. Census, there are approximately 250,000 workers and 29,000 occupational non-users potentially exposed to PCE as an aerosol degreasing solvent or aerosol lubricant (see Table 2-37) (U.S. BLS 2016; U. S. Census Bureau 2015; CARB 2000).

Table 2-37. Estimated Number of Workers Potentially Exposed to PCE During Use of Aerosol Degreasers and Aerosol Lubricants

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site[a]	Total Exposed Workers[b]	Total Exposed Occupational Non-Users[b]	Total Exposed[b]
75,938	3	0.4	250,000	29,000	280,000

[a] Number of workers and occupational non-users per site are calculated by dividing the exposed number of workers or occupational non-users by the number of establishments. The number of workers per site is rounded to the nearest integer. The number of occupational non-users per site is shown as 0.4, as it rounds down to zero.
[b] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data related to the use of PCE in aerosol degreasers for brake servicing. However, PCE is used in a variety of other aerosol degreasing applications and other aerosol products for which EPA did not identify any inhalation exposure monitoring data. Therefore, EPA supplemented the identified monitoring data using the Brake Servicing Near-Field/Far-Field Inhalation Exposure Model. EPA used the brake servicing model as a representative scenario for this condition of use as there was ample data describing the brake servicing use and it is a significant use of

PCE-based aerosol products. Details of the model design and parameters is provided in Appendix H of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

Table 2-38 summarizes 8-hr TWA PBZ monitoring data and 15-min TWA PBZ monitoring data for the use of PCE-based aerosol products. The 95th percentile of the identified monitoring data is presented as the high-end exposure and the 50th percentile is presented as the central tendency. The data were obtained from three studies on the use of aerosol brake cleaners during commercial brake servicing and from data provided to EPA from the Department of Defense (DoD) (U.S. DOD and Environmental Health Readiness System - Industrial 2018; Cosgrove and Hygiene 1994; Vulcan 1993, 1992). It should be noted that one study evaluated various formulations of aerosol degreasers containing 25% PCE, and another study evaluated one formulation containing 30% PCE, and one with 60% PCE. Based on data from CARB (CARB 2000) and modeling results, PCE concentration in brake cleaning products ranges from 20% to 99% with a median concentration of 78.4%. The monitoring data collected in these two studies may underestimate "typical" exposures as the PCE concentration in the evaluated formulations were all below the median concentration.

Worker samples were determined to be any sample taken on a person while performing the aerosol degreasing tasks. ONUs samples were determined to be any sample taken on a person in the same location as the aerosol degreasing but not performing the aerosol degreasing themselves. The results only include values for workers as monitoring data for ONUs were not identified.

Table 2-38. Summary of Worker Inhalation Exposure Monitoring Data for Aerosol Degreasing

Exposure Concentration Type	Central Tendency (ppm)	High-End (ppm)	Number of Samples	Data Quality Rating of Air Concentration Data
8-hr TWA Exposure Concentration	1.4	7.8	130	High
Acute Exposure Concentration (AC)	0.5	2.6		
Average Daily Concentration (ADC)	0.3	1.8		
Lifetime Average Daily Concentration (LADC)	0.1	0.9		
15-min TWA Exposure Concentration	29	123	67	

AC = Acute Concentration; ADC = Average Daily Concentration and LADC = Lifetime Average Daily Concentration.
Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018; Cosgrove and Hygiene 1994; Vulcan 1993, 1992)

Key model inputs include number of aerosol applications per job, the amount of degreaser applied per brake job, and the concentration (weight fraction) of PCE in the aerosol degreaser. The values and distributions for these inputs are largely based on site data from maintenance and auto repair shops obtained by CARB (2000) for brake cleaning activities. The model estimates exposures for both workers and ONUs. Workers estimates are based on concentrations in the near-field where the aerosol degreasing work occurs, and ONU exposures are based on concentrations in the far-field away from the aerosol degreasing applications.

The results from model are provided in Table 2-39. It calculates both 8-hr TWA exposure concentrations and maximum 1-hr TWA exposure concentrations. The high-end and central tendency are the 95th and 50th percentiles, respectively, calculated by the model. The model exposure levels at both the central tendency and high-end for workers are higher than that found in the monitoring data but are within one order of magnitude of the monitoring data. The discrepancy is not unexpected as the model is meant to capture a wider range of shop conditions than is found in the monitoring data and the monitoring data includes data for sites using brake cleaning formulations containing concentrations less than the median concentration (78.4%) used in the model.

Table 2-39. Summary of Exposure Modeling Results for Use of PCE in Aerosol Degreasing and Aerosol Lubricants

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures		Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)	Central Tendency (ppm)	High-End (ppm)	
8-hr TWA Exposure Concentration	5.5	17	0.1	0.7	N/A – modeled data
Acute Exposure Concentration (AC)	1.8	5.7	3.4E-02	0.2	
Average Daily Concentration (ADC)	1.3	3.9	2.0E-02	0.2	
Lifetime Average Daily Concentration (LADC)	0.5	1.6	1.0E-02	7.0E-02	
Maximum 1-hr TWA Exposure Concentration	17	50	0.3	2.2	

AC = Acute Concentration; ADC = Average Daily Concentration and LADC = Lifetime Average Daily Concentration.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from several different sources, with confidence ratings of "high", as determined through EPA's systematic review process. The exposure data are supplemented with near-field/far-field exposure modeling using a Monte Carlo analysis, which incorporates variability in the model input parameters. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. Based on the reasonably available information above, EPA has a high level of confidence in the assessed exposure for this condition of use.

2.4.1.16 Dry Cleaning and Spot Cleaning

Worker Activities

Worker activities at dry cleaning shops can include:

- Receiving garments and tagging garments for identification;
- Inspecting and sorting garments by color, weight, finish;

- Pre-treating any visible stain on the garment with a spotter, typically from a spray or squeeze bottle;
- Loading garments into the machine, running the wash cycle, and unloading the cleaned garments;
- Post-spotting any stain that was not already removed during the dry cleaning process; and
- Pressing and finishing, after which the pressed garment is returned to an overhead rack and wrapped in plastic for customer pickup (NIOSH 1997a).

EPA expects worker exposure at dry cleaning facilities to primarily occur when workers are: 1) unloading and loading garments from the machines; 2) performing manual stain removal (i.e., spot cleaning); and 3) transferring solvent from a storage container to the machine. Workers can also be exposed during maintenance activities, such as cleaning the machine lint trap, button trap and still, changing solvent filters, and disposing hazardous wastes. However, these maintenance activities occur on a much less frequent basis (NIOSH 1997a).

ONUs at dry cleaning facilities are employees who are not expected to handle PCE, operate dry cleaning machines, or perform spotting or finishing operations. They include cashiers, counter clerks and other similar employees.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed to PCE at dry cleaners using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015). Based on a market penetration of 60% for commercial facilities, assuming 12 industrial dry cleaners (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate), and data from the BLS and U.S. Census, there are approximately 44,000 workers and 14,000 occupational non-users potentially exposed to PCE at dry cleaning facilities (see Table 2-40) (DLI/NCA 2017; U.S. BLS 2016; U. S. Census Bureau 2015; U.S. EPA 2006b).

Table 2-40. Estimated Number of Workers Potentially Exposed to PCE During Dry Cleaning

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
12,834	3	1	44,000	14,000	57,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

Table 2-41 summarizes the 8-hr TWA PBZ monitoring data for workers and ONUs at dry cleaners obtained from OSHA facility inspections, NIOSH studies and data provided to EPA from DoD (U.S. DOD and Environmental Health Readiness System - Industrial 2018; OSHA 2017; Burroughs 2000; NIOSH 2000; Burroughs 1999a, b; NIOSH 1995). The data are divided into two categories: 1) statistics for data collected after the promulgation of the 2006 PCE NESHAP for Dry Cleaning Facilities; and 2) data collected for fourth or fifth generation machines only. The post-2006 NESHAP data are expected to contain exposures from shops using third, fourth and fifth generation machines as the purchase of new first generation (transfer machines) and second generation (dry-to-dry, vented machines) dry cleaning

machines were banned in the 1993 Perchloroethylene NESHAP for Dry Cleaning Facilities, the 2006 Perchloroethylene NESHAP for Dry Cleaning Facilities banned the use of PCE in all first-generation machines, and the typical useful life of these machines is approximately 15 years (U.S. EPA 2006b).

Third generation equipment are non-vented, dry-to-dry machines with refrigerated condensers. These machines are essentially closed systems and are only open to the atmosphere when the machine door is opened. In third generation machines, heated drying air is recirculated back to the drying drum through a vapor recovery system (NIOSH 1997b).

Fourth generation dry cleaning equipment are essentially third-generation machines with added secondary vapor control. These machines "rely on both a refrigerated condenser and carbon adsorbent to reduce the PCE concentration at the cylinder outlet below 300 ppm at the end of the dry cycle" and are more effective at recovering solvent vapors (NIOSH 1997b). Fifth generation equipment have the same features as fourth generation machines, but also have a monitor inside the machine drum and an interlocking system to ensure that the concentration is below approximately 300 ppm before the loading door can be opened (NIOSH 1997b).

For workers, the 95th percentile is presented as the high-end and the 50th percentile is presented as the central tendency. For the post-2006 NESHAP data, only a single data point was available for ONUs. For fourth and fifth generation machines, there was only four ONU data points available; therefore, the maximum is presented as the high-end and the median as the central tendency.

Approximately 28% of respondents to a 2003 survey of California dry cleaners indicated they used fourth generation machines and approximately 61% of respondents to a 2010 survey of dry cleaners in King County, WA reported using fourth or fifth generation machines (Whittaker and Johanson 2011; California Air Resources 2006). EPA did not identify data for other locales or for the overall U.S.; therefore, EPA used the California and King County, WA data to approximate the overall U.S. trends. Based on these survey results, EPA expects the industry to be trending towards higher usage of fourth and fifth generation machines as compared to third generation machines and expects current exposures at dry cleaning shops to fall somewhere between the post-2006 exposure concentrations and the concentrations from fourth and fifth generation machines only.

Worker samples were determined to be any sample taken on a person who engages in loading/unloading clothes from dry cleaning equipment, finishing operations, spot cleaning, and/or maintenance activities for the dry cleaning machine (e.g., replenishing spent solvent). ONUs samples were determined to be any sample taken on a person not expected to perform these activities (e.g., cashiers).

3668 **Table 2-41. Summary of Inhalation Exposure Monitoring Data for Dry Cleaning**

Data Category	Exposure Concentration Type	Worker Exposures		Number of Worker Samples	Occupational Non-User Exposures		Number of ONU Samples	Data Quality Rating of Air Concentration Data
		Central Tendency (ppm)	High-End (ppm)		Central Tendency (ppm)	High-End (ppm)		
Post-2006 NESHAP Data[a]	8-hr TWA Exposure Concentration	3.6	20	21	0.3[c]		1[d]	High
	Acute Exposure Concentration (AC)	1.2	6.5		0.1	0.1		
	Average Daily Concentration (ADC)	0.9	5.2		8.2E-02	9.3E-02		
	Lifetime Average Daily Concentration (LADC)	0.3	2.7		3.3E-02	4.8E-02		
	15-min TWA Exposure Concentration	33	94	9	No 15-min data identified for ONUs			
Fourth and Fifth Generation Statistics[b]	8-hr TWA Exposure Concentration	1.0	5.6	114	1.4E-02	0.1	4	High
	Acute Exposure Concentration (AC)	0.3	1.9		4.7E-03	4.1E-02		
	Average Daily Concentration (ADC)	0.2	1.5		3.3E-03	3.3E-02		
	Lifetime Average Daily Concentration (LADC)	9.2E-02	0.8		1.3E-03	1.7E-02		
	15-min TWA Exposure Concentration	48	899	6	No 15-min data identified for ONUs			

3669 AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] Post-2006 NESHAP data are air samples collected from OSHA inspections or DoD and, based on the date of collection, EPA assumed to be representative of the post-2006 mix of machine types as provided in the 2010 King County, WA survey (Whittaker and Johanson 2011).
[b] Fourth and fifth generation data include only data where EPA could clearly identify the machine type in the study as fourth or fifth generation. It does not include OSHA data, which are representative of a mix of machine generations but for which machine types for individual samples could not be determined.
[c] Only one data point was available for this scenario. However, different parameters are used for calculating high-end and central tendency ADC and LADC. Therefore, a high-end and central tendency are presented based on the single data point.
[d] The single ONU data point comes from a sample taken on an inspector at a dry cleaning site. EPA assumes exposures to the inspector would be similar to that of an ONU as inspectors are not expected to handle the chemical or operator dry cleaning machines.
Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018; OSHA 2017; Burroughs 2000; NIOSH 2000; Burroughs 1999a, b; NIOSH 1995)

As estimated in Section 2.2.1.2.2, PCE is expected to be used in thousands of dry cleaning shops throughout the U.S. and the monitoring data only captures a small fraction of those shops. Therefore, EPA supplemented the identified monitoring data using the Dry cleaning Multi-Zone Inhalation Exposure Model to capture variation amongst dry cleaning shops that may not be captured in the monitoring data. Details of the model design and parameters are provided in Appendix I of *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d).

Key model input parameters include solvent in concentration in the dry cleaning machine after the clean cycle has complete, residual solvent in clothing removed from the dry cleaning machine, and spot cleaning use rates. The value and distribution used for each of these parameters in the model are based on data observed in literature. The model estimates exposures for workers, spot cleaners, and ONUs. Workers estimates are based on concentrations in the near-field zone corresponding to unloading clothes from the dry cleaning equipment and the near-field zone corresponding to where finishing and pressing activities occur. Spot cleaner estimates are based on concentrations in the near-field zone corresponding to where the spot cleaning activity occurs. ONU exposures are based on concentrations in the far-field which corresponds to any area outside the near-field zones. The results from the model are provided in Table 2-42. The high-end and central tendency are the 95th and 50th percentiles, respectively, calculated by the model. It should be noted that the model calculates 12-hr TWAs based on suggestions from the peer review of the 2016 Draft Risk Assessment for the TSCA Work Plan Chemical 1-Bromopropane that dry cleaning workers may work up to 12 hours per day (U.S. EPA 2016e).

It should be noted that EPA did not identify information to estimate the use rate of PCE in spot cleaners; however, IRTA (2007) and ERG (2005) indicate that the use of PCE in spot cleaners is minimal. Specifically, IRTA (2007) state that only 150 gal of PCE -based spotting agents are used annually in California (compared to 42,000 gal of PCE -based spotting agents). ERG (2005) stated that many PCE spotting agents are categorized as oily type paint removers (OTPR), but that the majority of OTPR spotting agents contain no PCE. Therefore, EPA set the use rate of PCE spotting agents to zero causing the spotting zone of the model to become part of the far-field with exposure concentrations equivalent to ONUs.

When comparing the model results to the post-2006 NESHAP monitoring data results for workers, the model high-end is higher than the monitoring data. This is likely because the model is meant to capture a wider range of conditions than is likely captured in the monitoring data. The model central tendency for workers is slightly less than half the central tendency for the post-2006 NESHAP monitoring data. This may be due to the fact the majority of the post-2006 NESHAP data are from OSHA compliance

inspections that are often performed as a result of worker complaints and, therefore, may not necessarily be representative of PCE concentrations encountered in the typical commercial dry cleaning establishment. Additionally, the assumption that post-2006 NESHAP data is representative of the 2010 King County, WA survey results may be inaccurate, and the data could actually represent sites with a higher frequency of third generation machines, resulting in higher exposures. However, model results and monitoring data for the post-2006 NESHAP are within the same order of magnitude.

When comparing the model results to the fourth/fifth generation monitoring data results for workers, the model high-end and central tendency are both an order of magnitude greater than the monitoring data. This is expected as the model captures exposures from facilities with third and fourth/fifth generation machines.

Table 2-42. Summary of Worker and Occupational Non-Uses Inhalation Exposure Modeling Results for Dry Cleaning

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures		Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)	Central Tendency (ppm)	High-End (ppm)	
8-hr TWA Exposure Concentration	1.4	30	0.1	1.5	N/A – modeled data
Acute Exposure Concentration (AC)	0.7	15	5.4E-02	0.8	
Average Daily Concentration (ADC)	0.5	10	3.8E-02	0.6	
Lifetime Average Daily Concentration (LADC)	0.2	4.1	1.4E-02	0.2	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from several different sources, with confidence ratings of "high", as determined through EPA's systematic review process. The exposure data are supplemented with multi-zone exposure modeling using a Monte Carlo analysis, which incorporates variability in the model input parameters. This model was peer reviewed as part of the 2016 1-BP draft Risk Assessment (U.S. EPA 2016f) has been updated to address peer review comments, incorporate additional available data, and use PCE-relevant data. Although the model results differ from the monitoring data, they are the same order of magnitude as the post-2006 NESHAP data. The model results are higher than the fourth and fifth generation machine monitoring data which is expected as the model incorporates third generation machines. Based on the reasonably available information above, EPA has a high level of confidence in the assessed exposure for this condition of use.

2.4.1.17 Adhesives, Sealants, Paints, and Coatings

Worker Activities

Worker activities may include unloading adhesive or coating products from containers into application equipment, and, where used, manual application of the adhesive or coatings (e.g., use of spray guns or brushes to apply product to substrate) (OECD 2015). Workers may be exposed to PCE during the application process if mists are generated such as during spray and roll applications (OECD 2015). Workers may also be exposed to PCE vapors that evaporate from the adhesive or coating as it is applied or during the drying/curing process (OECD 2015). EPA expects ONUs may be exposed to mists or vapors that enter their breathing zone during routine work in areas where coating applications are occurring.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE-containing adhesives and coatings using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the NAICS code reported by sites in the 2014 NEI (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate) (U.S. EPA 2018a). In the 2014 NEI, there were two sites with coating operations that reported a NAICS code for which no Census data were available. To estimate the number of workers and ONUs at these sites, EPA used the average workers per site and ONUs per site from the sites with known data. There are approximately 410 workers and 160 ONUs potentially exposed during use of adhesives/sealants and 1,900 workers and 1,100 ONUs potentially exposed during use of paints/coatings (see Table 2-43).

Table 2-43. Estimated Number of Workers Potentially Exposed to PCE During of Use Adhesives, Sealants, Paints, and Coatings

Scenario	Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
Adhesives/Sealants	14	30	11	410	160	570
Paints/Coatings	46	41	24	1,900	1,100	3,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from a study at a single site in Poland using a PCE-based adhesive, from three NIOSH investigations at three sites using PCE-based coatings, a study submitted to EPA under TSCA for a truck plant using PCE-based coatings, and data provided to EPA from DoD for spray coating processes (U.S. DOD and Environmental Health Readiness System - Industrial 2018; Gromiec et al. 2002; Hanley 1993; Stephenson and Albrecht 1986; Chrostek and Levine 1981; Ford Motor 1981). Due to the large variety in shop types that may use PCE-based adhesives and coatings, it is unclear how representative these data are of a "typical" site using these products. However, EPA does not have a model for estimating exposures from use of adhesives or paints/coatings; therefore, the assessment is based on the identified monitoring data. Table 2-44 summarizes the identified monitoring data.

Worker samples were determined to be any sample taken on a person while performing adhesive or coating applications. ONUs samples were determined to be any sample taken on a person in the same location as the applications but not performing the adhesive/coating application themselves. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

For adhesives, the study did not provide discrete sample results; therefore, the high-end exposure value is based on the max concentration and the central tendency is based on the mean reported in the study (Gromiec et al. 2002). For paints/coatings 8-hr TWA, the 95th percentile of the data is presented as the high-end and the 50th percentile as the central tendency. Due to the limited number of data points for the 15-minute TWA, the maximum is presented as the high-end and the median is the central tendency.

Table 2-44. Summary of Inhalation Exposure Monitoring Data for Use of PCE-Based Adhesives, Sealants, Paints, and Coatings

Scenario	Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
		Central Tendency (ppm)	High-End (ppm)			
Adhesives/ Sealants	8-hr TWA Exposure Concentration[b]	8.8E-02	0.8	13	8.8E-02	Medium
	Acute Exposure Concentration (AC)	2.9E-02	0.3		2.9E-02	
	Average Daily Concentration (ADC)	2.0E-02	0.2		2.0E-02	
	Lifetime Average Daily Concentration (LADC)	8.0E-03	9.5E-02		8.0E-03	
Paints/ Coatings	8-hr TWA Exposure Concentration	0.2	4.6	15	0.2	Medium to High
	Acute Exposure Concentration (AC)	7.8E-02	1.5		7.8E-02	
	Average Daily Concentration (ADC)	5.3E-02	1.0		5.3E-02	
	Lifetime Average Daily Concentration (LADC)	2.1E-02	0.5		2.1E-02	
	15-min TWA Exposure Concentration	4.1	7.9	5	4.1	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] Exact sample times not given in study; however, study indicates that samples were taken for a minimum of 75% of the shift (360 min). Therefore, EPA assumes that the results are representative of an 8-hr TWA exposure.
Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018; Gromiec et al. 2002; Hanley 1993; Stephenson and Albrecht 1986; Chrostek and Levine 1981; Ford Motor 1981)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data from several different sources, with confidence rating of the data ranging from medium to high, as determined through EPA's systematic review process. Due to potential variations in the types of sites that may use PCE-based adhesives, sealants, paints, and coatings, there is some uncertainty in how representative the monitoring data are of other sites using these types of products. Despite this uncertainty, EPA has a medium level of confidence in the assessed worker exposure for this condition of use.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.18 Maskant for Chemical Milling

Worker Activities

Information from stakeholder meetings and public comments indicate that in typical maskant application processes the potential for exposure is low as the process is automated and performed in a dedicated room (Ducommun 2017; Spirit AeroSystems 2017; Tech Met 2017). However, at least one stakeholder indicated that employees may be exposed during maintenance operations (Spirit AeroSystems 2017). Specific maintenance activities were not described but may include adding fresh maskant and handling of re-captured maskants.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE as a chemical maskant using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by sites in the 2016 TRI, 2016 DMR, and/or the 2014 NEI (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate).

The data from the 2016 TRI, 2016 DMR, and 2014 NEI only covers 28 unique sites; however, market data from ACP indicates there are up to 71 sites using PCE-based maskants (Products 2017). To estimate the number of workers and ONUs at the remaining sites EPA calculated the average number of workers and ONUs per site from the 28 known sites. This resulted in 95 workers per site and 75 ONUs per site at the unknown sites and a total of approximately 6,700 workers and 5,300 ONUs potentially exposed during maskant uses of PCE (see Table 2-45).

Table 2-45. Estimated Number of Workers Potentially Exposed to PCE During Use of Chemical Maskants

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
71	94	75	6,700	5,300	12,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from a single NIOSH investigation at an aircraft parts manufacturing site using a dip coating application process for the maskants (Hervin et al. 1977). The NIOSH report does not specify if PCE is the primary solvent in the maskant, the concentration of PCE in the maskant, or the typical maskant use rates at the site. The identified monitoring data also included 15-min TWA samples collected by the DoD between July 2013 and May 2017 during masking activities (U.S. DOD and Environmental Health Readiness System - Industrial 2018). The DoD data contained nine samples that were measured below the LOD (U.S. DOD and Environmental Health Readiness System - Industrial 2018). To estimate exposure concentrations for data below the LOD, EPA followed the *Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b) as discussed in Section 1.4.5.2. The geometric standard deviation for the data was above 3.0; therefore, EPA used the $\frac{LOD}{2}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b).

Due to uncertainty in worker activities for chemical milling operations, EPA typically identified samples as worker samples unless it was explicitly clear from the job title and the description of activities in the report that the employee was not working with the maskant chemicals during the sampling period. Samples from employees determined not to be working with the maskant chemicals were designated as ONU samples. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Due to the variety in both industry types and typical per site maskant use rates and the uncertainty of the PCE concentration in the maskant, it is unclear if these data are representative of a "typical" site. Additionally, the 8-hr and 4-hr data were collected prior to the promulgation of the Aerospace Manufacturing and Rework Facilities NESHAP which regulates the emissions of hazardous air pollutants (HAPs) from various operation at aerospace facilities including chemical milling. To the extent that this NESHAP reduces emissions of PCE into the workroom worker exposures may be lower than identified data. EPA does not have a model for estimating exposures from maskant uses; therefore, the assessment is based on the identified monitoring data. Table 2-46 summarizes the 8-hr, 4-hr, and 15-min TWA monitoring data for the use of PCE in maskants. The 95th percentile of the data is presented as the high-end and the 50th percentile as the central tendency.

Table 2-46. Summary of Inhalation Exposure Monitoring Data for Chemical Maskants

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-Uses Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	1.2	2.1	24	1.2	High
Acute Exposure Concentration (AC)	0.4	0.7		0.4	
Average Daily Concentration (ADC)	0.3	0.5		0.3	
Lifetime Average Daily Concentration (LADC)	0.1	0.2		0.1	
15-min TWA Exposure Concentration	0.6	28	20	0.6	
4-hr TWA Exposure Concentration	2.4	3.2	9	2.4	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018; Hervin et al. 1977)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data from two sources with a confidence rating of "high", as determined through EPA's systematic review process. However, the 8-hr TWA data were collected prior to the Aerospace Manufacturing and Rework Facilities NESHAP. There is some uncertainty in how implementing the requirements of the NESHAP may have reduced worker exposures (if at all). Despite this uncertainty, EPA has a medium to high level of confidence in the assessed worker exposure for this condition of use, based on the strength of the monitoring data.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.19 Industrial Processing Aid

Worker Activities

At industrial facilities, workers are potentially exposed when unloading PCE from transport containers into intermediate storage tanks and process vessels. Workers may be exposed via inhalation of vapor or via dermal contact with liquids while connecting and disconnecting hoses and transfer lines. Once PCE is unloaded into process vessels, it may be consumed in the process (e.g. when used for catalyst regeneration) or be used until spent and sent for disposal.

ONUs are employees who work at the facilities that process and use PCE, but who do not directly handle the material. ONUs may also be exposed to PCE 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.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during use of PCE as a processing aid using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). This results in approximately 14,000 workers and 6,000 ONUs potentially exposed during use of PCE as a processing aid (see Table 2-47).

Table 2-47. Estimated Number of Workers Potentially Exposed to PCE During Use of Processing Aids

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
98	140	61	14,000	6,000	20,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from four studies submitted to EPA under TSCA by Dow Chemical(Dow Chem 1983a, b, 1982, 1979). The exact function of PCE is each study is not explicitly stated; however, the data was collected in the agricultural chemical production and distribution, trichloroethylene production, and chloropyridines process areas. Based on CDR reporting, PCE is used as a processing aid in agricultural chemical manufacturing; therefore, monitoring data collected in the agricultural chemical production area is assessed as a processing aid use of PCE. Similarly, chloropyridines are used as intermediates in both the pharmaceutical and agrochemical industries (Scriven and Murugan 2005). Both pharmaceutical and agrochemical industries are expected to use PCE as a processing aid; therefore, monitoring data collected in the chloropyridine unit are also assessed as a processing aid use. PCE can also be used as an inert material in trichloroethylene production (Snedecor et al. 2004). Use as an inert material would fall under processing aid uses; therefore, monitoring data collected during trichloroethylene production is assessed as a processing aid use.

Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-48 presents a summary of the identified 8-hr TWA and 30-minute TWA monitoring data. For the 8-hr TWA, the 95th percentile is presented as the high-end and the 50th percentile presented as the central tendency. It should be noted that approximately 55% of the 8-hr TWA data were below the LOD.

To estimate exposure concentrations for these data, EPA followed *the Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b). The geometric standard deviation for the data was above 3.0; therefore, EPA used the $\frac{LOD}{2}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b). Because over 50% of the data are below the LOD, calculating statistics from this data does present the potential to introduce biases into the results. Estimation of exposure values for results below the LOD may over- or under-estimate actual exposure thus skewing the calculated statistics higher or lower, respectively. The overall directional bias of the exposure assessment, accounting for both the overestimate and underestimate, is not known.

For the 30-minute TWA, only two data point were available, one of which measured below the LOD. Because only a single data point with a measured value was available, EPA could not calculate a geometric standard deviation. Therefore, EPA presents two scenarios: 1) using the maximum as a "higher value"; and 2) using the midpoint between the maximum and the LOD as a "midpoint" value.

Table 2-48. Summary of Worker Inhalation Exposure Monitoring Data for Use of PCE as a Processing Aid

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	6.0E-02	1.2	89	6.0E-02	Medium
Acute Exposure Concentration (AC)	2.0E-02	0.4		2.0E-02	
Average Daily Concentration (ADC)	1.4E-02	0.3		1.4E-02	
Lifetime Average Daily Concentration (LADC)	5.4E-03	0.1		5.4E-03	
30-min TWA Exposure Concentration[b]	1.7	2.2	2	1.7	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] Due to only two data points, one of which measured below the LOD, EPA presents two scenarios: 1) using the higher of the two values; and 2) using the midpoint of the LOD and the maximum.
Source: (Dow Chem 1983a, b, 1982, 1979)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data from several different sources all with a confidence rating of "medium," as determined through EPA's systematic review process. There is some uncertainty in how PCE is used within each process, but literature corroborates categorizing the use as a processing aid. Based on the available information above, EPA has a medium level of confidence in the assessed worker exposure for this condition of use.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is

expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.20 Metalworking Fluids

Worker Activities

Workers are expected to unload the metalworking fluid from containers; clean containers; dilute water-based metalworking fluids; transfer fluids to the trough; performing metal shaping operations; rinse, wipe, and/or transfer the completed part; change filters; transfer spent fluids; and clean equipment (OECD 2011).

ONUs include employees that work at the site where PCE is used in an industrial setting as a metalworking fluid, but they typically do not directly handle the chemical and are therefore expected to have lower exposures. ONUs for metalworking fluids include supervisors, managers, and tradesmen that may be in the processing area but do not perform tasks that result in the same level of exposures as machinists.

Since PCE has a high vapor pressure (18.5 mmHg at 25°C), workers may be exposed to PCE when handling liquid metalworking fluid, such as unloading, transferring, and disposing spent metalworking fluids and cleaning machines and troughs. The greatest source of potential exposure is during metal shaping operations. The high machine speeds can generate airborne mists of the metalworking fluids to which workers can be exposed. Additionally, the high vapor pressure of PCE may lead to its evaporation from the airborne mist droplets, potentially creating a fog of vapor and mist.

Number of Workers and Occupational Non-Users

The ESD on the Use of Metalworking Fluids cites a NIOSH study of 79 small machine shops, which observed an average of 46 machinists per site (OECD 2011). The ESD also cites an EPA effluent limit guideline development for the MP&M industry, which estimated a single shift supervisor per shift, who may perform tasks such as transferring and diluting neat metalworking fluids, disposing spent metalworking fluids, and cleaning the machines and troughs (OECD 2011). Since the machinists perform the metal shaping operations, during which metalworking fluid mists are generated, EPA assesses the machinists as workers, as they have the highest potential exposure. EPA assessed the single shift supervisor per site as an ONU, as this employee is not expected to have as high an exposure as the machinists. Assuming two shifts per day (hence two shift supervisors per day), EPA assesses 46 workers and two ONUs per site (OECD 2011). The number of establishments that use PCE-based metalworking fluids is unknown (see discussion in the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d)); therefore, EPA does not have data to estimate the total workers and ONUs exposed to PCE from use of metalworking fluids.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation exposure monitoring data related to the use of PCE-based metalworking fluids. Therefore, EPA assessed inhalation exposures using the ESD on the Use of Metalworking Fluids (OECD 2011). The ESD estimates typical and high-end exposures for different types of metalworking fluids. The "typical" mist concentration is the geometric mean of the data and the "high-end" is the 90th percentile of the data (OECD 2011). The recommended use of the PCE-based metalworking fluid is an oil-based cutting and tapping fluid; therefore, EPA assesses exposure to the PCE-based metalworking fluids using the straight oil mist concentrations and the max concentration of

PCE in the metalworking fluid. Straight oils are not diluted; therefore, the concentration of PCE specified in the identified SDS (<10%) is equal to the concentration of PCE in the mist.

Table 2-49 presents the exposure estimates for the use of PCE-based metalworking fluids. It should be noted that these estimates may underestimate exposures to PCE during use of metalworking fluids as they do not account for exposure to PCE that evaporates from the mist droplets into the air. This exposure is difficult to estimate and is not considered in this assessment. However, due to the relatively low concentration of PCE in the metalworking fluid, the partial pressure may be low enough such that evaporation of PCE from the mist is limited and this not a significant route of exposure.

The results only include values for workers as the ESD does not include an approach for estimating ONU exposures. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-49. Summary of Exposure Results for Use of PCE in Metalworking Fluids Based on ESD Estimates

Exposure Concentration Type	Worker Exposure		Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration[b]	5.8E-03	2.1E-02	5.8E-03	N/A – ESD data
Acute Exposure Concentration (AC)	1.9E-03	7.0E-03	1.9E-03	
Average Daily Concentration (ADC)	1.3E-03	4.8E-03	1.3E-03	
Lifetime Average Daily Concentration (LADC)	5.2E-04	2.5E-03	5.2E-04	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

[b] The PCE exposure concentrations are calculated by multiplying the straight oil mist concentrations in the ESD by 10% (the concentration of PCE in the metalworking fluid) and converting to ppm.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using estimates from the Metalworking Fluid ESD for typical and high-end mist exposures for straight oils. The ESD estimates are for a "generic" straight oil rather than a PCE-specific metalworking fluid; therefore, there is some uncertainty in how this data applies to PCE-based metalworking fluids. Additionally, the ESD estimates also only account for the exposure to mist; however, PCE is volatile and expected to evaporate from the mist into the air. Therefore, the ESD estimates may underestimate actual PCE exposure. Due to the low concentration of PCE in the metalworking fluid, the partial pressure of PCE in the mist may be low enough such that this is not a significant route of exposure, thus mitigating the overall underestimate. Based on the available information above, EPA has a medium level of confidence in the assessed worker exposure for this condition of use.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.21 Wipe Cleaning and Metal/Stone Polishes

Worker Activities

Workers are expected to be exposed to PCE vapors that evaporate from the PCE-soaked rag or the solvent residue left behind on the substrate after wiping. Additional activities and use patterns will vary depending on the specific site at which the PCE cleaning product or polish is being used.

Number of Workers and Occupational Non-Users

EPA did not identify information to estimate the number of workers or ONUs exposed to PCE during use for wipe cleaning and metal/stone polishes. It is possible some workers/ONUs at sites using vapor degreasers or cold cleaners are also exposed to PCE from wipe cleaning activities.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE for wipe cleaning (Moody et al. 1983; Gunter and Lybarger 1979). EPA did not identify exposure data specific to metal/stone polish applications; therefore, these data were also used to assess the use of metal/stone polishes based on expected similarities in the uses. Due to the large variety in the types of shops that may use PCE as a wipe cleaning solvent or metal/stone polish, it is unclear how representative these data are of a "typical" site. EPA does not have a model for estimating exposures from wipe cleaning or metal/stone polishes; therefore, the assessment is based on the identified monitoring data. Table 2-50 summarizes 8-hr, 4-hr and 15-minute TWA monitoring data for the use of PCE as a wipe cleaning solvent and metal/stone polish.

Worker samples were determined to be any sample taken on a person while performing the wipe cleaning or polishing task. ONUs samples were determined to be any sample taken on a person in the same location as the wipe cleaning or polishing task but were not performing the wipe cleaning or polishing themselves.

Due to the limited number of data points for workers 8-hr and 15-minute TWA results, the maximum of identified data is presented as the high-end and the median is presented as the central tendency. There is only a single 4-hr TWA data point for workers. Results based on a single value are plausible exposure concentrations, but EPA cannot determine the statistical representativeness of the value. For the ONU 8-hr TWA, the 95th percentile is presented as the high-end and the 50th percentile as the central tendency. The ONU data included four data points that are below the LOD. To estimate exposure concentrations for these data, EPA followed the *Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b). The geometric standard deviation for the data was above 3.0; therefore, EPA used the $\frac{LOD}{2}$ to estimate the exposure value as specified in the guidelines (U.S. EPA 1994b).

Table 2-50. Summary of Worker Inhalation Monitoring Data for Use of PCE as a Wipe Cleaning Solvent and Metal/Stone Polish

Exposure Concentration Type	Worker Exposures		Number of Worker Samples	Occupational Non-User Exposures		Number of ONU Samples	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	132	228	4	2.2E-02	23	6	High
Acute Exposure Concentration (AC)	44	76		7.3E-03	7.7		
Average Daily Concentration (ADC)	30	52		5.0E-03	5.3		
Lifetime Average Daily Concentration (LADC)	12	27		2.0E-03	2.7		
15-min TWA Exposure Concentration	66	103	9	No 15-min or 4-hr data identified for ONUs			
4-hr TWA Exposure Concentration	9.5		1				

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
Source: (Moody et al. 1983; Gunter and Lybarger 1979)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from two sources with a confidence rating of "high", as determined through EPA's systematic review process. There is some uncertainty in how representative this data is of exposure at other facilities performing wipe cleaning or polishing tasks. The data identified is also specific to wipe cleaning activities not polishing. Although the application processes are expected to be similar, the frequency and duration of polish applications may be less than those used for wipe cleaning. Therefore, the exposure values may overestimate exposures during use of polishes. Despite these uncertainties, EPA has a medium level of confidence in the assessed exposure for this condition of use.

2.4.1.22 Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

Worker Activities

As previously described, workers are expected to spray PCE on to the stained textiles and then manually scrape away the stain using a brush or fingers.

Number of Workers and Occupational Non-Users

EPA did not identify information to estimate the total number of workers and ONUs exposed from use of spot cleaners/spot removers. Both the Fabric Finishing GS (U.S. EPA 1994a) and the ESD on the Use of Textile Dyes (OECD 2017b) estimate three to six workers exposed per site. It is unknown how many of those workers may be involved in the spot cleaning process.

Occupational Inhalation Exposure Results

EPA identified inhalation exposure monitoring data from a single NIOSH investigation at a garment manufacturer (Burton and Monestersky 1996). It is unclear how representative these data are of a "typical" spot cleaning/spot remover scenario. Table 2-51 summarizes the 8-hr TWA monitoring data for the use of PCE in spot cleaners/spot removers.

Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE.

Table 2-51. Summary of Worker Inhalation Exposure Monitoring Data for Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

Exposure Concentration Type	Worker Exposures[a]		Number of Worker Samples	Occupational Non-User Exposures[b]		Number of ONU Samples	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	0.2	0.2	2	3.0E-02		1	High
Acute Exposure Concentration (AC)	5.7E-02	7.7E-02		1.0E-02	1.0E-02		
Average Daily Concentration (ADC)	3.9E-02	5.3E-02		6.8E-03	6.8E-03		
Lifetime Average Daily Concentration (LADC)	1.6E-02	2.7E-02		2.7E-03	3.5E-03		

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] Due to only two data points identified for workers, EPA presents two scenarios: 1) using the higher of the two values; and 2) using the midpoint of the two values.
[b] Only one data point identified for ONUs; however, different parameters are used for calculating high-end and central tendency ADC and LADC. Therefore, a high-end and central tendency are presented based on the single data point.
Source: (Burton and Monestersky 1996)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure is assessed using PCE personal breathing zone monitoring data from a single source with a confidence rating of "high", as determined through EPA's systematic review process. There is some uncertainty in how representative this data is of exposure at other facilities performing carpet cleaning or spot remover tasks. Based on the available information above, EPA has a medium level of confidence in the assessed exposure for this condition of use.

2.4.1.23 Other Industrial Uses

Worker Activities

Based on information identified in EPA's preliminary data gathering and information obtained from TRI and DMR, a variety of other industrial uses of PCE may exist. Based on information in the Use

Document (U.S. EPA 2017f), market profile (U.S. EPA 2017b), and NAICS/SIC codes reported in TRI (U.S. EPA 2017k) and DMR (U.S. EPA 2016a), examples of these uses include, but are not limited to, uses in textile processing, wood furniture manufacturing, foundry applications, food manufacturing, and scientific research and development. EPA did not identify information on how PCE may be used at these facilities

Although information on worker activities at these sites was not identified, EPA expects workers to perform activities similar to other industrial facilities. Therefore, workers may potentially be exposed when unloading PCE from transport containers into intermediate storage tanks and process vessels. Workers may be exposed via inhalation of vapor or via dermal contact with liquids while connecting and disconnecting hoses and transfer lines.

ONUs are employees who work at the facilities that process and use PCE, but who do not directly handle the material. ONUs may also be exposed to PCE 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.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during processing of PCE as a reactant using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively (see the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d) for number of sites estimate). In the 2016 DMR (U.S. EPA 2016a) there was one site that did not report a SIC code but after review of the company's website, EPA determined that NAICS 311411 – Frozen Fruit, Juice, and Vegetable Manufacturing was the most appropriate NAICS code to use for this site. There are approximately 2,700 workers and 1,300 ONUs potentially exposed during other industrial uses (see Table 2-52).

Table 2-52. Estimated Number of Workers Potentially Exposed to PCE During Other Industrial Uses

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
130	21	10	2,700	1,300	4,000

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation exposure monitoring data for the other industrial uses. Therefore, EPA assessed inhalation exposures during these uses using the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model, assuming PCE is present at 100 percent concentration when used. Details of the model design and parameters is provided in Appendix E of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). Table 2-53 summarizes the model results.

The results only include values for workers as the model does not estimate ONU exposures. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-53. Summary of Exposure Modeling Results for Other Industrial Uses of PCE

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	8.0E-03	3.6E-02	8.0E-03	N/A – modeled data
Acute Exposure Concentration (AC)	2.7E-03	1.2E-02	2.7E-03	
Average Daily Concentration (ADC)	1.8E-03	8.2E-03	1.8E-03	
Lifetime Average Daily Concentration (LADC)	7.2E-04	4.2E-03	7.2E-04	
30-min TWA Exposure Concentration	0.1	–[b]	0.1	
1-hr TWA Exposure Concentration	–[b]	0.3	–[b]	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] High-end for short-term exposures is calculated as a 1-hr TWA and central tendency is calculated as a 30-min TWA.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

The Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model is used to estimate worker exposure. The model uses a combination of published EPA emission factors and engineering judgment to estimate central tendency and high-end exposures. EPA believes the model exposures are likely to be representative of exposure associated with bulk container loading. However, the model does not account for other potential sources of exposure at industrial facilities, such as sampling, equipment cleaning, and other process activities. The model also assumes only one container is loaded per day, although larger facilities may have higher product loading frequencies. These model uncertainties could result in an underestimate of the worker exposure. Based on reasonably available information above, EPA has a medium level of confidence in the assessed worker exposure.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.24 Other Commercial Uses

Worker Activities

The worker activity, use pattern, and associated exposure will vary for each condition of use. For polishes, ink removal products, and mold release, EPA expects workers may be exposed to PCE vapors that evaporate from the application material (rag, brush, etc.) or the substrate surface during use. For

inks, workers may be exposed to mists generated during the ink application process. For photographic film, workers may be exposed to PCE that evaporates from the gating process.

Number of Workers and Occupational Non-Users

EPA has not identified information on the number of sites and potentially exposed workers associated with these uses. The use of PCE for these conditions of use is expected to be minimal.

Occupational Inhalation Exposure Results

EPA assessed exposure to other commercial uses of PCE using data from identified studies. EPA identified exposure data for printing uses (inks and ink removal products), photocopy shops, photographic film, and mold release uses. Table 2-54 summarizes the 8-hr TWA and 15-min TWA data identified for these uses. Note: Data for mold release products are area samples not worker breathing zone samples; it is unclear how representative area samples are of actual exposures.

Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-54. Summary of Exposure Monitoring Data for Other Commercial Uses of PCE

Scenario	Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
		Central Tendency (ppm)	High-End (ppm)			
Printing Applications (Ink and Ink Removal Products)	8-hr TWA Exposure Concentration	1.9	5.9	23	1.9	Medium to High
	Acute Exposure Concentration (AC)	0.6	2.0		0.6	
	Average Daily Concentration (ADC)	0.4	1.4		0.4	
	Lifetime Average Daily Concentration (LADC)	0.2	0.7		0.2	
	15-min TWA Exposure Concentration	0.2		1	0.2	
Photocopying	8-hr TWA Exposure Concentration	1.9E-04	5.0E-04	3	1.9E-04	High
	Acute Exposure Concentration (AC)	6.3E-05	1.7E-04		6.3E-05	

Scenario	Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
		Central Tendency (ppm)	High-End (ppm)			
	Average Daily Concentration (ADC)	4.3E-05	1.1E-04		4.3E-05	
	Lifetime Average Daily Concentration (LADC)	1.7E-05	5.9E-05		1.7E-05	
Photographic Film Applications	8-hr TWA Exposure Concentration	6.3	56	62	6.3	Medium
	Acute Exposure Concentration (AC)	2.1	19		2.1	
	Average Daily Concentration (ADC)	1.4	13		1.4	
	Lifetime Average Daily Concentration (LADC)	0.6	6.6		0.6	
	15-min TWA Exposure Concentration	13	117	40	13	
Mold Release Products	8-hr TWA Exposure Concentration	0.1	0.2	4	0.1	High
	Acute Exposure Concentration (AC)	3.3E-02	6.7E-02		3.3E-02	
	Average Daily Concentration (ADC)	2.3E-02	4.6E-02		2.3E-02	
	Lifetime Average Daily Concentration (LADC)	9.1E-03	2.3E-02		9.1E-03	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
Source: (Gold et al. 2008; NIOSH 1980)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

For printing applications, photocopying, and photographic film applications, worker exposure is assessed using PCE personal breathing zone monitoring data from multiple sources with confidence ratings ranging from "medium" to "high", as determined through EPA's systematic review process. EPA

has a medium to high level of confidence in the assessed worker exposure for these uses based on the strength of the monitoring data.

For mold release products, worker exposure is assessed using PCE area monitoring data from a single source with a confidence rating of "high", as determined through EPA's systematic review process. There is some uncertainty in how representative the area samples are of actual exposures. Based on the above information, EPA has a medium confidence in the assessed worker exposure for this use.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.25 Laboratory Chemicals

Worker Activities

Specific worker activities for using laboratory uses were not identified, but EPA expects that workers may be potentially exposed to PCE in laboratories during multiple activities, including unloading of PCE from the containers in which they were received, transferring PCE into laboratory equipment (i.e., beakers, flasks, other intermediate storage containers), dissolving substances into PCE or otherwise preparing samples that contain PCE, analyzing these samples, and discarding the samples.

ONUs for this condition of use include supervisors, managers, and other employees that may be in the laboratory but do not perform tasks that result in the same level of exposures as those workers that engage in tasks related to the use of PCE.

Number of Workers and Occupational Non-Users

EPA did not identify information to estimate the total number of workers exposed to PCE at laboratory facilities. However, EPA estimated the number of workers and ONUs per site using information from the Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015). EPA identified the NAICS code 541380, Testing Laboratories, as the code expected to include laboratory chemical uses of PCE. Based on data from the BLS for this NAICS code and related SOC codes, there are an average of one worker and nine ONUs per site, or a total of ten potentially exposed workers and ONUs per site.

Occupational Inhalation Exposure Results

EPA does not have reasonable available information to assess worker exposures to PCE during laboratory use. However, due to the expected safety practices when using chemicals in a laboratory setting, PCE is expected to be applied in small amounts under a fume hood, thus reducing the potential for inhalation exposures.

2.4.1.26 Waste Handling, Disposal, Treatment, and Recycling

Worker Activities

At waste disposal sites, workers are potentially exposed via dermal contact with waste containing PCE or via inhalation of PCE vapor. Depending on the concentration of PCE in the waste stream, the route and level of exposure may be similar to that associated with container unloading activities. See Section 2.4.1.23 for the assessment of worker exposure from chemical unloading activities.

Number of Workers and Occupational Non-Users

EPA estimated the number of workers and occupational non-users potentially exposed during disposal/treatment of PCE using Bureau of Labor Statistics' OES data (U.S. BLS 2016) and the U.S. Census' SUSB (U. S. Census Bureau 2015) as well as the primary NAICS and SIC code reported by each site in the 2016 TRI or 2016 DMR, respectively. There are approximately 1,600 workers and 700 ONUs potentially exposed during disposal/treatment of PCE wastes (see Table 2-55)

Table 2-55. Estimated Number of Workers Potentially Exposed to PCE During Waste Handling, Disposal, Treatment, and Recycling

Number of Sites	Exposed Workers per Site	Exposed Occupational Non-Users per Site	Total Exposed Workers[a]	Total Exposed Occupational Non-Users[a]	Total Exposed[a]
94	17	7	1,600	700	2,300

[a] Totals have been rounded to two significant figures. Totals may not add exactly due to rounding.

Occupational Inhalation Exposure Results

EPA did not identify any inhalation exposure monitoring data for disposal/treatment. Therefore, EPA assessed inhalation exposures during these uses using the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model, assuming PCE is present at 100 percent concentration when used. Details of the model design and parameters is provided in Appendix E of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d). Table 2-56 summarizes the model results.

The results only include values for workers as the model does not estimate ONU exposures. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-56. Summary of Exposure Modeling Results for Waste Handling, Disposal, Treatment, and Recycling

Exposure Concentration Type	Worker Exposures		Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)		
8-hr TWA Exposure Concentration	8.0E-03	3.6E-02	8.0E-03	N/A – modeled data
Acute Exposure Concentration (AC)	2.7E-03	1.2E-02	2.7E-03	
Average Daily Concentration (ADC)	1.8E-03	8.2E-03	1.8E-03	
Lifetime Average Daily Concentration (LADC)	7.2E-04	4.2E-03	7.2E-04	
30-min TWA Exposure Concentration	0.1	–[b]	0.1	
1-hr TWA Exposure Concentration	–[b]	0.3	–[b]	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] High-end for Acute exposures is calculated as a 1-hr TWA and central tendency is calculated as a 30-min TWA.

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

The Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model is used to estimate worker exposure. The model uses a combination of published EPA emission factors and engineering judgment to estimate central tendency and high-end exposures. EPA believes the model exposures are likely to be representative of exposure associated with bulk container loading. However, the model does not account for other potential sources of exposure at industrial facilities, such as sampling, equipment cleaning, and other process activities. The model also assumes only one container is loaded per day, although larger facilities may have higher product loading frequencies. These model uncertainties could result in an underestimate of the worker exposure. Based on reasonably available information above, EPA has a medium level of confidence in the assessed worker exposure.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.27 Other Department of Defense Uses

EPA reached out to the Department of Defense (DoD) for monitoring data for the first 10 chemical substances that are the subject of the Agency's initial chemical risk evaluations. The DoD provided monitoring data from its Defense Occupational and Environmental Health Readiness System – Industrial Hygiene (DOEHRS-IH), which collects occupational and environmental health risk data from each service branch. The DoD provided inhalation monitoring data for three branches of the military: Army, Air Force, and Navy (U.S. DOD and Environmental Health Readiness System - Industrial 2018). These data are not distinguished among the three branches.

Where the condition of use of the collected monitoring data could be clearly determined and fit into one of the conditions of use assessed in Sections 2.4.1.6 through 2.4.1.26. The following conditions of use include DoD data:

- Aerosol Degreasing;
- Dry Cleaning;
- Adhesives, Sealants, Paints, and Coatings; and
- Chemical Maskants.

This section provides analysis of additional DoD data that did not fit into another previously identified condition of use.

Worker Activities

The DoD data did not provide worker activities for these data.

Number of Workers and Occupational Non-Users

The DoD data did not provide information to estimate the number of workers and ONUs exposed from these uses.

Occupational Inhalation Results

EPA assessed exposures from two processes in the DoD data: oil analysis and water pipe repair. The sample times for other processes in the dataset were less than 50% of an 8-hr shift (assumed shift-time for these activities) and, therefore, may not be representative of actual 8-hr TWA exposures. Therefore, EPA could not estimate exposures for these processes.

Oil Analysis

For the oil analysis process, one data point was available; however, different parameters are used for calculating high-end and central tendency ADC and LADC. Therefore, a high-end and central tendency are presented based on the single data point.

EPA adjusted the exposure frequency when calculating ADC and LADC to reflect the expected number of exposure days based on the process frequency reported by DoD. For the oil analysis the frequency was two to three times per week. EPA used the midpoint of the ranges to estimate the central tendency ADC and LADC and the maximum frequency to calculate the high-end ADC and LADC. This resulted in 150 exposure days/yr at the high-end and 125 exposure days at the central tendency for the oil analysis.

Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-57. Summary of Inhalation Monitoring Data for Other DoD Uses (Oil Analysis) of PCE

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Central Tendency (ppm)	High-End (ppm)			
8-hr TWA Exposure Concentration	0.9[b]		1	0.9	High
Acute Exposure Concentration (AC)	0.3	0.3		0.3	
Average Daily Concentration (ADC)	0.1	0.1		0.1	
Lifetime Average Daily Concentration (LADC)	4.0E-02	6.2E-02		4.0E-02	
15-min TWA Exposure Concentration	4.2		1	4.2	
1-hr TWA Exposure Concentration	6.6		1	6.6	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

[b] Only one data point identified for oil analysis. However, different parameters are used for calculating high-end and central tendency ADC and LADC. Therefore, a high-end and central tendency are presented based on the single data point.

Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018)

Water Pipe Repair

For the water pipe repair, there was only one data point available as well; however, it measured below the LOD. To estimate values below the LOD, EPA referenced the *Guidelines for Statistical Analysis of Occupational Exposure Data* (U.S. EPA 1994b). However, there is only a single data point, so the geometric standard deviation is not statistically meaningful. Therefore, EPA assesses the exposure as ranging from zero to the LOD (2.31 ppm) and presents two scenarios: 1) using the LOD as a "higher value"; and 2) using half the LOD as a "midpoint" value.

EPA adjusted the exposure frequency when calculating ADC and LADC to reflect the expected number of exposure days based on the process frequency reported by DoD. For the water pipe repair the frequency was two to three times per month. EPA used the midpoint of the ranges to estimate the central tendency ADC and LADC and the maximum frequency to calculate the high-end ADC and LADC. This resulted in 36 exposure days/yr at the high-end and 30 exposure days at the central tendency for the water pipe repair.

Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. The results only include values for workers as monitoring data for ONUs were not identified. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs.

Table 2-58. Summary of Inhalation Monitoring Data for Other DoD Uses (Water Pipe Repair) of PCE

Exposure Concentration Type	Worker Exposures		Number of Samples	Occupational Non-User Exposures (ppm)[a]	Data Quality Rating of Air Concentration Data
	Midpoint Value (ppm)	Higher Value (ppm)			
8-hr TWA Exposure Concentration	1.2	2.3	1	1.2	High
Acute Exposure Concentration (AC)	0.4	0.8		0.4	
Average Daily Concentration (ADC)	3.2E-02	7.6E-02		3.2E-02	
Lifetime Average Daily Concentration (LADC)	1.3E-02	3.9E-02		1.3E-02	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.

Source: (U.S. DOD and Environmental Health Readiness System - Industrial 2018)

Strength, Limitation, and Uncertainty of the Inhalation Exposure Assessment

Exposure to workers is assessed using PCE personal breathing zone monitoring data from DoD which has a confidence rating of "high", as determined through EPA's systematic review process. The data is

directly applicable to the use being assessed. For the water pipe repair there is some uncertainty in the assessed values as the measurement was below the LOD. Despite this uncertainty, EPA has a high level of confidence in the assessed worker exposure for these uses based on the strength of the monitoring data.

Exposure to ONUs is assessed using the worker central tendency exposure values. The statistical representativeness of this value for ONUs is unknown; however, the central tendency for ONUs is expected to be lower than that of workers as EPA expects ONUs to be farther from the source of exposure than workers. Therefore, EPA's confidence in the exposure estimate for ONUs is low.

2.4.1.28 Summary of Inhalation Exposure Assessment

The following table summarizes the inhalation exposure estimates for all occupational exposure scenarios. Where statistics can be calculated, the central tendency estimate represents the 50th percentile exposure level of the available data set, and the high-end estimate represents the 95th percentile exposure level.

4474 **Table 2-59. Summary of Inhalation Exposure Results**

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Manufacturing (8-hr TWA)	Worker	2.6	3.3E-02	0.9	1.1E-02	0.6	7.4E-03	0.3	2.9E-03	50th and 95th Percentile	Monitoring Data
Manufacturing (8-hr TWA)	ONU[a]	3.3E-02		1.1E-02		7.4E-03		2.9E-03		Unknown	Worker Central Tendency
Manufacturing (12-hr TWA)	Worker	0.2	2.1E-02	0.1	1.0E-02	7.3E-02	7.0E-03	3.7E-03	2.8E-03	50th and 95th Percentile	Monitoring Data
Manufacturing (12-hr TWA)	ONU[a]	2.1E-02		1.0E-02		7.0E-03		2.8E-03		Unknown	Worker Central Tendency
Repackaging	Worker	0.8	0.4	0.3	0.1	0.2	9.9E-02	9.6E-02	3.9E-02	50th and 95th Percentile	Monitoring Data
Repackaging	ONU[a]	0.4		0.1		9.9E-02		3.9E-02		Unknown	Worker Central Tendency
Processing as Reactant/ Intermediate (8-hr TWA)	Worker	2.6	3.3E-02	0.9	1.1E-02	0.6	7.4E-03	0.3	2.9E-03	50th and 95th Percentile	Monitoring Data
Processing as Reactant/ Intermediate (8-hr TWA)	ONU[a]	3.3E-02		1.1E-02		7.4E-03		2.9E-03		Unknown	Worker Central Tendency
Processing as Reactant/ Intermediate (12-hr TWA)	Worker	0.2	2.1E-02	0.1	1.0E-02	7.3E-02	7.0E-03	3.7E-03	2.8E-03	50th and 95th Percentile	Monitoring Data
Processing as Reactant/ Intermediate (12-hr TWA)	ONU[a]	2.1E-02		1.0E-02		7.0E-03		2.8E-03		Unknown	Worker Central Tendency
Incorporation into Formulation - Aerosol Packing	Worker	13	8.3	4.4	2.8	3.0	1.9	1.5	0.8	Median and Maximum	Monitoring Data

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Incorporation into Formulation - Aerosol Packing	ONU[a]	8.3		2.8		1.9		0.8		Unknown	Worker Central Tendency
Incorporation into Formulation - Degreasing Solvent	Worker	2.6	0.7	0.4	0.1	5.7E-02	1.6E-02	8.4E-03	2.3E-03	50th and 95th Percentile	Model (probabilistic)
Incorporation into Formulation - Degreasing Solvent	ONU[a]	0.7		0.1		1.6E-02		2.3E-03		Unknown	Worker Central Tendency
Incorporation into Formulation - Dry Cleaning Solvent	Worker	14	4.0	2.1	0.6	0.3	8.6E-02	4.5E-02	1.3E-02	50th and 95th Percentile	Model (probabilistic)
Incorporation into Formulation - Dry Cleaning Solvent	ONU[a]	4.0		0.6		8.6E-02		1.3E-02		Unknown	Worker Central Tendency
Incorporation into Formulation - Miscellaneous	Worker	1.4	0.4	0.2	5.9E-02	3.1E-02	8.6E-03	4.5E-03	1.3E-03	50th and 95th Percentile	Model (probabilistic)
Incorporation into Formulation - Miscellaneous	ONUa	0.4		5.9E-02		8.6E-03		1.3E-03		Unknown	Worker Central Tendency

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
OTVD	Worker	32	2.1	11	0.7	7.3	0.5	3.8	0.2	50th and 95th Percentile	Monitoring Data
OTVD	ONU	5.2	0.6	1.7	0.2	1.2	0.1	0.6	5.5E-02	50th and 95th Percentile	Monitoring Data
Closed Loop Vapor Degreasing	Worker	0.3	7.2E-02	8.4E-02	2.4E-02	5.8E-02	1.6E-02	3.0E-02	6.6E-03	50th and 95th Percentile	Monitoring Data
Closed Loop Vapor Degreasing	ONU	0.1	6.5E-02	3.2E-02	2.2E-02	2.2E-02	1.5E-02	1.1E-02	5.9E-03	Median and Maximum	Monitoring Data
Conveyorized Vapor Degreasing	Worker	186	78	62	26	42	18	17	6.7	50th and 95th Percentile	Model (probabilistic)
Conveyorized Vapor Degreasing	ONU	126	41	42	14	29	9.3	12	3.5	50th and 95th Percentile	Model (probabilistic)
Web Degreasing	Worker	1.8	0.6	0.6	0.2	0.4	0.1	0.2	5.3E-02	50th and 95th Percentile	Model (probabilistic)
Web Degreasing	ONU	1.3	0.3	0.4	0.1	0.3	7.3E-02	0.1	2.7E-02	50th and 95th Percentile	Model (probabilistic)
Cold Cleaning	Worker	4.1	1.4	1.4	0.5	0.9	0.3	0.5	0.1	50th and 95th Percentile	Monitoring Data
Cold Cleaning	Worker	1.5	2.4E-03	0.5	8.0E-04	0.4	5.5E-04	0.1	2.0E-04	50th and 95th Percentile	Model (probabilistic)
Cold Cleaning	ONU	0.8	1.2E-03	0.3	4.1E-04	0.2	2.8E-04	6.7E-02	1.1E-04	50th and 95th Percentile	Model (probabilistic)

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Aerosol Degreasing/ Lubricants	Worker	7.8	1.4	2.6	0.5	1.8	0.3	0.9	0.1	50th and 95th Percentile	Monitoring Data
Aerosol Degreasing/ Lubricants	Worker	17	5.5	5.7	1.8	3.9	1.3	1.6	0.5	50th and 95th Percentile	Model (probabilistic)
Aerosol Degreasing/ Lubricants	ONU	0.7	0.1	0.2	3.4E-02	0.2	2.0E-02	7.0E-02	1.0E-02	50th and 95th Percentile	Model (probabilistic)
Post-2006 NESHAP Dry Cleaning	Worker	20	3.6	6.5	1.2	5.2	0.9	2.7	0.3	50th and 95th Percentile	Monitoring Data
Post-2006 NESHAP Dry Cleaning	ONU	0.3	0.3	0.1	0.1	9.3E-02	8.2E-02	4.8E-02	3.3E-02	N/A (one data point)	Monitoring Data
4th/5th Gen Only Dry Cleaning	Worker	5.6	1.0	1.9	0.3	1.5	0.2	0.8	9.2E-02	50th and 95th Percentile	Monitoring Data
4th/5th Gen Only Dry Cleaning	ONU	0.1	1.4E-02	4.1E-02	4.7E-03	3.3E-02	3.3E-03	1.7E-02	1.3E-03	Median and Maximum	Monitoring Data
Dry Cleaning (12-hr TWA)	Worker	30	1.4	15	0.7	10	0.5	4.1	0.2	50th and 95th Percentile	Model (probabilistic)
Dry Cleaning (12-hr TWA)	ONU	1.5	0.1	0.8	5.4E-02	0.6	3.8E-02	0.2	1.4E-02	50th and 95th Percentile	Model (probabilistic)
Paints/Coatings	Worker	4.6	0.2	1.5	7.8E-02	1.0	5.3E-02	0.5	2.1E-02	50th and 95th Percentile	Monitoring Data
Paints/Coatings	ONU[a]	0.2		7.8E-02		5.3E-02		2.1E-02		Unknown	Worker Central Tendency

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Adhesives	Worker	0.8	8.8E-02	0.3	2.9E-02	0.2	2.0E-02	9.5E-02	8.0E-03	Arithmetic Mean and Maximum	Monitoring Data
Adhesives	ONU[a]	8.8E-02		2.9E-02		2.0E-02		8.0E-03		Unknown	Worker Central Tendency
Chemical Maskant	Worker	2.1	1.2	0.7	0.4	0.5	0.3	0.2	0.1	50th and 95th Percentile	Monitoring Data
Chemical Maskant	ONU[a]	1.2		0.4		0.3		0.1		Unknown	Worker Central Tendency
Industrial Processing Aid	Worker	1.2	6.0E-02	0.4	2.0E-02	0.3	1.4E-02	0.1	5.4E-03	50th and 95th Percentile	Monitoring Data
Industrial Processing Aid	ONU[a]	6.0E-02		2.0E-02		1.4E-02		5.4E-03		Unknown	Worker Central Tendency
Other Industrial Uses	Worker	3.6E-02	8.0E-03	1.2E-02	2.7E-03	8.2E-03	1.8E-03	4.2E-03	7.2E-04	N/A – CT and HE[b]	Model (deterministic)
Other Industrial Uses	ONU[a]	8.0E-03		2.7E-03		1.8E-03		7.2E-04		Unknown	Worker Central Tendency
Metalworking Fluid	Worker	2.1E-02	5.8E-03	7.0E-03	1.9E-03	4.8E-03	1.3E-03	2.5E-03	5.2E-04	Geometric mean and 90th percentile	ESD
Metalworking Fluid	ONU[a]	5.8E-03		1.9E-03		1.3E-03		5.2E-04		Unknown	Worker Central Tendency
Wipe Cleaning and Metal/Stone Polishes	Worker	228	132	76	44	52	30	27	12	Median and Maximum	Monitoring Data
Wipe Cleaning and Metal/Stone Polishes	ONU	23	2.2E-02	7.7	7.3E-03	5.3	5.0E-03	2.7	2.0E-03	50th and 95th Percentile	Monitoring Data

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	Worker	0.2	0.2	7.7E-02	5.7E-02	5.3E-02	3.9E-02	2.7E-02	1.6E-02	Median and Maximum	Monitoring Data
Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	ONU	3.0E-02[c]		1.0E-02	1.0E-02	6.8E-03	6.8E-03	3.5E-03	2.7E-03	N/A (one data point)	Monitoring Data
Other Commercial Uses - Printing	Worker	5.9	1.9	2.0	0.6	1.4	0.4	0.7	0.2	50th and 95th Percentile	Monitoring Data
Other Commercial Uses - Printing	ONU[a]	1.9		0.6		0.4		0.2		Unknown	Worker Central Tendency
Other Commercial Uses - Photocopying	Worker	5.0E-04	1.9E-04	1.7E-04	6.3E-05	1.1E-04	4.3E-05	5.9E-05	1.7E-05	Median and Maximum	Monitoring Data
Other Commercial Uses - Photocopying	ONU[a]	1.9E-04		6.3E-05		4.3E-05		1.7E-05		Unknown	Worker Central Tendency
Other Commercial Uses - Photographic Film	Worker	56	6.3	19	2.1	13	1.4	6.6	0.6	50th and 95th Percentile	Monitoring Data

Condition of Use	Category	8- or 12-Hour TWA Exposures (ppm)		AC (ppm)		ADC (ppm)		LADC (ppm)		Statistical Value for Central Tendency and High-End	Data Type
		High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency	High-End	Central Tendency		
Other Commercial Uses - Photographic Film	ONU[a]	6.3		2.1		1.4		0.6		Unknown	Worker Central Tendency
Other Commercial Uses - Mold Release	Worker	0.2	0.1	6.7E-02	3.3E-02	4.6E-02	2.3E-02	2.3E-02	9.1E-03	Arithmetic Mean and Maximum	Monitoring Data
Other Commercial Uses - Mold Release	ONU[a]	0.1		3.3E-02		2.3E-02		9.1E-03		Unknown	Worker Central Tendency
Other DOD Uses - Water Pipe Repair	Worker	2.3	1.2	0.8	0.4	7.6E-02	3.2E-02	3.9E-02	1.3E-02	Half the LOD and the LOD	Monitoring Data
Other DOD Uses - Water Pipe Repair	ONU[a]	1.2		0.4		3.2E-02		1.3E-02		Unknown	Worker Central Tendency
Other DOD Uses - Oil analysis	Worker	0.9[c]		0.3	0.3	0.1	0.1	6.2E-02	4.0E-02	N/A (one data point)	Monitoring Data
Other DOD Uses - Oil analysis	ONU[a]	0.9		0.3		0.1		4.0E-02		Unknown	Worker Central Tendency
Disposal/ Recycling	Worker	3.6E-02	8.0E-03	1.2E-02	2.7E-03	8.2E-03	1.8E-03	4.2E-03	7.2E-04	N/A – CT and HE[b]	Model (deterministic)
Disposal/ Recycling	ONU[a]	8.0E-03		2.7E-03		1.8E-03		7.2E-04		Unknown	Worker Central Tendency

[a] EPA did not identify monitoring data or models to estimate exposures for ONUs. In lieu of ONU-specific data, EPA uses worker central tendency exposure results as a surrogate to estimate exposures for ONUs. The statistical representativeness of this value for ONUs is unknown.
[b] Based on distinct model scenarios that are likely representative of central tendency (CT) and high-end (HE) exposures.
[c] Only a single data point was available for this condition of use.

2.4.1.29 Dermal Exposure Assessment

Dermal absorption of PCE depends on the type and duration of exposure. Where exposure is non-occluded, only a fraction of PCE that comes into contact with the skin will be absorbed as the chemical readily evaporates from the skin. However, dermal exposure may be significant in cases of occluded exposure, repeated contacts, or dermal immersion. For example, work activities with a high degree of splash potential may result in PCE liquids trapped inside the gloves, inhibiting the evaporation of PCE and increasing the exposure duration.

To assess exposure, EPA used the *Dermal Exposure to Volatile Liquids Model* (see following equation and Appendix K of the *Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene (Ethene, 1,1,2,2,-Tetrachloro) CASRN: 127-18-4* (Supplemental Engineering Report) (U.S. EPA 2020d)) to calculate the dermal retained dose. The equation modifies *EPA/OPPT 2-Hand Dermal Exposure to Liquids Model* (peer reviewed) by incorporating a "fraction absorbed (f_{abs})" parameter to account for the evaporation of volatile chemicals and a "protection factor (PF)" to account for glove use:

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

Where:

D_{exp} is the dermal retained dose (mg/kg-day)
S is the surface area of contact (cm^2)
Q_u is the quantity remaining on the skin after an exposure event (mg/cm^2-event)
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)
f_{abs} is the fraction of applied mass that is absorbed (Default for PCE: 0.13 for industrial facilities and 0.19 for commercial facilities[13])
PF is the glove protection factor (Default: see Table 2-60)
BW is the body weight (Default: 80 kg)

Default glove PF values, which vary depending on the type of glove used and the presence of employee training program, are shown in Table 2-60.

Table 2-60. Glove Protection Factors for Different Dermal Protection Strategies

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 *b* 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

[13] The absorbed fraction (f_{abs}) is a function of indoor air speed, which differs for industrial and commercial settings.

Source: (Marquart et al. 2017)

Table 2-61 presents the estimated dermal acute retained dose for *workers* in various exposure scenarios, including what-if scenarios for glove use. The dose estimates assume one exposure event (applied dose) per work day and that 13 to 19 percent of the applied dose is absorbed through the skin. The exposure estimates are provided for each condition of use, where the conditions of uses are "binned" based on the maximum possible exposure concentration (Y_{derm}) and the likely level of exposure. The exposure concentration is determined based on EPA's review of currently available products and formulations containing PCE:

- **Bin 1:** Bin 1 covers industrial uses that generally occur in closed systems. For these uses, dermal exposure is likely limited to chemical loading/unloading activities (e.g. connecting hoses) and taking quality control samples.
- **Bin 2:** Bin 2 covers industrial degreasing and chemical maskant uses, which are not closed systems. For these uses, there is greater opportunity for dermal exposure during activities such as charging and draining degreasing/milling equipment, drumming waste solvent, handling recycled/re-captured maskants, and removing waste sludge.
- **Bin 3:** Bin 3 covers aerosol uses, where workers are likely to have direct dermal contact with film applied to substrate and incidental deposition of aerosol to skin.
- **Bin 4:** Bin 4 covers commercial activities of similar maximum concentration. Most of these uses are uses at dry cleaners, and/or uses expected to have direct dermal contact with bulk liquids. At dry cleaning shops, workers may be exposed to bulk liquids while charging and draining solvent to/from machines, removing and disposing sludge, and maintaining equipment. Workers can also be exposed to PCE used in spot cleaning products at the same shop.
- **Bin 5:** Bin 5 covers uses of metalworking fluids containing PCE. These product formulations are expected to be used in industrial settings and workers may be exposed when unloading the metalworking fluid from containers; transferring fluids to the trough; and performing metal shaping operations.
- **Bin 6:** Bin 6 covers uses of adhesives, sealants, paints, and coatings containing PCE. These product formulations may have both industrial and commercial uses and workers may be exposed when mixing coating/adhesive, charging products to application equipment (e.g., spray guns, roll applicators, etc.), and cleaning application equipment. Other workers may also have incidental contact with applied products during subsequent fabrication steps.

Dermal exposure to liquid is not expected for occupational non-users, as they do not directly handle PCE.

Strength, Limitation, and Uncertainty of the Dermal Exposure Assessment

Dermal exposures are assessed using *the Dermal Exposure to Volatile Liquids Model*, which relies on the theoretical framework presented by Kasting and Miller (2006) to estimate the fractional absorption in accounting for chemical volatilization. EPA has a medium level of confidence in the assessed baseline exposure. Glove protection factors are presented as what-if scenarios to show the potential effect of glove use on exposure levels. EPA does not know the actual frequency, type, and effectiveness of glove use in specific workplaces with PCE conditions of use.

Table 2-61. Estimated Dermal Acute Retained Dose for Workers in All Conditions of Use

Exposure Scenario	Bin	Max Y_{derm}	Dermal Exposure (mg/kg-day)			
			No Gloves (PF = 1)	Protective Gloves (PF = 5)	Protective Gloves (PF = 10)	Protective Gloves (Industrial uses, PF = 20)
Manufacture	Bin 1	1.0	1.2 (CT) 3.5(HE)	0.2 (CT) 0.7 (HE)	0.1 (CT) 0.4 (HE)	5.9E-02 (CT) 0.2 (HE)
Import/Repackaging						
Processing as a Reactant						
Incorporation into Formulation, Mixture, or Reaction Product						
Industrial Processing Aid						
Other Industrial Uses						
Waste Handling, Disposal, Treatment, and Recycling						
Batch Open-Top Vapor Degreasing	Bin 2	1.0	1.2(CT) 3.5 (HE)	0.2 (CT) 0.7 (HE)	0.1 (CT) 0.4 (HE)	5.9E-02 (CT) 0.2 (HE)
Batch Closed-Loop Vapor Degreasing						
Conveyorized Vapor Degreasing						
Web Degreasing						
Cold Cleaning						
Maskant for Chemical Milling						
Aerosol Degreasing and Aerosol Lubricants	Bin 3	1.0	1.8 (CT) 5.3 (HE)	0.4 (CT) 1.1 (HE)	0.2 (CT) 0.5 (HE)	N/A

Exposure Scenario	Bin	Max Y_{derm}	Dermal Exposure (mg/kg-day)			
Dry Cleaning and Spot Cleaning	Bin 4	1.0	1.8 (CT) 5.4 (HE)	0.4 (CT) 1.1 (HE)	0.2 (CT) 0.5 (HE)	N/A
Wipe Cleaning and Metal/Stone Polishes						
Other Spot Cleaning/Spot Remover						
Other Commercial Uses						
Metalworking Fluids	Bin 5	0.10	0.1 (CT) 0.4 (HE)	2.5E-02 (CT) 7.1E-02 (HE)	1.2E-02 (CT) 3.5E-02 (HE)	5.9E-03 (CT) 1.8E-02 (HE)
Adhesives, Sealants, Paints, and Coatings (Industrial)	Bin 6	0.80	0.9 (CT) 2.8 (HE)	0.2 (CT) 0.6 (HE)	9.4E-02 (CT) 0.3 (HE)	4.7E-02 (CT) 0.1 (HE)
Adhesives, Sealants, Paints, and Coatings (Commercial)		0.80	1.4 (CT) 4.3 (HE)	0.3 (CT) 0.9 (HE)	0.1 (CT) 0.4 (HE)	N/A

CT = Central Tendency; HE = High-End

2.4.1.30 Key Assumptions and Uncertainties of the Occupational Exposure Assessment

EPA addressed variability in models by identifying key model parameters to apply a statistical distribution that mathematically defines the parameter's variability. EPA defined statistical distributions for parameters using documented statistical variations where available. Where the statistical variation is not known, assumptions are made to estimate the parameter distribution using available literature data. See the *Draft Risk Evaluation for Perchloroethylene Supplemental Information: Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene* (U.S. EPA 2019a) for statistical distribution for each model input parameter. The following sections discuss uncertainties in the occupational exposure assessment.

Number of Workers

There are a number of uncertainties surrounding the estimated number of workers potentially exposed to PCE, as outlined below. Most are unlikely to result in a systematic underestimate or overestimate but could result in an inaccurate estimate.

CDR data are used to estimate the number of workers associated with manufacturing. There are inherent limitations to the use of CDR data as they are reported by manufacturers and importers of PCE. Manufacturers and importers are only required to report if they manufactured or imported PCE in excess of 25,000 pounds at a single site during any calendar from 2012 to 2015; as such, CDR may not capture all sites and workers associated with any given chemical. Second, the estimate is based on information that is known or reasonably ascertainable to the submitter. CDR submitters (chemical manufacturers and importers) do not always have accurate information on the number of potentially exposed workers at downstream processing sites.

There are also uncertainties with BLS data, which are used to estimate the number of workers for the remaining conditions of use. First, BLS' OES employment data for each industry/occupation combination are only available at the 3-, 4-, or 5-digit NAICS level, rather than the full 6-digit NAICS level. This lack of granularity could result in an overestimate of the number of exposed workers if some 6-digit NAICS are included in the less granular BLS estimates but are not, in reality, likely to use PCE for the assessed conditions of use. EPA addressed this issue by refining the OES estimates using total employment data from the U.S. Census' SUSB. However, this approach assumes that the distribution of occupation types (SOC codes) in each 6-digit NAICS is equal to the distribution of occupation types at the parent 5-digit NAICS level. If the distribution of workers in occupations with PCE exposure differs from the overall distribution of workers in each NAICS, then this approach will result in inaccuracy.

Second, EPA's judgments about which industries (represented by NAICS codes) and occupations (represented by SOC codes) are associated with the uses assessed in this report are based on EPA's understanding of how PCE is used in each industry. Designations of which industries and occupations have potential exposures is nevertheless subjective, and some industries/occupations 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 count of exposed workers**.**

Analysis of Exposure Monitoring Data

To analyze the exposure data, EPA categorized individual PBZ data points as either "worker" or "occupational non-user". The categorizations are based on descriptions of worker job activity as provided in literature and EPA's judgment. In general, samples for employees that are expected to have the highest exposure from direct handling of PCE are categorized as "worker" and samples for employees that are expected to have lower exposure and do not directly handle PCE are categorized as "occupational non-user".

Exposures for occupational non-users can vary substantially. Most data sources do not sufficiently describe the proximity of these employees to the PCE 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.

Some data sources may have a bias. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. Similarly, OSHA Chemical Exposure Health Data (CEHD) are obtained from OSHA inspections, which may be the result of worker complaints, and may provide exposure results that are generally more conservative than the industry average.

Some scenarios have limited exposure monitoring data in literature, if any. Where few data are available, the assessed exposure levels are unlikely to be representative of worker exposure across the entire job category or industry. In addition, exposure data for compliance safety and health officers may not be representative of typical exposure levels for occupational non-users.

In cases where there was no exposure monitoring data, EPA attempted to identify monitoring data from similar conditions of use as surrogate. While these conditions of use have similar worker activities contributing to exposures, it is unknown if the results will be fully representative of worker exposure across different conditions of use.

Where the sample data set contains six or more data points, the 50^{th} and 95^{th} percentile exposure concentrations were calculated from the sample to represent central tendency and high-end exposure levels. using available data. The underlying distribution of the data, and the representativeness of the available data, are not known. Where discrete data was not available, EPA used reported statistics (i.e., median, mean, 90^{th} percentile, etc.). Since EPA could not verify these values, there is an added level of uncertainty.

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 zone. 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.
- For certain PCE applications (e.g. vapor degreasing and cold cleaning), PCE 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 PCE condition of use. The models have not been regressed or fitted with monitoring data.

Each subsequent section below discusses uncertainties associated with the individual model.

Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model

For the other industrial uses and waste handling, disposal, treatment, and recycling conditions of use, the *Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model* is used to estimate the airborne concentration associated with generic chemical loading scenarios at industrial facilities. Specific uncertainties associated with this model are described below:

- After each loading event, the model assumes saturated air containing PCE that remains in the transfer hose and/or loading arm is released to air. The model calculates the quantity of saturated air using design dimensions of loading systems published in the OPW Engineered Systems catalog and engineering judgment. These dimensions may not be representative of the whole range of loading equipment used at industrial facilities handling PCE.
- The model estimates fugitive emissions from equipment leaks using total organic compound emission factors from EPA's *Protocol for Equipment Leak Emission Estimates* (U.S. EPA 1995), and engineering judgement on the likely equipment type used for transfer (e.g. number of valves, seals, lines, and connections). The applicability of these emission factors to PCE, and the accuracy of EPA's assumption on equipment type are not known.
- The model assumes the use of a vapor balance system to minimize fugitive emissions. Although most industrial facilities are likely to use a vapor balance system when

loading/unloading volatile chemicals, EPA does not know whether these systems are used by all facilities that potentially handle PCE.

Vapor Degreasing and Cold Cleaning Models

The conveyorized vapor degreasing, web degreasing, and cold cleaning assessments use a near-field/far-field approach to model worker exposure. In addition to the uncertainties described above, the vapor degreasing and cold cleaning 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/cold cleaning 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/cold cleaning 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 PCE in air.

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 applications involving PCE.
- The CARB study (CARB 2000) presented 13 different aerosol degreasing formulations containing PCE. For each Monte Carlo iteration, the model determines the PCE concentration in product by selecting one of 13 possible formulations, assuming the distribution for each formulation is equal to that found in a survey of brake cleaning shops in California. It is uncertain if this distribution is representative of other geographic locations within the U.S.
- Some of the aerosol formulations presented in the CARB study (CARB 2000) were provided as ranges. For each Monte Carlo iteration the model selects a PCE concentration within the range of concentrations using a uniform distribution. In reality, the PCE concentration in the formulation may be more consistent than the range provided.

Dry Cleaning Model

The multi-zone dry cleaning model also uses a near-field/far-field approach. Specific uncertainties associated with the dry cleaning scenario are presented below (see also Section 2.4.1.16):

- The model assumes each facility only has one dry cleaning machine, cleaning one to fourteen loads of garments per day. The number of machines is based on the 2010 King

County, WA survey (Whittaker and Johanson 2011) where 96 percent of 151 respondents reported having only one machine at their facility. It is uncertain if this distribution is representative of other geographic locations in the U.S. Larger facilities are likely to have more machines, which could result in additional PCE exposures.

- The model conservatively uses a hemispherical volume based on the dry cleaning machine door diameter as the near-field for machine unloading. The small near-field volume results in a large spike in concentration when the machine door is opened, where any residual PCE solvent is assumed to be instantaneously released into the near-field. In reality, the residual solvent will likely be released continuously over a period of time. In addition, the worker may move around while unloading the garments, such that the worker's breathing zone will not always be next to the machine door throughout the duration of this activity. Therefore, these assumptions may result in an overestimate of worker exposure during machine unloading.
- Many of the model input parameters were obtained from von Grote (2003), which is a German study. Aspects of the U.S. dry cleaning facilities may differ from German facilities. However, it is not known whether the use of German data will under- or over-estimate exposure.
- The model does not cover all potential worker activities at dry cleaners. For example, workers could be exposed to PCE emitted due to equipment leaks, when re-filling PCE solvent into dry cleaning machines, when interrupting a dry cleaning cycle, or when performing maintenance activities (e.g., cleaning lint and button traps, raking out the still, changing solvent filter, and handling solvent waste) (OSHA 2005). However, there is a lack of information on these activities in the literature, and the frequency of these activities is not well understood. The likelihood of equipment leaks is dependent on whether the machines are properly maintained. The frequency of solvent re-filling depends on a specific dry cleaner's workload and solvent consumption rate, which is also affected by the presence of leaks. Based on observations reported by NIOSH (2010) and Blando (2010), solvent charging is not performed every day. EPA was unable to develop a modeling approach for these exposure activities due to the lack of available information.

Modeled Dermal Exposures

The *Dermal Exposure to Volatile Liquids Model* used to estimate dermal exposure to PCE in occupational settings. The model assumes a fixed fractional absorption of the applied dose; however, fractional absorption may be dependent on skin loading conditions. The model also assumes a single exposure event per day based on existing framework of the *EPA/OPPT 2-Hand Dermal Exposure to Liquids Model* and does not address variability in exposure duration and frequency.

2.4.2 Consumer Exposures

EPA evaluated PCE exposure resulting from the use of relevant consumer products and consumer articles. EPA gathered and evaluated consumer exposure information according to the process described in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA 2018b). PCE concentrations measured in residential air or personal breathing zone samples are reported in Section 2.4.2.1. Monitoring and/or controlled laboratory data were available for a

limited number of consumer use scenarios. To fill data gaps, EPA utilized a modeling approach to estimate PCE exposure via use of consumer products and articles (Section 2.4.2.3 and Section 2.4.2.4, respectively).

2.4.2.1 Overview and Literature Summary

Concentrations of volatile organic compounds, such as PCE, are often higher in indoor air than outdoor air due to their presence in consumer products and articles (Lehmann et al. 2002; Fishbein 1992; Thomas et al. 1991). In developed counties, people generally spend 90% of their time indoors (de Blas et al. 2012; Fishbein 1992), and indoor air quality can be greatly compromised due to volatile emissions from cleaning agents, dry cleaned clothes, adhesives, paints and other commercial and consumer products (Canada 2017; de Blas et al. 2012; D'Souza et al. 2009; Lehmann et al. 2002; Thomas et al. 1991).

Systematic review was conducted to identify consumer specific exposure data for PCE containing products and articles (data evaluation tables are available in the Draft Risk Evaluation for PCE Systematic Review Supplemental File Data Quality Evaluation of Consumer Exposure Studies). The literature review returned limited information about chemical-specific consumer monitoring. Most results from the systematic review pertained to indoor air and personal breathing zone concentrations of PCE in residential and consumer settings. Monitoring sites included the United States, Canada, Mexico, Sweden, Finland, Estonia, Lithuania, Belgium, United Kingdom, France, Austria, Germany, Poland, Slovakia, Czech Republic, Hungary, Romania, Bulgaria, Serbia, Bosnia and Herzegovina, Italy, Portugal, Malta, Greece, Cyprus, Albania, Netherlands, China, Japan, Saudi Arabia and Hong Kong.

EPA identified 19 acceptable studies from the United States and Canada deemed to be in the scope of this risk assessment, which monitored residential or commercial indoor air for PCE concentrations, for a total of 3172 measured samples. Identified studies were conducted between the years 1980 and 2013. The detection frequency of PCE in the identified studies ranged from 30% to 100% detection, with a median of 95% detection (with 4 studies not reporting detection frequency). Measured PCE concentrations in indoor air ranged from non-detects (detection limits varied) 94985 ug/m^3, with reported central tendency (mean) values ranging from 0.2 ug/m^3 to 58348 ug/m^3. The maximum air concentration of PCE was measured in a do-it-yourself laundry facility with coin-operated dry cleaning machines (Howie 1981). Full data extraction details for residential indoor air samples conducted in schools and commercial establishments in the US and Canada is provided in the Draft Risk Evaluation for PCE Data Extraction for Consumer and Aquatic Exposure Monitoring Studies.

Of the identified studies, 11 pertained to air concentrations of PCE limited to residential homes in the United States and Canada (Table 2-61). Residential indoor air monitoring studies were conducted between 1986 and 2010, with roughly 1,900 samples collected across eleven US states (CA, CO, IL, IN, MA, MI, MN, NJ, NY, OH, and TX) and Canada (exact location not reported). Concentrations ranged from non-detect (limits varied) to 171 µg/m^3. The highest concentration was from the Canadian study (Chan et al. 1990), which sampled air concentration in Canadian residences. The next highest concentration was 78 µg/m^3, collected from inner-city homes in New York, New York (Sax et al. 2004). Maximum concentrations of approximately 30 µg/m^3 were detected in garages in Boston, Massachusetts (Dodson et al. 2008) and in living areas of

industrial, urban, and suburban homes in Michigan (Jia et al. 2008a). All other maximum reported concentrations were less than 14 µg/m^3. Measures of central tendency (average or median) across all datasets were less than 7 µg/m^3, except for the Canadian study at 28.1 µg/m^3.

4836
4837 **Table 2-62**. Residential Indoor Air Concentrations (µg/m^3) of PCE in the United States and Canada

Study Info	Site Description	Detection Limit	Min.	Mean	GM	Median	Max.	Variance	Data Quality Rating
(Chin et al. 2014); US, 2009-2010 (n = 126; DF = 0.91)	Detroit, MI area; Homes (n=126) with asthmatic children, sampled in living rooms and bedroom	0.091	ND	0.71	--	0.26	13.7	1.66 (SD)	High
(Batterman et al. 2007); US, 2005 (n = 15; DF = 0.73)	Southeast MI; Homes (n = 15) sampled in various locations in the home (upstairs, downstairs)	0.069	--	0.6	--	--	4.4	1.2 (SD)	High
(Batterman et al. 2007); US, 2005 (n = 15; DF = 0.33)	Southeast MI; Garages of residences (n = 15)	0.069	--	0.3	--	--	1.6	0.5 (SD)	High
(Jia et al. 2008a); US, 2004-2005 (n = 252; DF = 0.99)	Ann Arbor, Ypsilanti, and Dearborn MI; Homes (n=159) in industrial, urban, and suburban cities over two seasons	0.02	ND	0.93	--	0.39	27.84	--	Medium
(Dodson et al. 2008)[a]; US, 2004-2005 (n = 16; DF = 0.81)	Boston, MA; Garage of residences	0.07-0.17	ND	2.8	--	0.3	31 (95th)	7.8 (SD)	High
(Dodson et al. 2008)[a]; US, 2004-2005 (n = 10; DF = 0.9)	Boston, MA; Apartment hallway of residences	0.07-0.17	ND	1.9	--	0.8	11 (95th)	3.4 (SD)	High
(Dodson et al. 2008)[a]; US, 2004-2005 (n = 52; DF = 0.98)	Boston, MA; Basement of residences	0.07-0.17	ND	1.7	--	0.5	1.7 (95th)	0.92 (SD)	High
(Dodson et al. 2008)[a]; US, 2004-2005 (n = 83; DF = 0.92)	Boston, MA; Interior room of residences	0.07-0.17	ND	1.9	--	0.6	8.6 (95th)	3.1 (SD)	High
(Adgate et al. 2004); US, 2000 (n = 113; DF = 0.949)	Minneapolis, MN in spring; Sampling from room where child spent the most time.	--	ND (10^{th} 0.02)	--	--	0.4	1 (90th)	--	Medium
(Adgate et al. 2004); US, 2000 (n=113; DF = 0.98)	Minneapolis, MN in winter; Sampling from room where child spent the most time.	--	ND (10^{th} 0.02)	--	--	0.5	1.3 (90th)	--	Medium
(Sax et al. 2004); US, 2000 (n = 32; DF = 1)	Los Angeles, CA in fall; Homes in inner-city neighborhood	0.15	0.6	1.8	--	1.3	6.8	1.4 (SD)	High

Study Info	Site Description	Detection Limit	Min.	Mean	GM	Median	Max.	Variance	Data Quality Rating
(Sax et al. 2004); US, 2000 (n = 40; DF = 1)	Los Angeles, CA in winter; Homes in inner-city neighborhood	0.15	0.7	2.3	--	1.9	11	1.9 (SD)	High
(Sax et al. 2004); US, 1999 (n = 30; DF = 0.78)	New York, NY in summer; Homes in inner-city neighborhood.	0.15	ND	5.3	--	2	43	8.7 (SD)	High
(Sax et al. 2004); US, 1999 (n = 36; DF = 1)	New York, NY in winter; Homes in inner-city neighborhood.	0.15	0.8	6.7	--	3.5	78	13.1 (SD)	High
(Clayton et al. 1999); US, 1995-1997 (n = 402; DF = 0.571)	IL, IN, OH, MI, MN, WI (Great Lakes Region); Non-institutionalized persons	--	ND	5.82	--	1.89	6.83 (90th)	--	High
(Su et al. 2013)[b]; US, 1999-2001 (n = 539; DF = NR)	Elizabeth, NJ; Houston, TX; and Los Angeles, CA; Non-smoking households (n=310)	0.21	--	1.85	--	0.82	6.03 (95th)	4.53 (SD)	Medium
(Van Winkle and Scheff 2001); US, 1994-1995 (n = 48; DF = 1)	Southeast Chicago, IL; Urban homes (n=10) sampled over a 10-month period from the kitchen in the breathing zone.	--	0.54	2.61	--	2.17	4.74 (90th)	2.15 (SD)	High
(Lindstrom et al. 1995); US, 1994 (n = 9; DF = 0.89)	Denver, CO; Homes, occupied (n=9)	0.14	ND	0.66	--	0.33	1.99	--	Medium
(Chan et al. 1990); CA, 1987 (n = 6; DF = 1)	Homes (n=6), main floor	--	2	6.2	--	--	18	--	Medium
(Chan et al. 1990); CA, 1986 (n = 12; DF = 1)	Homes (n=12), main floor	--	1	28.1	--	--	171	--	Medium

Study Info: The information provided includes the HERO ID and citation; country and year samples collected; number of samples and detection frequency.
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. DF = detection frequency. NR = Not reported. US = United States. CA = Canada
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] Samples from this study (Su et al. 2013) were collected as part of the RIOPA study.

EPA identified 20 acceptable studies conducted outside of North America (Mexico, and the previously listed countries in Europe, Asia and the Middle East), for a total of 4369 measured samples. Identified studies were conducted between the years 1981 and 2015. The detection frequency of PCE in the identified foreign studies ranged from 30% to 100% detection, with a median of 100% detection (with 12 studies not reporting detection frequency). Measured PCE concentrations in indoor air ranged from non-detects (detection limits varied) to $9.63x10^4$ ug/m^3, with reported central tendency (mean) values ranging from 0.46 ug/m^3 to $4.95x10^3$ ug/m^3. The maximum air concentration of $9.63x10^4$ ug/m^3 was measured near a photocopy shop (Kiurski et al. 2016). The next highest reported concentration was $2.48x10^4$ ug/m^3 in a vehicle exposed to dry cleaned articles (Gulyas and Hemmerling 1990). The highest PCE concentration measured in residential air was 245 ug/m^3 measured in urban homes in Paris, France (Roda et al. 2013). Full data extraction details for indoor residential air samples, from studies conducted within and outside of North America, is provided in the Draft Risk Evaluation for PCE Data Extraction for Consumer and Aquatic Exposure Monitoring Studies.

Personal Breathing Zone

Concentrations of PCE in personal breathing zone measurements are reported in Table 2-62 for seven US studies. Overall, the measured concentration dataset contains approximately 3,000 samples that were collected between 1981 and 2001, and represents time spent in various microenvironments (i.e., home, school, work, transit) during the monitoring period (48- to 72-hr periods in four studies, and 3-hr, 12-hr, and/or 6-day periods for the remainder). Only the 3-hr samples from Heavner (1995) represent time inside the home only. Concentrations ranged from non-detects (detections limits varied) to 659 µg/m^3. The highest concentration was observed in NHANES survey data from 1999-2000 (Jia et al. 2008a). The study notes that two participants had exposure to highly elevated levels of PCE; one participant spent more time than usual at work/school and the other participant worked with paint thinners, brush cleaners, or strippers as well as glues, adhesives, hobbies or crafts, and also reported having new carpet installed in the past 6 months. The 95th percentile concentration for the NHANES study was 18.5 µg/m^3. Maximum reported concentrations in other studies were less than 11 µg/m^3 (including the 90^{th} or 95^{th} percentile if a maximum was not provided). Median values ranged from 0.4 to 2 µg/m^3; whereas, average values were higher, reaching a maximum of approximately 30 µg/m^3 (Sexton et al. 2007; Clayton et al. 1999). Full data extraction details for personal breathing zone samples, from studies conducted within and outside of North America, is provided in the Draft Risk Evaluation for PCE Data Extraction for Consumer and Aquatic Exposure Monitoring Studies.

Table 2-63. Personal Breathing Zone Air Concentrations (µg/m^3) for PCE in the United States (General/Residential)

Study Info	Type	Site/Population Description	Detection Limit	Min.	Mean	GM	Median	Max.	Variance	Data Eval. Score
(Su et al. 2013)[a] US, 1999-2001 (n=544; DF = NR)	48-hr	Elizabeth, NJ; Houston, TX; and Los Angeles, CA; Adults (n=309) and children (n=118) from 310 non-smoking households.	0.21	--	7.17	--	0.89	6.82 (95th)	112.35 (SD)	Medium
(Jia et al. 2008b)[b] US, 1999-2000 (n=665; DF = 0.69)	48- to 72-hr	Nation-wide; Adults (ages 20–59 years) in NHANES study	0.42	ND (0.1)	5.2	1.0	0.7	659.1 (18.5 - 95th)	31.2 (SD); 4.1 (GSD)	Medium
(Adgate et al. 2004) US, 2000 (n=113; DF = 1)	48-hr	Minneapolis, MN in winter; children ages 6-10 yrs	--	0.2 (10th)		--	0.4	1.3 (90th)	--	Medium
(Adgate et al. 2004) US, 2000 (n=113; DF = 0.966)	48-hr	Minneapolis, MN in spring; children ages 6-10 yrs	--	ND (0.2 10th)		--	0.4	0.9 (90th)	--	Medium
(Sexton et al. 2007) US, 1999 (n=333; DF = 0.997)	48-hr	Minneapolis -St. Paul, MN; Adults, non-smoking (n=70) living in three neighborhoods: (inner-city, blue-collar/near manufacturing plants, and affluent)	--	ND (0.3 10th)	27.8	--	0.9	6.4 (90th)	--	High
(Clayton et al. 1999)[c] US, 1995-1997 (n=386; DF = 0.613)	6-day	IL, IN, OH, MI, MN, WI (Great Lakes Region); Non-institutionalized persons	--	ND	31.92	--	1.98	10.78 (90th)	--	High
(Heavner et al. 1995) [d] US, 1991 (n=25; DF = NR)	3-hrs (in home only)	Columbus, OH; Non-smoking (n=25) women with smoking husbands	--	ND	0.89	--	0.68	3.78	0.96 (SD)	Medium
(Heavner et al. 1995) [d] US, 1991 (n=24; DF = NR)	3-hrs (in home only)	Columbus, OH; Non-smoking women (n=24) with non-smoking husbands	--	ND	1.24	--	0.7	5.13	1.46 (SD)	Medium
(Wallace 1987)[e] US, 1981-1984 (n=772; DF = 0-0.97)	12-hrs	Elizabeth and Bayonne, NJ, Los Angeles, CA, and Contra Costa, CA; Adults s in industrial/chemical manufacturing and /or petroleum refining regions of the US.	--	--	5.6 to 45	--	--			High

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. DF = detection frequency. NR = Not reported. US = United States.
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 (Su et al. 2013) were collected as part of the RIOPA study. The study notes that PCE exposures increased by visiting a drycleaner.
[b] Samples from this study (Jia et al. 2008b) were collected as part of the NHANES 1999-2000. Two measurements with high values (659 and 490 μg /m^3) were more than five times higher than the next measurement. These two participants did not report dry cleaning exposure, breathing fumes from or using dry cleaning fluid or spot remover. One participant spent an unusually large amount of time at work/school and another subject worked with paint thinners, brush cleaners, or strippers as well as glues, adhesives, hobbies or crafts, and also reported having new carpet installed in the past 6 months.
[c] Samples from this study (Clayton et al. 1999) were collected as part of the NHEXAS Phase 1 field study.
[d] In Heavner (1995), elevated concentrations of PCE were associated with wearing dry cleaned clothes ($p \leq 0.05$) when all homes were combined, but not for smoking and non-smoking separately. Statistical power was low since only 2 of 49 participants wore dry cleaned clothes within the previous week.
[e] Samples from this study (Wallace 1987) were collected as part of the TEAMS study.

2.4.2.2 Consumer Exposure Approach and Methodology

Consumer exposures to PCE are expected via inhalation and dermal routes based on physical-chemical properties and identified consumer uses. PCE can be found in consumer and/or commercial products that are readily available for public purchase at common retailers ((U.S. EPA 2017f), Sections 3, 4 and 5) and can therefore result in exposures to consumers and bystanders (non-product users that are incidentally exposed to the product). The magnitude of exposure depends upon the concentration of PCE products, use patterns (including frequency, duration, amount of product used, room of use) and application methods. Several consumer product use scenarios were analyzed based on identified PCE products and articles available to consumers, including solvents for cleaning and degreasing, lubricants and greases, adhesives and sealant chemicals, paints and coatings, mold release products, metal and stone polishes, and exposure to recently dry cleaned articles. Consumer exposure to elevated indoor air concentrations of PCE due to the use of coin-operated dry cleaning machines and retail print-shops was summarized based on available literature.

Consumer product application activities include using aerosol and liquid products for spraying, wiping, immersive cleaning and painting. Other activities include pouring and applying various types of liquids and pastes. Information regarding use patterns and application methods was obtained from national solvent usage surveys (Westat 1987), as well as EPA's Consumer Exposure Model (CEM) Version 2.1 (see CEM 2.1 User Guide (U.S. EPA 2019b)). PCE weight fractions and product densities of PCE containing products were compiled from publicly available product MSDS or SDS documents (Material Safety Data Sheet or Safety Data Sheet, see EPAs Preliminary Information on Manufacturing, Processing, Distribution, use and Disposal: Tetrachloroethylene (2017f)). If product densities were not reported, the product density was estimated based on reported mass percent composition of the product relative to constituent densities. Other physical-chemical parameters for PCE are referenced in the Scoping and Problem Formulation documents.

2.4.2.2.1 Routes of Exposure

Inhalation

Consumer and bystander inhalation exposure to PCE-containing products primarily include direct inhalation of vapors, mists and aerosols (e.g., aerosols from spray applications) and indirect inhalation exposures after application. EPA assumed mists are absorbed via inhalation, rather than ingestion, due to deposition of vapors and mists in the upper respiratory tract. The magnitude of inhalation exposure depends upon the concentration of PCE in products, use patterns (including frequency, duration, amount of product used, room of use) and application methods. Several product types and scenarios were analyzed for inhalation exposure including spray adhesives, spray lubricants, spray paints and primers, spray degreasers (brake and engine cleaning, parts cleaning and electronics cleaning), spray protectants and stain removers. Consumer inhalation exposure to PCE emitted from recently dry cleaned articles was also evaluated. Given the high vapor pressure of PCE, products used in the liquid form are also likely to result in inhalation exposure to consumers and bystanders. PCE containing liquid product use categories include parts cleaners and degreasers, stone and marble polishes, adhesives and sealants, ceramic overglaze, and paint primers.

Dermal

Consumer dermal exposure to PCE-containing products occurs via vapor or mist deposition onto the skin, or via direct contact with liquids during product use, and direct contact with treated articles (U.S. EPA 2012d). PCE is absorbed dermally, and exposure magnitude depends on exposure characteristics such as skin surface area, product volume, chemical loading and weight fraction, and exposure duration. PCE is a volatile solvent, expected to evaporate from skin quickly. However, there are certain consumer use scenarios for which product evaporation may be limited, for example due to immersion of hands into a reservoir of cleaning solvent (reasonable given that consumers are not assumed to use PPE, as well as the nature of PCE containing products and uses), the wearing of recently dry cleaned fabrics, or handling/wiping using a solvent soaked rag. Consumer uses analyzed for dermal exposure with impeded evaporation include immersive parts cleaning, aerosol degreasers, liquid stone and marble polishes, liquid sealants, liquid paint primers and the wearing of recently dry cleaned articles.

Ingestion

Consumers may be exposed to PCE via transfer of chemical from hand to mouth. However, this exposure pathway is expected to be limited by a combination of dermal absorption and high volatilization of PCE. Due to the expected very low magnitude of accidental hand to mouth exposure, 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.

2.4.2.2.2 Modeling Approach

EPA estimated consumer exposures for all currently known use scenarios for products containing PCE. A variety of sources were reviewed during the Systematic Review process to identify these products and/or articles, including Safety Data Sheets (SDS), National Institutes of Health (NIH) Household Products Database, the Chemical and Products (CPCat) Database, Peer-reviewed and gray literature and the Kirk-Othmer Encyclopedia of Chemical Technology.

Consumer exposures were assessed for all PCE containing products identified as available for consumer purchase, as described in EPAs Preliminary Information on Manufacturing, Processing, Distribution, use and Disposal: Tetrachloroethylene (2017f). No chemical-specific personal monitoring data was identified during Systematic Review, except in the case of exposure to PCE from recently dry cleaned articles, and indoor air concentrations from coin-operated laundry and printshop proximity. Due to the lack of consumer monitoring data, a modeling approach was used to estimate potential consumer exposures. EPA's Consumer Exposure Model (U.S. EPA 2017a) was selected as the most appropriate model for PCE consumer product use scenarios, as described in below and in the Draft Risk Evaluation for PCE Supplemental Information on Consumer Exposure. CEM was used to estimate indoor air concentrations of PCE and dermal exposure to PCE in certain scenarios, generated from the use of consumer products. Consumer exposure to recently dry cleaned fabrics was also estimated, based on reasonably available monitoring data. Inhalation exposure due to off-gassing from recently dry cleaned articles was assessed using EPA's Multi-Chamber Concentration and

Exposure Model (MCCEM, (U.S. EPA 2019e)), and dermal exposure due to wearing dry cleaned articles was assessed using CEM, as described in the Draft Risk Evaluation for PCE Supplemental Information on Consumer Exposure.

EPA's Consumer Exposure Model was chosen based on model relevance to consumer use scenarios, the in-model database of consumer relevant default parameters, and model flexibility to modify parameters when chemical-specific information is available. CEM was also preferred because it does not require chemical- and/or product-specific emission data, as is required to run more complex indoor/consumer models. CEM is a deterministic model utilizing user provided input parameters and/or assumptions to generate exposure estimates. A full discussion of CEM features and general parameterization can be found in the *Draft Risk Evaluation for Perchloroethylene Supplemental Information on Consumer Exposure* (U.S. EPA 2020f).

Model parameters were determined based on physical chemical properties and product information (e.g., product density, water solubility, vapor pressure, etc.), use-specific consumer survey data (Westat (1987); e.g., duration of use, frequency of use, mass of product used per event, etc.), and where applicable, model scenario defaults (e.g., room of use, activity patterns, air exchange rates, environment volume). A negligible background concentration of PCE was assumed for all scenarios. Room of use was selected based on either CEM scenario default room of use or a Westat survey category room of use (often in agreement with one another), based on professional judgement. The CEM model does not currently accommodate outdoor scenarios. For products that are intended to be used outdoors, modifications to the CEM inputs were made to simulate an outdoor scenario by adjusting Zone 1 parameters (which represents the room of use or use environment). In modeling caulk and column adhesives, the garage was selected as the room of use, but the room volume was changed to 16 m^3 to represent a half-dome chemical cloud around the person using the product. Additionally, the air exchange rate for Zone 1 was set to 100 to reflect the high rate between the cloud and the rest of outside. The interzonal ventilation rate was set to 0, which effectively blocks the exchange of air between Zone 1 and the rest of the house. Thus, the concentrations users are exposed to inside the home after product use is zero. In the outside scenario, bystanders in the home are assumed to have zero exposures. However, bystanders in the outdoor environment were not modeled, but could potentially be exposed to similar levels as the user.

While inhalation exposure can be acute or chronic in nature, EPA does not expect consumer exposure to be chronic in nature because product use patterns tend to be infrequent with relatively short durations of use. As a result, we only present the acute consumer results in this risk evaluation. 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 (U.S. EPA 2011a). In addition to exposure doses, indoor air concentrations were estimated and reported as maximum 24 hour time-weighted-averages (24 hr TWA).

Thirteen distinct product categories were identified for CEM modeling. Product categories were assigned based on the physical form of the product (aerosol, liquid, wipe, etc.) and intended use. See Table 2-64 and Table 2-65 for groupings and the corresponding CEM parameters for each scenario.

To characterize the potential range of consumer exposures, modeling for each scenario was conducted by varying three key parameters while keeping all other input parameters constant. The key parameters included duration of use per event (minutes/use), amount of chemical in the product or article (weight fraction), and mass of product or article used per event (gram/use). Duration of use and mass of product used were assigned to each use category based on the Westat (1987) survey of consumer behavior patterns. Each scenario was evaluated at a low, medium, and high value (10^{th}, 50^{th}, and 95^{th} percentiles) for duration of use and mass of product used, based on the most representative product use category. Product weight fractions were determined from review of product Safety Data Sheets and any other information identified during Systematic Review. This input parameter was varied using minimum, mean and maximum values, unless only a single product was identified for a given use scenario. Input parameters for PCE containing consumer product scenarios modeled in CEM are given in Table 2-63 and Table 2-64. For full parametrization details see the *Draft Risk Evaluation for Perchloroethylene Supplemental Information on Consumer Exposure* (U.S. EPA 2020f).

Inhalation Exposure Estimation

Inhalation exposure to PCE containing products was estimated using CEM, which predicts indoor air concentrations by implementing a deterministic, mass-balance calculation selected by the user (see CEM 2.1 User Guide (U.S. EPA 2019b) and *Draft Risk Evaluation for Perchloroethylene Supplemental Information on Consumer Exposure* (U.S. EPA 2020f)). 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. Product users and bystanders follow prescribed activity patterns and inhale airborne concentrations determined by the activity zone. All PCE scenarios were assessed using the near-field/far-field model option to capture the potentially higher concentration in the breathing zone of a product user during use.

Inhalation exposure to PCE as a result of proximity to recently dry cleaned articles was estimated using MCCEM (U.S. EPA 2019e), which utilizes chemical- and article-specific emission parameters to predict indoor air concentrations (see Section 2.4.2.2.2 for further details).

Dermal Exposure Estimation

Dermal exposure to PCE from consumer product use was estimated using CEM's permeability method (P_DER2b). The permeability method is based on the ability of a chemical to penetrate the skin layer once contact occurs. The model assumes a constant supply of chemical, directly in contact with the skin, throughout the exposure duration. Evaporative loss of PCE from the skin during product use is expected to be considerable, except in cases where the nature of use limits evaporation, such as from the use of a solvent soaked rag, or immersion of hands in a container of PCE based cleaner. Only product use scenarios where a reasonable assumption could be made for limited evaporation from skin were assessed for dermal exposure. A chemical-specific skin permeability coefficient of 1.8×10^{-2} cm/hr was used for permeability estimates (Nakai et al. 1999).

Dermal exposure to PCE from recently dry cleaned fabrics was estimated using CEM's direct-contact article model (A_DER2). This model estimates dermal exposure based on the migration rate of a chemical from an article to the skin, which is governed by the solid phase diffusion coefficient, in combination with age-specific activity patterns to estimate potential loading on the skin.

Exposure Receptors

Consumer use scenarios were assessed for adults (age 21+) and two youth age-groups (16-20 years and 11-15 years) as product users. All other individuals were considered as non-users (treated as bystanders). CEM was parameterized based on characteristics of exposed populations and receptor factors (such as age-specific body weight, skin surface area, inhalation rates, etc. all based on Exposure Factors Handbook (U.S. EPA 2011a)); user and bystander activity patterns; building volumes and air exchange rates; and product use considerations.

5086 **Table 2-64. CEM Consumer Product Modeling Scenarios and Key Product Parameters**

Consumer Conditions of Use	Form	No. of Products Identified[1]	Range of Weight Fractions Identified (% PCE)[2]	Weight Fractions Selected for Use in Modeling (% PCE)			Selected Product Density (g/cm^3)[3]	Selected CEM 2.1 Modeling Scenario[4]	Emission Model Applied[5]	Dermal Exposure Model Applied[6]	Dermal SA/BW[7]
				Min	Mean	Max					
Solvent; Cleaner; Marine cleaner; Degreaser; Coil cleaner; Electric motor cleaner ; Parts cleaner; Cable cleaner; Stainless Steel Polish; Electrical/Energized Cleaner; Wire and ignition demoisturants; Electric motor cleaner	Aerosol	15	10-100	10	80	100	1.62	Degreasers	E3	P_DER1b	10% of hands
Parts cleaner	Liquid	1	50-60	50	60	---	1.34	Generic	E5	P_DER1b	Both hands
Brake Cleaner	Aerosol	14	40-100	40	91	100	1.32	Degreasers	E3	P_DER1b	10% of hands
Vandalism Mark & Stain Remover; Mold Cleaner; Weld Splatter Protectant	Aerosol	5	5-100	5	40	100	1.62	All Purpose Spray Cleaner	E3	none	n/a
Marble Polish, Stone Cleaner	Liquid	3	10-100	10	85	100	1.62	All Purpose Liquid Cleaner	E1	P_DER1b	Inside of both hands[8]
Cutting Fluid	Liquid	1	10	10	---	---	7.72	Non-Spray Lubricant	E1	P_DER1b	Inside of both hands
Spray Lubricant; Penetrating Oil	Aerosol	9	5-100	5	54	100	1.62	Spray Lubricant	E3	none	n/a
Industrial adhesive; Adhesive; Arts and crafts adhesive; Gun ammunition sealant	Liquid	15	30-100	30	89	100	1.31	Glues and Adhesives (small scale)	E1	none	n/a

Consumer Conditions of Use	Form	No. of Products Identified[1]	Range of Weight Fractions Identified (% PCE)[2]	Weight Fractions Selected for Use in Modeling (% PCE)			Selected Product Density (g/cm^3)[3]	Selected CEM 2.1 Modeling Scenario[4]	Emission Model Applied[5]	Dermal Exposure Model Applied[6]	Dermal SA/BW[7]
				Min	Mean	Max					
Livestock Grooming Adhesive	Aerosol	1	15	15	---	---	1.45	Spray Fixative and Finishing Spray Coatings	E3	none	n/a
Column Adhesive; Caulk; Sealant	Gel/ Liquid	16	5-75	5	48	75	1.19	Caulk	E1	None	n/a
Coatings and Primers	Aerosol	10	9-14	9	10	14	1.3952	Aerosol Spray Paints	E3	none	n/a
Rust primer; Sealant	Liquid	9	9-11	9	10	11	1.3952	Solvent-Based Wall Paint	E2	P_DER1b	Face, hands and arms
Sealant (Water Shield)	Liquid	1	45	45	---	---	1.28	Solvent-Based Wall Paint	E2	P_DER1b	Face, hands and arms
Metallic Overglaze (for ceramics)	Liquid	1	20-30	20	30	---	1	Lacquers and Stains	E2	none	n/a
Marble Polish, Stone Cleaner	Liquid Wax	1	85-100	85	95	100	1.4	All Purpose Waxes and Polishes	E1	P_DER1b	Inside of both hands

[1] The number of products identified is based on the product lists in EPA's 2017 *Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: Tetrachloroethylene (PCE)* (2017f). It is possible that specific products and/or formulations identified in those reports and used herein to select appropriate weight fractions, formulation types, and formulation densities for use in modeling no longer contain PCE or are no longer readily available to consumers for purchase; however, they were still considered for sourcing such information since they were identified as in these recent EPA publications and therefore represent reasonably-foreseen uses. *See Draft Risk Evaluation for Perchloroethylene Supplemental Information for Consumer Exposure* (U.S. EPA 2020f) for the full product list utilized.

[2] The range in weight fractions is reflective of the identified products containing PCE and not reflective of hypothetical levels or theoretical functionality-based limits. Weight fractions were sourced from product Safety Data Sheets (SDSs) or Material Safety Data Sheets (MSDSs).

[3] Product densities were identified from product SDSs or MSDSs. When density was not reported in product MSDS or SDSs, products with high PCE weight fractions (>90% PCE) were assumed to have the density of pure PCE (1.62 g/cm^3), otherwise the product density was calculated based on the percent

contribution of each ingredient per the MSDS ingredient list. See *See Draft Risk Evaluation for Perchloroethylene Supplemental Information for Consumer Exposure* (U.S. EPA 2020f) for the full product list utilized.

[4] The listed CEM 2.1 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).

[5] Emission models used for PCE include E1 – Emission from Product Applied to a Surface Indoors Incremental Source Model, E2 – Emission from Product Applied to a Surface Indoors Double Exponential Model, E3 – Emission from Product Sprayed, and E5 – Emission from Product Placed in Environment.

[6] All product scenarios utilized the P_DER1b model for dermal exposure – Dermal Dose from Product Applied to Skin, Permeability Model.

7Suface Area to Body Weight (SA/BW) ratios are default parameters for the selected CEM use scenarios, values are based on central tendency (mean) values (Exposure Factors Handbook (U.S. EPA 2011a), CEM 2.1 User Guide (U.S. EPA 2019b))

[8]CEM default dermal SABW ratio for the All-Purpose Liquid Cleaner category is one hand, however both hands were modeled for consistency between wax vs. liquid stone polish use categories.

5110 **Table 2-65. Consumer Product Modeling Scenarios and Key Westat Product Use Parameters**

Consumer Conditions of Use	Form	Selected Westat (1987) Survey Scenario[1]	Room of Use[2]	Duration of Use (Percentile) (min) (10th)[3]	50th	95th	Mass of Product Used (Percentile) (g)[4] 10th	50th	95th
Solvent; Cleaner; Marine cleaner; Degreaser; Coil cleaner; Electric motor cleaner ; Parts cleaner; Cable cleaner; Stainless Steel Polish; Electrical/Energized Cleaner; Wire and ignition demoisturants; Electric motor cleaner	Aerosol	Solvent-Type Cleaning Fluids or Degreasers	Utility Room	2	15	120	26.83	155.69	1532.91
Parts cleaner	Liquid	Spot Remover	Utility Room	0.5 (0.25)	5	30	9.91	52.70	441.01
Brake Cleaner	Aerosol	Brake Quieters/ Cleaners	Garage	1	15	120	39.03	156.13	624.52
Vandalism Mark & Stain Remover; Mold Cleaner; Weld Splatter Protectant	Aerosol	Solvent-Type Cleaning Fluids or Degreasers	Utility Room	2	15	120	26.83	155.69	1532.91
Stone Polish	Liquid	Solvent-Type Cleaning Fluids or Degreasers	Utility Room	2	15	120	26.83	155.69	1532.91
Cutting Fluid	Liquid	Other Lubricants (Excluding Automotive)	Utility Room	0.5 (0.08)	2	30	26.83	155.69	1532.91
Spray Lubricant; Penetrating Oil	Aerosol	Other Lubricants (Excluding Automotive)	Utility Room	0.5 (0.08)	2	30	4.79	26.35	239.51
Industrial adhesive; Adhesive; Arts and crafts	Liquid	Contact Cement, Super Glues, and	Utility Room	0.5 (0.33)	4.25	60	1.16	9.68	167.34

Consumer Conditions of Use	Form	Selected Westat (1987) Survey Scenario[1]	Room of Use[2]	Duration of Use (Percentile) (min) (10th)[3]	50th	95th	Mass of Product Used (Percentile) (g)[4] 10th	50th	95th
adhesive; Gun ammunition sealant		Spray Adhesives							
Livestock Grooming Adhesive	Aerosol	Contact Cement, Super Glues, and Spray Adhesives	Utility Room	0.5 (0.33)	4.25	60	1.29	10.72	185.23
Column Adhesive; Caulk; Sealant	Gel/ Liquid	Primers and Special Primers (excluding automotive)	Garage	5	30	360	45.39	387.07	8121.46
Coatings and Primers	Aerosol	Aerosol Spray Paint	Utility Room	5	20	120	61.88	330.05	1608.99
Rust primer; Sealant	Liquid	Primers and Special Primers (excluding automotive)	Garage	5	30	360	53.22	453.82	9521.90
Sealant (Water Shield)	Liquid	Outdoor Water Repellent	Garage	15	60	300	302.8	2422.37	24223.74
Metallic Overglaze (for ceramics)	Liquid	Contact Cement, Super Glues, and Spray Adhesives	Utility Room	0.5 (0.33)	4.25	60	0.89	7.39	127.74
Marble and Stone Polish	Wax	Solvent-Type Cleaning Fluids or Degreasers	Utility Room	2	15	120	23.18	134.54	1324.74

[1] (Westat 1987)
[2] Room of use is either default scenario option within CEM or based on Westat survey data for the specific product use category.

[3] CEM has a minimum timestep of 0.5 min. If the 10th percentile duration of use was less than 0.5 min, then the actual 10th percentile is reported in parenthesis.

[4] Westat Survey scenario data for mass of product used is reported in ounces. The product density was used to convert percentile results from ounces to grams for use in CEM. As a result, mass of product used will be different for product categories with the same identified Westat Survey use scenario, but different product densities.

2.4.2.3 Consumer Product Exposure Scenarios

Consumer products were assessed for human user and bystander inhalation exposure, and for user dermal exposure when it was reasonable to assume that use characteristics would limit product evaporation from skin. The results of modeled consumer scenarios are presented below, in order of the consumer product Categories of Use (COUs) identified in Table 2-12 (Crosswalk of Subcategories of Use).

2.4.2.3.1 Degreasers

PCE containing aerosol-based degreasers were identified as available for consumer use. Two sub-categories of degreasers were identified, general aerosol degreasers and brake cleaners, based on the most appropriate use scenario.

2.4.2.3.1.1 Aerosol Cleaners for Motors, Coils, Electrical Parts, Cables, Stainless Steel and Marine Equipment, and Wire and Ignition Demoisturants

Aerosol-based degreasers for motors, coils, electrical parts, cables, stainless steel and marine equipment, and wire and ignition demoisturants were identified as available for consumer use, with reported PCE weight fractions of 10% to 100%. Inhalation and dermal exposures were evaluated users, and inhalation exposures were evaluated bystanders, for three use scenarios (Table 2-66 and Table 2-67). Dermal exposure was considered relevant for this product category due to the large volume of liquid emitted from the spray can during use, and likelihood of handling product-soaked rags during normal product use, as per manufacturer instructional videos. Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 1.5 to 869 mg/m^3 for users, and 0.3 to 216 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 0.1 to 74 mg/kg/day across all user age groups.

Table 2-66. Consumer inhalation exposure to PCE during use in degreasers for motors, coils, electrical parts, cables, stainless steel and marine equipment, and wire and ignition demoisturants

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (2)	Min (10)	10th (26.83)	User Bystander	1.5 0.3
Moderate Intensity User	50th (15)	Mean (80)	50th (155.69)	User Bystander	74 14
High Intensity User[1]	95th (120)	Max (100)	95th (1532.91)	User Bystander	869 216

[1]The maximum 24 hr TWA air concentration for the User was the 50th percentile duration -maximum weight fraction-50th percentile mass used iteration, with a PCE air concentration of 904 mg/m^3.

Table 2-67. Consumer dermal exposure to PCE during use in degreasers for motors, coils, electrical parts, cables, stainless steel and marine equipment, and wire and ignition demoisturants

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10th (2)	Min (10)	10th (26.83)	User, Adult (≥21 yr) User, Youth (16-20 yr)	0.1 0.1

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
				User, Youth (11-15 yr)	0.1
Moderate Intensity User	50th (15)	Mean (80)	50th (155.69)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	7.2 6.8 7.4
High Intensity User	95th (120)	Max (100)	95th (1532.91)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	72 68 74

Confidence in the selected model and default parameters is high for inhalation exposure during aerosol degreasing. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the selected model is medium for dermal exposure during aerosol degreasing. CEM's permeability model assumes limited evaporation, which is appropriate for aerosol degreasing considering the common use of solvent soaked rags when using aerosol degreasing products. However, if consumers used this product in such a way that evaporation was not impeded, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use is high due to a good match in the Westat survey data, which received a high- quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the aerosol degreaser inhalation exposure estimations is high. The overall confidence in the aerosol degreaser dermal exposure estimations is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation from the hands is not impeded.

2.4.2.3.1.2 Aerosol Brake Cleaners

Aerosol-based degreasers in the form of brake cleaners were identified as available for consumer use, with reported PCE weight fractions of 40% to 100%. Inhalation and dermal exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-68 and Table 2-69). Dermal exposure was considered relevant for this product category due to the large volume of liquid emitted from the spray can during use, and likelihood of handling product-soaked rags during normal product use, as per manufacturer instructional videos. Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 5.7 to 250 mg/m^3 for users, and 1.6 to 73 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 0.2 to 60 mg/kg/day across all user age groups.

Table 2-68. Consumer inhalation exposure to PCE during use in brake cleaner

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
	10th	Min	10th	User	5.7

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	(1)	(40)	(39.03)	Bystander	1.6
Moderate Intensity User	50th (15)	Mean (91)	50th (156.13)	User Bystander	59 15
High Intensity User[1]	95th (120)	Max (100)	95th (624.52)	User Bystander	250 73

[1]The maximum 24 hr TWI air concentration for the User was the 50th percentile duration -maximum weight fraction-95th percentile mass used iteration, with a PCE concentration of 259 mg/m^3.

Table 2-69. Consumer dermal exposure to PCE during use in brake cleaner

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10th (1)	Min (40)	10th (39.03)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	0.2 0.2 0.2
Moderate Intensity User	50th (15)	Mean (91)	50th (156.13)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	6.7 6.3 6.9
High Intensity User	95th (120)	Max (100)	95th (624.52)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	59 55 60

Confidence in the selected model and default parameters is high for inhalation exposure during brake cleaning. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the selected model is medium for dermal exposure during brake cleaning. CEM's permeability model assumes limited evaporation, which is appropriate for brake cleaning considering the common use of solvent soaked rags when using brake cleaning products. However, if consumers used this product in such a way that evaporation was not impeded, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high- quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the brake cleaner inhalation exposure estimations is high. The overall confidence in the brake cleaner dermal exposure estimations is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation from the hands is not impeded.

2.4.2.3.2 Parts Cleaners

Liquid-based parts cleaner (wipe or immersive) was identified as available for consumer use, with reported PCE weight fraction of 50% to 60%. Inhalation and dermal exposures were evaluated users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-70 andTable 2-71). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 0.4 to 161 mg/m^3 for users, and 6.5E-02 to 29 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 25 to 2030 mg/kg/day across all user age groups.

Table 2-70. Consumer inhalation exposure to PCE during use in parts cleaners

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (0.25)[2]	Min (50)	10th (9.91)	User Bystander	0.3 6.5E-02
Moderate Intensity User	50th (5)	Max (60) [1]	50th (52.70)	User Bystander	19 3.5
High Intensity User	95th (30)	Max (60)	95th (441.01)	User Bystander	161 29

[1]A single product was identified for immersive and/or wipe cleaning, with a range given for the weight fraction. The weight fraction range was evaluated as minimum and maximum, with no average weight fraction used in modeling.
[2]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Table 2-71. Consumer dermal exposure to PCE during use in parts cleaners

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10th (0.25)[2]	Min (50)	10th (9.91)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	25 26 28
Moderate Intensity User	50th (5)	Max (60) [1]	50th (52.70)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	296 310 338
High Intensity User	95th (30)	Max (60)	95th (441.01)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	1780 1860 2030

[1]A single product was identified for immersive and/or wipe cleaning, with a range given for the weight fraction. The weight fraction range was evaluated as minimum and maximum, with no average weight fraction used in modeling.
[2]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Confidence in the selected model and default parameters is high for inhalation exposure during immersive parts cleaning estimation, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high. A generic emission model (E5) was selected in CEM due to the lack of an existing scenario that would represent a good fit for immersive parts cleaning. However, the

selected emission model is a good fit for this condition of use. Confidence in the selected model is medium for dermal exposure during immersive parts cleaning. CEM's permeability model assumes limited evaporation, which is appropriate considering the likelihood of a user immersing their hands in an immersive cleaning product during use. However, if consumers used this product in such a way that evaporation was not impeded, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in the mass used and duration of use is medium. Lacking an exact match in the Westat survey for immersive parts cleaning, the spot remover scenario was selected to parameterize CEM. The spot remover scenario was of relatively short duration and low mass of product used, and thus the results may underestimate the inhalation exposure for immersive parts cleaning. The overall confidence in the immersive parts cleaner inhalation exposure estimations is medium, with possible underestimation of inhalation exposures. The overall confidence in the immersive parts cleaner dermal exposure estimations is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation from the hands is not impeded.

2.4.2.3.3 Vandalism Stain Removers, Mold Cleaners, and Weld Splatter Protectants

Aerosol-based mark and stain removers and splatter protectors were identified as available for consumer use, with reported PCE weight fractions of 5% to 100%. Inhalation exposures were evaluated for users, and for bystanders, for three use scenarios (Table 2-72). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 0.7 to 869 mg/m^3 for users, and 0.2 to 216 mg/m^3 for bystanders.

Table 2-72. Consumer inhalation exposure to PCE during use in vandalism stain removers, mold cleaners, weld splatter protectants

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (2)	Min (5)	10th (26.83)	User Bystander	0.7 0.2
Moderate Intensity User	50th (15)	Mean (40)	50th (155.69)	User Bystander	37 7.2
High Intensity User	95th (120)	Max (100)	95th (1532.91)	User Bystander	869 216

Confidence in the selected model and default parameters is high for estimation of inhalation exposure during use of stain removers, mold cleaner and splatter protectors, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the inhalation exposure estimation for use of stain removers, mold cleaners and splatter protectors is high.

2.4.2.3.4 Marble Polish

A liquid-based stone polish was identified as available for consumer use, with reported PCE weight fraction of 10% to 100%. Inhalation and dermal exposures were evaluated users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-73 andTable 2-74). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 3.4 to 911 mg/m^3 for users, and 0.7 to 227 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 1.1 to 739 mg/kg/day across all user age groups.

Table 2-73. Consumer inhalation exposure to PCE during use in marble polish

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (2)	Min (10)	10th (26.83)	User Bystander	3.4 0.7
Moderate Intensity User	50th (15)	Mean (85)	50th (155.69)	User Bystander	166 32
High Intensity User	95th (120)	Max (100)	95th (1532.91)	User Bystander	911 227

Table 2-74. Consumer dermal exposure to PCE during use in marble polish

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10th (2)	Min (10)	10th (26.83)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	1.2 1.1 1.2
Moderate Intensity User	50th (15)	Mean (85)	50th (155.69)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	77 72 79
High Intensity User	95th (120)	Max (100)	95th (1532.91)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	722 676 739

Confidence in the selected model and default parameters is high for inhalation exposure during marble polish use. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. The utility room was selected as the room of use for this scenario. While it is also reasonable to assume that marble polish may be used in the kitchen, the room volumes are similar and air exchange rates identical, resulting in similar user inhalation exposure. However, a difference may occur for the bystander inhalation exposure when considering utility room use versus kitchen use, based on bystander activity patterns. For example, amount of time the bystander spends in the kitchen is greater than time spent in the utility room, resulting in a lower bystander inhalation exposure for the utility room scenario. If the product was used in the kitchen, the bystander inhalation exposure would be greater than estimated, up to the air concentration experienced by the user. Confidence in the selected model is medium for dermal exposure during marble polish use. CEM's

permeability model assumes limited evaporation, which is appropriate for marble polish considering the common use of solvent soaked rags when using marble cleaning products. However, if consumers used this product in such a way that evaporation was not impeded, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high- quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the marble polish user inhalation exposure estimations is high, with possible underestimation of bystander inhalation exposures if the room of use changed. The overall confidence in the marble polish use dermal exposure estimations is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation from the hands is not impeded.

2.4.2.3.5 Cutting Fluid

Cutting fluid was identified as available for consumer use, with a reported PCE weight fraction of 10%. Inhalation exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-75). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 1.4 to 91 mg/m^3 for users, and 0.3 to 19 mg/m^3 for bystanders.

Table 2-75. Consumer inhalation exposure to PCE during use in cutting fluids

Scenario Description	Duration Percentile (min)	Weight Fraction[1] (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (0.08)[2]	Single (10)	10th (26.83)	User Bystander	1.4 0.3
Moderate Intensity User	50th (2)	Single (10)	50th (155.69)	User Bystander	8.5 1.7
High Intensity User	95th (30)	Single (10)	95th (1532.91)	User Bystander	91 19

[1]A single product was identified for cutting fluid, with a single weight fraction reported.
[2]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Confidence in the selected model and default parameters is high for estimation of inhalation exposure during use of cutting fluids, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the inhalation exposure estimation during use of cutting fluids is high.

2.4.2.3.6 Lubricants and Penetrating Oils (aerosol)

Aerosol-based lubricants and penetrating oils were identified as available for consumer use, with reported PCE weight fractions of 5% to 100%. Inhalation exposures were evaluated for users, and

inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-76). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 0.1 to 142 mg/m^3 for users, and 2.6E-02 to 29 mg/m^3 for bystanders.

Table 2-76. Consumer inhalation exposure to PCE during use in lubricating and penetrating oils

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (0.08)[1]	Min (5)	10th (4.79)	User Bystander	0.1 2.6E-02
Moderate Intensity User	50th (2)	Mean (54)	50th (26.35)	User Bystander	7.9 1.6
High Intensity User	95th (30)	Max (100)	95th (239.51)	User Bystander	142 29

[1]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Confidence in the selected model and default parameters is high for estimation of inhalation exposure during use of aerosol lubricants and penetrating oils, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the inhalation exposure estimation during use of aerosol lubricants and penetrating oils is high.

2.4.2.3.7 Adhesives

Industrial adhesives, arts and crafts adhesives, and gun ammunition sealant was identified as available for consumer use, with PCE weight fractions of 10% to 100%. Inhalation exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-77). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 0.2 to 90 mg/m^3 for users, and 3.8E-02 to 23 mg/m^3 for bystanders.

Table 2-77. Consumer inhalation exposure to PCE during use in adhesives

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (0.33)[2]	Min (30)	10th (1.16)	User Bystander	0.2 3.8E-02
Moderate Intensity User	50th (4.25)	Mean (89)	50th (9.68)	User Bystander	4.9 1.0
High Intensity User [1]	95th (60)	Max (100)	95th (167.34)	User Bystander	90 23

[1]The maximum 24 hr TWA air concentration for the User was the 50th percentile duration-maximum weight fraction-95th percentile mass used iteration, with a PCE concentration of 94 mg/m^3.
[2]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Confidence in the selected model and default parameters is high for estimation of inhalation exposure during adhesive use, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the inhalation exposure estimation during use of adhesives is high.

2.4.2.3.8 Livestock Grooming Adhesive (aerosol)

Livestock grooming adhesive spray was identified as available for consumer use, with a reported PCE weight fraction of 15%. Inhalation exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-78). Use was modeled indoors, as product may be used a or horse stable or other enclosed space. Indoor maximum 24-hour time weighted average (TWA) concentrations ranged from 0.1 to 15 mg/m^3 for users, and 2.1E-02 to 3.7 mg/m^3 for bystanders.

Table 2-78. Consumer inhalation exposure to PCE during use in livestock grooming adhesive

Scenario Description	Duration Percentile (min)	Weight Fraction[1] (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (0.33)[3]	Single (15)	10th (1.29)	User Bystander	0.1 2.1E-02
Moderate Intensity User	50th (4.25)	Single (15)	50th (10.72)	User Bystander	0.9 0.2
High Intensity User[2]	95th (60)	Single (15)	95th (185.23)	User Bystander	15 3.7

[1]A single product was identified for livestock grooming adhesive, with a single reported weight fraction.
[2]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.
[3]The maximum 24 hr TWA air concentration for the User was the 50th percentile duration -single weight fraction-95th percentile iteration, with a PCE concentration of 16 mg/m^3.

Confidence in the selected model and default parameters is high for estimation of inhalation exposure during livestock grooming adhesive use, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. The utility room was selected as the room of use for this scenario, assuming the product was used as a general spray fixative. If the product was used in a barn the inhalation exposure would be reduced. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The overall confidence in the inhalation exposure estimation during use of livestock

grooming adhesive is high, but overestimate exposures if the product is used in a barn rather than a utility room.

2.4.2.3.9 Caulks, Sealants and Column Adhesives

Caulks, sealants and column adhesives were identified as available for consumer use, with reported PCE weight fractions of 5% to 75%. Inhalation exposures were evaluated for users, for three use scenarios (Table 2-79). Area of use was assumed to be outdoors, so bystander exposure was not estimated. A modified garage with a high air exchange rate was used to model outdoor use. Maximum 24-hour time weighted average (TWA) air concentrations ranged from 5.9E-02 to 159 mg/m^3 for users.

Table 2-79. Consumer inhalation exposure to PCE during use in caulks, sealants and column adhesives

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (5)	Min (5)	10th (45.39)	User	5.9E-02
Moderate Intensity User	50th (30)	Mean (48)	50th (387.07)	User	4.8
High Intensity User	95th (360)	Max (75)	95th (8121.46)	User	159

Confidence in the selected model and default parameters is high for estimation of inhalation exposure from caulks, sealants and column adhesives, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. A modified garage with a high air exchange rate was used to model outdoor use, resulting in no bystander exposure. Greater user and bystander inhalation exposure would be expected for use of caulk and column adhesive products indoors. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is medium as there was not an exact match in the Westat survey data. As such, the primers and special primers (non-automotive) scenario was selected. It may be that primers are used for longer periods and in larger quantities than caulks, sealants and column adhesives, and thus the selected scenario may overestimate inhalation exposure. The overall confidence in the inhalation exposure estimation from caulks, sealants and column adhesives is medium with the possibility of overestimation based on selected scenario mass used and duration of use parameters, and/or underestimation of exposures, particularly for bystanders, based on the assumption of outdoor product use.

2.4.2.3.10 Outdoor Water Shield

Liquid-based outdoor water sealant was identified as available for consumer use, with a reported weight fraction of 45%. Inhalation and dermal exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-80 andTable 2-81). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 1.5 to 127 mg/m^3 for users, and 0.4 to 33 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 39 to 851 mg/kg/day across all user age groups.

Table 2-80. Consumer inhalation exposure to PCE during use in outdoor water shield sealants

Scenario Description	Duration Percentile (min)	Weight Fraction[1] (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User[2]	10th (15)	Single (45)	10th (302.8)	User Bystander	1.5 0.4
Moderate Intensity User	50th (60)	Single (45)	50th (2422.37)	User Bystander	10 3.4
High Intensity User[3]	95th (300)	Single (45)	95th (24223.74)	User Bystander	127 33

[1]A single product was identified for outdoor water shield, with a single reported weight fraction.
[2]The minimum 24 hr TWA air concentration for the User was the 50th percentile duration-single weight fraction-10th percentile mass used iteration, with a PCE concentration of 1.3 mg/m^3.
[3]The maximum 24 hr TWA air concentration for the Bystander was the 50th percentile duration-single weight fraction-95th percentile mass used iteration, with a PCE concentration of 34 mg/m^3.

Table 2-81. Consumer dermal exposure to PCE during use in outdoor water shield sealants

Scenario Description	Duration Percentile (min)	Weight Fraction[1] (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10th (15)	Single (45)	10th (302.8)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	41 39 42
Moderate Intensity User	50th (60)	Single (45)	50th (2422.37)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	163 155 170
High Intensity User	95th (300)	Single (45)	95th (24223.74)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	815 774 851

[1]A single product was identified for outdoor water shield, with a single reported weight fraction.

Confidence in the selected model and default parameters is high for inhalation exposure during use of an outdoor water sealant. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. The garage was selected as the room of use for this scenario, assuming application of waterproofing sealant to an item that will later be installed outside. If the product were used outside inhalation exposures would be reduced. Confidence in the selected model is medium for dermal exposure during use of an outdoor water sealant. CEM's permeability model assumes limited evaporation, which may be appropriate for liquid sealant considering a large volume is generally used with significant potential for coating of skin during use. However, if consumers used this product in such a way that evaporation was not impeded, or dermal exposure was limited, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency

assessments. The overall confidence in inhalation exposure estimations during use of an outdoor water sealant is high, but possibly overestimates inhalation exposure if the product were to be used outside, rather than inside a garage. The overall confidence in dermal exposure estimations during use of an outdoor water sealant is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation is not impeded or dermal contact is limited.

2.4.2.3.11 Aerosol Coatings and Primers

Aerosol-based rust primers and battery reconditioners were identified as available for consumer use, with reported PCE weight fractions of 9% to 14%. Inhalation exposures were evaluated for users and bystanders, for three use scenarios (Table 2-82). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 2.2E-02 to 1.9 mg/m^3 for users, and 8.4E-04 to 5.4E-02 mg/m^3 for bystanders.

Table 2-82. Consumer inhalation exposure to PCE during use in aerosol coatings and primers

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10th (5)	Min (9)	10th (61.88)	User Bystander	2.2E-02 8.4E-04
Moderate Intensity User	50th (20)	Mean (10)	50th (330.05)	User Bystander	0.2 5.3E-03
High Intensity User	95th (120)	Max (14)	95th (1608.99)	User Bystander	1.9 5.4E-02

Confidence in the selected model and default parameters is high for estimation of inhalation exposure from use of aerosol coatings and primers, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high as there is a good match in the Westat survey data. The overall confidence in the inhalation exposure estimation from use of aerosol coatings and primers is high.

2.4.2.3.12 Liquid Primers and Sealants

Rust Primer

Liquid-based rust primer and sealant was identified as available for consumer use, with reported PCE weight fractions of 9% to 11%. Inhalation and dermal exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-83andTable 2-84). Indoor use was assumed as a more conservative estimate of consumer exposure. Consumer exposure would likely be lower if the product was used outdoors. Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 1.1E-03 to 0.3 mg/m^3 for users, and 8.8E-05 to 4.9E-02 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 2.8 to 272 mg/kg/day across all user age groups.

Table 2-83. Consumer inhalation exposure to PCE during use in rust primers and sealants

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User [1]	10^{th} (5)	Min (9)	10^{th} (53.22)	User Bystander	1.1E-03 8.8E-05
Moderate Intensity User	50^{th} (30)	Mean (10)	50^{th} (453.82)	User Bystander	9.7E-03 9.1E-04
High Intensity User	95^{th} (360)	Max (11)	95^{th} (9521.90)	User Bystander	0.3 4.9E-02

[1]The minimum 24 hr TWA air concentration for the User was the 50^{th} percentile duration-minimum weight fraction-10^{th} percentile mass used iteration, with a PCE concentration of 1.0E-03 mg/m^3.

Table 2-84. Consumer dermal exposure to PCE during use in rust primers and sealants

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10^{th} (5)	Min (9)	10^{th} (53.22)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	3.0 2.8 3.1
Moderate Intensity User	50^{th} (30)	Mean (10)	50^{th} (453.82)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	237 225 247
High Intensity User	95^{th} (360)	Max (11)	95^{th} (9521.90)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	261 248 272

Confidence in the selected model and default parameters is high for inhalation exposure during use of liquid rust primers. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high as there was a good match in CEM. Confidence in the selected model is medium for dermal exposure during use of liquid rust primers. CEM's permeability model assumes limited evaporation, which may be appropriate for liquid rust primers considering a large volume may be used with potential for coating of skin during use. However, if consumers used this product in such a way that evaporation was not impeded, or dermal exposure was limited, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in previous agency assessments. The product was assumed to be used indoors, which represents a reasonable, but likely more conservative, exposure estimate than if outdoor use had been assumed. The overall confidence in inhalation exposure estimations during use of liquid rust primers is high, however outdoor use would likely result in lower consumer inhalation exposure. The overall confidence in dermal exposure estimations during use liquid rust primers is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation is not impeded or dermal contact is limited.

2.4.2.3.13 Metallic Overglaze

Metallic overglaze for ceramics was identified as available for consumer use, with a reported PCE weight fractions of 20 to 30%. Inhalation and dermal exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-85. Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 2.6E-03 to 0.5 mg/m^3 for users, and 5.4E-04 to 0.1 mg/m^3 for bystanders.

Table 2-85. Consumer inhalation exposure to PCE during use in metallic overglaze

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User [1]	10th (0.33)[4]	Min (20)	10th (0.89)	User Bystander	2.6E-03 5.4E-04
Moderate Intensity User [2]	50th (4.25)	Max (30)	50th (7.39)	User Bystander	3.4E-02 6.8E-03
High Intensity User [3]	95th (60)	Max (30)	95th (127.74)	User Bystander	0.5 0.1

[1]The minimum 24 hr TWA air concentration for the User was the 95th percentile duration-minimum weight fraction-10th percentile mass used iteration, with a PCE concentration of 2.5E-03 mg/m^3.
[2]A single product was identified for metallic overglaze, with a range given for the weight fraction. The weight fraction range was evaluated as minimum and maximum, with no average weight fraction used in modeling.
[3]The maximum 24 hr TWA air concentration for the User was the 50th percentile duration-maximum weight fraction-95th percentile mass used iteration, with a PCE concentration of 0.6 mg/m^3.
[4]CEM has a minimum timestep of 0.5 minutes. If the 10th percentile duration is less 0.5 min, then the minimum timestep was used for modeling, rather than the percentile.

Confidence in the selected model and default parameters is high for estimation of inhalation exposure from use of metallic overglaze, as this model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is medium as there was not an exact match in the Westat survey data. As such, the Contact Cement, Super Glues and Spray Adhesives scenario was selected. Metallic overglaze is sold in small quantities, and thus the 95th percentile mass used for the selected scenario is likely an overestimate for pottery glazing applications. The overall confidence in the inhalation exposure estimation from use of metallic overglaze is medium due to possible overestimation of inhalation exposure for the high intensity user.

2.4.2.3.14 Metal and Stone Polish

Liquid wax-based polishes for metal and stone were identified as available for consumer use, with reported PCE weight fraction of 85% to 100%. Inhalation and dermal exposures were evaluated for users, and inhalation exposures were evaluated for bystanders, for three use scenarios (Table 2-86and Table 2-87). Indoor maximum 24-hour time weighted average (TWA) air concentrations ranged from 11 to 750 mg/m^3 for users, and 2.2 to 187 mg/m^3 for bystanders. Dermal acute dose rate (ADR) ranged from 4.1 to 319 mg/kg/day across all user age groups.

Table 2-86. Consumer inhalation exposure to PCE during use in wax-based metal and stone polish

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor	24 hr Max TWA (mg/m^3)
Low Intensity User	10^{th} (2)	Min (85)	10^{th} (23.18)	User Bystander	11 2.2
Moderate Intensity User	50^{th} (15)	Mean (95)	50^{th} (134.54)	User Bystander	76 15
High Intensity User	95th (120)	Max (100)	95^{th} (1324.74)	User Bystander	750 187

Table 2-87. Consumer dermal exposure to PCE during use in wax-based metal and stone polish

Scenario Description	Duration Percentile (min)	Weight Fraction (%)	Mass Used Percentile (g)	Exposed Receptor (age group)	ADR (mg/kg/d)
Low Intensity User	10^{th} (2)	Min (85)	10^{th} (23.18)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	4.4 4.1 4.5
Moderate Intensity User	50^{th} (15)	Mean (95)	50^{th} (134.54)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	37 35 38
High Intensity User	95th (120)	Max (100)	95^{th} (1324.74)	User, Adult (≥21 yr) User, Youth (16-20 yr) User, Youth (11-15 yr)	312 292 319

Confidence in the selected model and default parameters is high for inhalation exposure during use of liquid wax polishes for metal and stone. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the selected inhalation emission scenario is high, as there was a good match in CEM. The utility room was selected as the room of use for this scenario. While it is also reasonable to assume that marble polish may be used in the kitchen, the room volumes are similar and air exchange rates identical, resulting in similar user inhalation exposure. However, a difference may occur for the bystander inhalation exposure when considering utility room use versus kitchen use, based on bystander activity patterns. For example, amount of time the bystander spends in the kitchen is greater than time spent in the utility room, resulting in a lower bystander inhalation exposure for the utility room scenario. If the product was used in the kitchen, the bystander inhalation exposure would be greater than estimated, up to the air concentration experienced by the user. Confidence in the selected model is medium for dermal exposure during use of liquid wax polishes for metal and stone. CEM's permeability model assumes limited evaporation, which is appropriate for marble polish considering the common use of solvent soaked rags when using marble cleaning products. However, if consumers used this product in such a way that evaporation was not impeded, then the selected model would be an overestimate of dermal exposure. Confidence in dermal model default parameters is high due to the high quality of source data. Confidence in the weight fraction is high as this information was pulled directly from product safety data sheets (SDSs). Confidence in mass used and duration of use data is high due to a good match in the Westat survey data, which received a high quality rating during data evaluation and has been applied in

previous agency assessments. The overall confidence in the liquid wax polishes for metal and stone user inhalation exposure estimations is high, with possible underestimation of bystander inhalation exposures if the room of use changed. The overall confidence in the liquid wax polishes for metal and stone dermal exposure estimations is medium with possible overestimation of dermal exposures in use scenarios where chemical evaporation from the hands is not impeded.

2.4.2.3.15 Consumer Product Exposure Summary

Consumer exposure to PCE due to use of PCE-containing products was evaluated for 15 product scenarios. A modeling approach was taken, based heavily on empirical and survey data, to estimate dermal and inhalation exposures. Ideally, consumer product exposure estimates would be compared to monitoring data for product use, however such monitoring data was not available in the literature. Air monitoring data for PCE were collected as background indoor air concentrations, i.e. not during product use. The North American residential background indoor maximum concentration was 0.17 mg/m^3, with central tendencies at or below 0.028 mg/m^3. Modeling estimates represent exposure during active product use and immediately after. The "moderate intensity user" estimates returned maximum 24-hour TWA indoor air concentrations for product users between 0.0097 and 166 mg/m^3 and bystander maximum 24-hour TWA indoor air concentrations between 0.009 and 32.2 mg/m^3. These estimated central values are in some instances below monitored central tendency background levels of PCE in residential air. Estimated central values for users and bystanders exceed the maximum monitored background concentration by three and two orders of magnitude, respectively, which is reasonable for direct product contact.

2.4.2.4 Consumer Article Exposure Scenarios

2.4.2.4.1 Literature Summary

PCE is a common dry cleaning solvent used to clean a wide variety of clothing and fabrics. Residual solvent is emitted from cleaned fabrics during transportation, storage and wear; and the introduction of dry cleaned articles into residences has been shown to increase indoor PCE. EPA identified concentrations of PCE in residential indoor air, personal air, and exhaled breath due to the controlled and monitored introduction of freshly dry cleaned garments in residential homes and apartments (results summarized in Table 2-88). These studies were conducted in the United States, China, and Japan, between 1980 and 1996. In all studies, the dry cleaned garments were placed in the bedroom closet, hall closet, or dresser drawer. Following introduction of the dry cleaned clothes, reported concentrations of PCE in the indoor air (excluding the storage closet or drawer) ranged from 0.93 to 692 $\mu g/m^3$. The maximum concentration was from a US study ((Howie 1981), conducted in a rural residential area outside of Washington DC) in which samples were collected from a closed bedroom after freshly dry cleaned garments were placed in the bedroom closet. Two other US studies reported slightly lower maximum concentrations, including 297 $\mu g/m^3$ in an experiment conducted in nine homes in NJ by Thomas (1991) and 195 $\mu g/m^3$ in a series of experiments conducted in one test house by Tichenor (1990). The data in Thomas (1991) showed that PCE levels can increase after bringing freshly dry cleaned clothes into the home (seven of the nine test homes showed PCE concentrations increases). This study includes a calculated source strength at four homes and determined that sources of PCE outside the house were not responsible for observed concentration increases after introduction of dry cleaned clothing. Personal air concentrations of PCE were higher when test subjects spent more time in the home, and wearing dry cleaned garments was a less important predictor of personal air concentration than the number of garments per home volume and number of hours spent in the home. The Tichenor (1990) study investigated concentrations over a seven-day period for multiple scenarios: storing clothes

with and without a plastic bag cover, and "airing out" the clothes before bringing them inside. A wide variation of concentrations was observed in this study. All the experiments, however, showed that PCE concentrations increased with the introduction of dry cleaned clothes, and levels dropped to near or below the detection limit after the clothes were removed. The authors also concluded that "airing out" of the clothing for short time periods does not reduce emissions. Concurrent to measuring concentrations in a test house, a chamber study was conducted, and modeled concentrations were calculated based on empirical data. Modeled concentrations were similar to measured concentration, reaching a maximum of approximately 100 µg/m^3. In the storage location within the homes, the maximum concentration (daily average) observed in this dataset was 2,900 µg/m^3, as reported by Tichenor (1990).

In addition to homes, a German study (Gulyas and Hemmerling 1990) investigated the concentration of PCE in a car after driving with a freshly dry cleaned down jacket placed in the car. Prior to introduction, the concentration inside the car was the same as background ambient concentrations (1 to 2 µg/m^3). Concentrations increased to a maximum 24,800 µg/m^3 at 108 minutes after article introduction. Another study, Park (1998), predicted PCE concentration in a car containing freshly dry cleaned clothes, using the EPA Indoor Air Quality model set to simulate driving a car. The model used emission data from Tichenor (1990) (initial emission rate of 1.2 mg·m^2·hr^{-1} and first order rate constant of 3.3 x 10^{-2} hr^{-1}) combined with air exchange rates experimentally determined in the study (1 per hour while stopped or 10 per hour while driving). Concentrations peaked at 2,300 µg/m^3 which occurred at the end of a 30-minute stopped/parking period.

Table 2-88 Concentrations (µg/m3) of PCE in indoor air, personal breathing zones, and breath from exposure studies with dry cleaned textiles placed in the home or automobile

Study Info	Media Type	Site Description	Detection Limit	Sample Size	DF	Min.	Mean	Max.	Data Evaluation Score
Residential Homes									
(Chao et al. 1999)[a] CN, 1996	24-hr (indoor air)	Hong Kong, CN; Residential Home (Site A) with dry cleaned clothes in closet. Four tests (each 7 days) in urban 5th floor apartment bedroom. Windows open and no AC unit.	--	28	1	4.6	--	76	Medium
		Hong Kong, CN; Residential Home (Site B) with dry cleaned clothes in closet. Four tests (each 7 days) in suburban 2nd floor apartment bedroom. Windows never opened and AC occasionally on.	--	28	1	21	--	494	Medium
		Hong Kong, CN; Residential Home (Site C) with dry cleaned clothes in closet. Four tests (each 7 days) in urban 10th floor apartment bedroom. Windows closed when AC on and windows open when AC off.	--	28	1	0.93	--	100	Medium
(Thomas et al. 1991)[b] US	12-hr (indoor air)	Bayonne and Elizabeth, NJ; Living rooms and bedrooms of nine homes. Six to ten 12-hr sampling periods per home. Two to ten sets of dry cleaned clothes were brought into the homes during the third monitoring period and stored based on the participants normal procedures. A resident wore a set of dry cleaned clothes during a later period. Number of maximum observations = 18.	--	--	--	--	--	8 - 297 (mean of max = 96±88)	High
	12-hr (personal air)	Bayonne and Elizabeth, NJ; Six to ten 12-hr sampling periods per home. Two to ten sets of dry cleaned clothes were brought into the homes during the third monitoring period and stored based on the participants normal procedures. The resident monitored wore a set of dry cleaned clothes during a later period. Number of maximum observations = 7.	1	--	--	--	--	8 - 303 (mean of max = 127±108)	High
	n/a (exhaled breath)	Bayonne and Elizabeth, NJ; Six to ten 12-hr sampling periods per home. Two to ten sets of dry cleaned clothes were brought into the homes during the third monitoring period and stored based on the participants normal procedures. A	--	--	--	--	--	9 - 61 (mean of max = 27±20)	High

Study Info	Media Type	Site Description	Detection Limit	Sample Size	DF	Min.	Mean	Max.	Data Evaluation Score
		breath sample was collected at end of each 12-hr monitoring period. The resident monitored wore a set of dry cleaned clothes during a later period. Number of maximum observations = 9.							
(Tichenor et al. 1990)[c] US	-- (indoor air)	Single story residential house with dry cleaning placed in closet. Closet door was closed and all other doors were open. HVAC fan operated. **Samples collected from the closet.**	1	--	--	--	100-2,900 (daily avg.) [model est. = 200-1,000]	--	High
		Single story residential house with dry cleaning placed in closet. Closet door was closed and all other doors were open. HVAC fan operated. **Samples collected from the bedroom**.	1	--	--	--	20-195 (daily avg.) [model est. = 30-100]	--	High
		Single story residential house with dry cleaning placed in closet. Closet door was closed and all other doors were open. HVAC fan operated. **Samples collected from the den.**	1	--	--	--	10-80 (daily avg.) [model est. = 15-50]	--	High
(Kawauchi and Nishiyama 1989)[d] JP	2-hr (indoor air)	Consumer homes in Japan (n=4). Dry cleaned clothes placed in chest of drawers. Samples collected from 2 to 4 pm during the weekday **inside chest of drawers**.	--	9	1	2.9	--	326.6	Medium
		Consumer homes in Japan (n=4). Dry cleaned clothes placed in chest of drawers. Room air samples collected from 2 to 4 pm during the weekday in **same room as chest of drawers**.	--	6	1	1.3	--	7.4	Medium
(Howie 1981) [e] US, 1980	24-hr (indoor air)	Washington, D.C., in late summer; Private home in rural residential area. Samples collected over 7 days after placing dry cleaned clothing in the house.	--	7	1	42.0	--	692	High
Automobiles									
(Gulyas and Hemmerling 1990) Germany, 1990		Vehicle with a dry cleaned down jacket placed in the car.	--	3	1	9,300	--	24,800	
(Park et al. 1998)	n/a	Modeled air concentration in vehicle with dry cleaned jacket. Assumptions: Volume = 3.24 m^3; surface area of jacket = 3.32 $m^{2;}$ initial emission rate of 1.2 mg/m^2/hr and first order rate constant of 3.3 x 10-2/hr (from Tichenor et al., 1990);	n/a	n/a	n/a	--	--	2,300	High

Study Info	Media Type	Site Description	Detection Limit	Sample Size	DF	Min.	Mean	Max.	Data Evaluation Score
		AER of 1/hr while stopped or 10/hr while driving							

Study Info: The information provided includes the HERO ID and citation; country and year samples collected.
Abbreviations: If a value was not reported, it is shown in this table as "--". ND = not detected at the reported detection limit. DF = detection frequency. NR = Not reported. CN = China. US = United States. JP = Japan. AC = air -conditioning.
Parameters: All statistics are shown as reported in the study.
[a] Results from this study (Chao et al. 1999) represent four tests at each of three test sites. Test 1: male clothes kept inside dry cleaner's original plastic bags. Test 2: male clothes kept outside dry cleaner's plastic bag. Test 3: male and female clothes kept inside drycleaner's plastic bags. Test 4: male and female clothes kept outside dry cleaner's plastic bags. Site A: min from Test 2 Day 7 and max from Test 4 Day 2. Site B: min from Test 1 Day 7 and max from Test 4 Day 1. Site C: min from Test 1 Day 2 and max from Test 4 Day 1.
[b] Results from this study (Thomas et al. 1991) represent a summary of the maximum indoor air, personal air, and breath concentrations measured at nine homes after introduction of dry cleaned clothes. Individual concentration values were not reported in the study. Indoor air (living area/bedroom): min from bedroom and max from living room. Concentrations before introduction of dry cleaned clothes were also measured for two 12-hr periods. Maximum concentrations ranged from 5 to 64 µg/m^3 in living room or bedroom, 8 to 35 µg/m^3 in personal air, and 3 to 30 µg/m^3 in breath.
[c] Results from this study (Tichenor et al. 1990)[c] represent a summary of daily average indoor air concentrations from a closet (with dry cleaned clothes), bedroom and den inside a residential home over seven days. The study provided the results (in graph form) for four tests performed during each day of sampling: (1) bag off; (2) bag on; (3) aired out; and (4) repeat of bag off. Closet: min from Test 1 Day 7 and max from Test 3 Day 1. Bedroom: min from Test 1 Day 7 and max from Test 3 Day 1. Den: min from Test 1 Day 7 and max from Test 3 Day 2. Model estimates were calculated using a source term based on small chamber data
[d] Results from this study (Kawauchi and Nishiyama 1989) represent indoor air concentrations from a chest of drawers and a bedroom in four homes.
[e] Results from this study (Howie 1981) represent measured indoor air concentrations over a 7 day period (24-hr samples).

Inhalation exposure to PCE in indoor air due to emissions from storage of dry cleaned articles was assessed for consumer users and bystanders, using measurements of PCE emissions from fabrics cleaned with older dry cleaning technologies (2nd and 3rd generation) as a worst-case emission scenario. Dermal exposure due to direct skin contact with recently dry cleaned fabrics during article wear was assessed for consumer users, for older and more modern dry cleaning technologies (2nd-5th generation). Preliminary estimations of inhalation exposure to PCE emissions during article wear was found to be much lower than either the storage or dermal exposure scenarios and was not further pursued. Dry cleaning consumer exposures could be cumulative for the user, including inhalation exposure during transport of dry cleaned articles in an automobile, inhalation exposure from dry cleaned articles stored in the home, and inhalation and dermal exposure from wearing dry cleaned articles.

Modeling Approach

Dermal exposure to PCE resulting from direct skin contact with recently dry cleaned articles, i.e. wearing dry cleaned clothing, was modeled with CEM. Inhalation exposure to PCE emitted from recently dry cleaned articles stored in a home was modeled using EPA's Multi-Chamber Concentration and Exposure Model (MCCEM). MCCEM is a higher tier model and utilizes chemical-specific emissions data to estimate air concentrations and inhalation exposure.

2.4.2.4.2 Dermal Exposure to Recently Dry cleaned Articles

EPA's CEM 2.1 dermal sub-model A_DER2: Dermal Dose from Skin Contact with Article, as presented in the CEM user guide (U.S. EPA 2019b) was used to model dermal exposure to PCE from direct contact with recently dry cleaned articles. This model calculates dermal exposure due to migration of a chemical within an article to the skin via direct article contact.

Residual Mass

Residual mass of PCE remaining in recently in dry cleaned articles can be thought of as the chemical "pool", or the amount of chemical potentially available for dermal exposure. Residual PCE mass was calculated from two sources (see Section 2.4.2.4.2) The first data source, based on Tichenor (1990) applies to 1st, 2nd and 3rd generation dry cleaning machines, due to the date the study was conducted[14]. Tichenor (1990) conducted chamber tests and test house studies to measure emission rates and emission half-lives of PCE from various commercially dry cleaned fabrics. Residual PCE was calculated using a simple exponential model based on measured PCE emissions. The second data source, based on Sherlach (2011), likely applies to 4th and 5th generation dry cleaning machines, due to the date the study was conducted. Sherlach (2011) extracted perchloroethylene residues from commercially dry cleaned fabrics after a single cleaning event, multiple cleaning events, and after one week of storage. Cotton, Polyester and wool fabric were shown to accumulate PCE with subsequent dry cleaning cycles. Multiple dry cleaning cycle estimates were included to model a high-end user (albeit using more modern commercial dry cleaners) who has their wool suit dry cleaned weekly, such that residual PCE

[14] Perchloroethylene related NESHAPs from 1993 and 2006 banned 1st generation machine and required more modern technologies for new dry cleaning machines but allowed certain 2nd and 3rd generation machines to continue to be used. Given the age of 2nd generation dry cleaning technology, it is likely that only a very small number of these machines are still in use today, but EPA cannot definitively rule out the possibility of their continued use. Similarly, an unknown but likely small number of 3rd generation dry cleaning machines may still be in use.

concentrations become saturated in the fabric (Sherlach (2011) showed that wool continued to accumulate PCE for at least 6 cleaning cycles). Residual PCE was calculated using reported residual concentration data and a simple emission model. Residual mass of PCE in dry cleaned fabrics was calculated for the first three days after the dry cleaning event[15]. Details of the calculation can be found in the *Draft Risk Evaluation for Perchloroethylene Supplemental Information for Consumer Exposure* (U.S. EPA 2020f).

Table 2-89. Cumulative mass released for number of days post dry cleaning and number of hours the garment was worn (10 hr), based on Tichenor (1990) and Sherlach (2011). Values were used as modeling inputs for the residual pool of PCE available for exposure.

Data Source (est. machine generation)	Fabric Type	Dry cleaning events	Average Residual Mass (mg) Time since article was dry cleaned		
			1 day	2 days	3 days
Tichenor (1990) (1st -3rd)	Polyester-wool blend	Single	105	81	63
Sherlach (2011)	Polyester[1]	Single	18	14	11
Sherlach (2011)	Wool[2]	Repeat[3]	58	45	35

[1] Based on average maximum measured PCE concentration in polyester fabric samples after single cleaning event
[2] Based on average maximum measured PCE concentration in wool fabric samples after multiple cleaning events
[3] Residual value used to parameterize model is based on 6th cycle data for wool from Sherlach (2011))

Factors affecting the value of residual mass include fabric type, number and proximity of dry cleaning events, total number of dry cleaned articles, total article surface area, the type (generation) of dry cleaning machine used and number of days elapsed since the fabric was dry cleaned. Different fabrics retain different amounts of PCE, the values estimated here are based on measured emissions from a variety of fabrics reported in Tichenor (1990) and Sherlach (2011).

Dry cleaned article parameters

An article with a surface area of $1m^2$ and $1.5m^2$ was assumed to calculate residual mass, with a wearer donning the garment(s) 1 to 3 days after dry cleaning, for a total duration of 10 hours (assumption of 8-hour work day, plus commute). An average fabric thickness of 0.1 cm was assumed based on the fabrics used in the Tichenor (1990) and Sherlach (2011) studies and thickness measurements of various types of fabrics (based on Küçük and Korkmaz (2012); Marolleau (2017); Van Amber (2010). Thickness of fabric is inversely proportional to dermal dose (as thinner fabrics require less diffusion distance to reach skin). A single, multi-hour contact per day was assumed for acute exposure.

CEM Dermal Results

[15] Measured PCE emissions from recently dry-cleaned fabrics were fit to a simple exponential model to describe the rate of emission, and thus calculate the residual mass of PCE remaining in the fabric at a certain time after the dry cleaning event. Residuals were calculated for days 1-3 post-cleaning, as 3 days was roughly one half-life in the fitted decay curve. A consumer that wore a garment more than three days after dry cleaning would have less potential dermal PCE exposure, although elevated air concentrations in the home and inhalation exposures would remain unchanged.

Dermal exposure to PCE due to direct contact with recently dry cleaned articles was evaluated for 1-3 days after dry cleaning, assuming different dry cleaning technologies and for four article thickness values, for both half-body (1 article) and full body (2 articles) exposure (Table 2-90). ADR results for half-body exposure ranged from 5.1E-02 to 0.5 $mg \cdot kg^{-1} \cdot day^{-1}$. ADR results for full-body exposure ranged from 0.2 to 1.5 $mg \cdot kg^{-1} \cdot day^{-1}$.

Table 2-90. Dermal exposure results to recently dry cleaned articles, based on CEM modeling

Assumed dry cleaning technology	Dry Cleaning Events	Days After Dry Cleaning	Half-body Dermal ADR (Surface Area 1 m^2, SABW 122.9) mg/kg-day	Full-body Dermal ADR (Surface Area 1.5 m^2, SABW 245.9) mg/kg-day
2nd and 3rd generation	Single	1	0.5	1.5
		2	0.3	1.1
		3	0.3	0.9
4th and 5th generation	Single	1	8.7E-02	0.3
		2	6.7E-02	0.2
		3	5.1E-02	0.2
4th and 5th generation	Repeat[1]	1	0.3	0.8
		2	0.2	0.6
		3	0.2	0.5

[1] Based on maximum average PCE concentration in wool after 6 dry cleaning cycles from Sherlach (2011); PCE concentration was still increasing in wool fabric after 6 cycles and had not yet reached saturation.

Confidence in the selected model and default parameters is medium to high for dermal exposure due to wearing recently dry cleaned articles. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in dermal model default parameters is high due to the high quality of source data. Residual PCE remaining in dry cleaned clothing was determined from high quality test chamber emission data from early generation dry cleaning machines (dates from 1990), and high-quality analytical data on PCE residuals from more modern dry cleaning technologies, which leave less residual PCE in dry cleaned fabrics. CEM's article diffusion model is sensitive to the thickness of material selected. An effort was made to best match the fabric type and assumed article thickness of the Tichenor (1990) and Sherlach (2011) test swatches to minimize over- or underestimating residual PCE. The quantity of residual PCE in articles varies based on fabric type and how much time has elapsed between subsequent dry cleaning events. Dermal exposure results may differ for other types of fabrics. The overall confidence in dermal exposure estimations due to wearing recently dry cleaned articles is medium to high with possible overestimation or underestimation based on differences in PCE retention in various fabric types and frequency of dry cleaning events.

2.4.2.4.3 Inhalation Exposure to Recently Dry cleaned Articles

MCCEM Modeling Approach

Inhalation exposure due to emissions of PCE from recently dry cleaned clothing was modeled using EPA's Multi-Chamber Concentration and Exposure Model (MCCEM, (U.S. EPA 2019e)) single-exponential emission model and emissions data available in published literature.

Tichenor (1990) measured PCE air concentrations due to emissions from recently dry cleaned articles in a test house (EPA's Air and Energy Engineering Research Laboratory, Indoor Air Quality test home). It is assumed, given the date of the study, that results likely reflect commercial cleaners using 2nd or 3rd generation dry cleaning machines. Newer technologies are presumed to result in lower residual PCE concentrations in dry cleaned fabrics, but EPA cannot definitely say that older model machines have been completely replaced with 4th generation (or later) technologies. As such, Tichenor (1990) was used for model parameterization as a high end estimate, and based on risk results (see Section 4.2.4.16), further modeling for 4th and 5th generation technologies was not done. Test house measurements were conducted by placing freshly dry cleaned garments (wool skirt, two polyester/rayon blouses and a two-piece wool-blend suit) in a bedroom closet. Indoor air samples were collected at three locations (closet, bedroom, and den), four times a day.

EPA used this data as a modeling basis to parameterize the MCCEM indoor air model for a generic residential house (Table 2-91). The EPA/Tichenor test house layout, along with reported house volume and whole-house air exchange rate (Chang et al. 1998; Tichenor et al. 1990) were used as the basis for a generic home. EPA assumed the zone of use to be a bedroom closet containing dry cleaned articles, defined as the near-field volume. The bedroom containing the closet was defined as the far-field volume. The third zone was termed the "rest of the house" (ROH) and included all areas outside of the bedroom. A user in this scenario was assumed to be a person who places dry cleaned articles in their bedroom closet and spends some short amount of time dressing in that closet, twice per day. The CEM activity pattern for a stay-at-home adult was selected as the basis for an MCCEM adult "user" pattern, with an addition of 5 minutes spent in the closet (near-field) in the morning and in the evening. A bystander in this scenario was considered to be a youth or child that remained in the rest of the house. PCE air concentrations were modeled over a ten-day period. Further details of the MCCEM model parameterization are given in the *Draft Risk Evaluation for Perchloroethylene Supplemental Information for Consumer Exposure* (U.S. EPA 2020f).

Table 2-91. Emission parameters for MCCEM modeling of PCE emissions from recently dry cleaned clothing.

Parameter Name	Value	Source
First order decay rate	0.011 hr^{-1}	Scaled from Tichenor (Tichenor et al. 1990)
Emission rate	7.38 mg/hr	Scaled from Tichenor (Tichenor et al. 1990)
Article surface area[1]	12.6 m^2	Scaled from Tichenor (Tichenor et al. 1990)
MCCEM model house volume	446 m^3	Scaled from Chang (1998)
Closet volume (near-field)	5 m^3	Scaled from Chang, (1998)

Parameter Name	Value	Source
Near-field: far-field air flow rate	8 m^3/hr	Scaled from Chang, (1998)
Whole house air exchange rate	0.45 hr^{-1}	CEM v2.1 default[2]
Length of run	240 hr (10 days)	EPA choice
Background concentration	0 mg/m^3	EPA choice

[1]An article surface area of 12.6 m^2 corresponds to roughly seven articles of adult clothing
[2]EPA's Consumer Exposure Model version 2.0 (2017a)

MCCEM Inhalation Results

Peak PCE air concentrations and maximum 24-hour TWAs for the dry cleaned article storage scenario are summarized in Table 2-92 and Table 2-93. Maximum PCE air concentrations occurred in the closet roughly 4 hours after placement of clothing ($9.67x10^{-1}$ mg/m^3). Air concentrations in the surrounding bedroom peaked roughly 7 hours after clothing placement ($8.72x10^{-2}$ mg/m^3), and 10 hours after placement for the rest of the house ($2.98x10^{-2}$ mg/m^3). The maximum 24-hour TWA PCE air concentrations were $7.24x10^{-2}$ mg/m^3 for the user and $2.33x10^{-2}$ mg/m^3 for the bystander. Indoor air concentrations of PCE remained elevated above pre-exposure levels for the duration of the 10-day modeling window.

Table 2-92. MCEEM calculated PCE air concentrations for storage of recently dry cleaned articles in a generic house.

Zone	Maximum Concentration (mg/m^3)	Time Elapsed at Maximum (hr)	Hour 10 Concentration (mg/m^3)
Closet (near-field)	9.7E-01	3.85	7.3E-02
Bedroom (far-field)	8.7E-02	7.27	6.9E-03
ROH	3.0E-02	9.62	2.4E-03

Table 2-93. MCEEM calculated PCE maximum 24-hour TWAs for storage of recently dry cleaned articles in a generic house.

Exposure Receptor	Maximum 24-hour TWA Concentration (mg/m^3)
User (stay-at-home adult)	7.2E-02
Bystander (stay-at-home child or youth)	2.3E-02

Confidence in the selected model and default parameters is medium to high for inhalation exposure during storage of recently dry cleaned articles in a home closet. Estimated exposures represent a higher-end scenario where articles have been cleaned at a commercial dry cleaner still employing older technology. The selected model underwent peer review, was designed explicitly for the purpose of this type of estimation and applied in the manner intended. Confidence in the parameterization of the

inhalation emission scenario is high, as there was a high-quality test chamber emission data and test house monitoring data available, however the total number of studies was limited. The master bedroom room was selected as the room of use for this scenario. This may underestimate bystander inhalation exposure, based on activity patterns, relative to storage of dry cleaned articles in a common area of the house. Residual PCE remining in dry cleaned clothing was determined from high quality test chamber emission data, using emissions parameters based on older (2^{nd} and 3^{rd} generation) dry cleaning technologies. More modern dry cleaning technologies presumably leave less residual PCE in dry cleaned fabrics. Based on risk results (see Section 4.2.4.16), further modeling for more modern dry cleaning technologies was unnecessary. The quantity of residual PCE in articles varies based on fabric type and how much time has elapsed between subsequent dry cleaning events. Inhalation exposure results may differ for other types of fabrics, for more or less frequently dry cleaned articles and based on the number of dry cleaned items stored. The overall confidence in inhalation exposure estimations due to storage of recently dry cleaned articles in a home is medium to high with possible overestimation based on the availability of more modern dry cleaning technologies, and possible overestimation or underestimation based on differences in PCE retention in various fabric types, frequency of dry cleaning events and number of dry cleaned items stored.

2.4.2.4.4 Consumer Article Exposure Summary

Consumer exposure to PCE due to off-gassing from recently dry cleaned articles was evaluated for two scenarios, direct dermal contact with clothing, and inhalation exposure from article storage in a home closet. A modeling approach was taken, based heavily on empirical data, to estimate dermal and inhalation exposures. No direct measurements were found for consumer dermal exposure to PCE from dry cleaned fabrics. Dermal exposure estimates ranged from 5.1E-02 to 1.5 mg/kg/day. Measurements of PCE concentrations in indoor air from storage of recently dry cleaned articles are in good agreement with modeling results. Elevated PCE concentrations measured in bedroom air, shortly after dry cleaned articles were stored in a dresser or closet, were reported as between 9.3E-03 and 0.7 mg/m^3, with modeling estimates for maximum PCE air concentration in the bedroom after article storage of 8.7E-02 mg/m^3. Dry cleaning consumer exposures could be cumulative for the user, including inhalation exposure during transport of dry cleaned articles in an automobile, inhalation exposure from dry cleaned articles stored in the home, and inhalation and dermal exposure from wearing dry cleaned articles.

2.4.2.5 Other Consumer Uses

Additional potential consumer exposures to PCE were identified, including off-gassing from new clothing and apparel, due to use of PCE in the textile industry; use of coin operated dry cleaning machines; and emissions from photocopy and printing equipment. Available data is summarized below. Due to limited available information on these conditions of use, risk for these scenarios will not be further assessed.

2.4.2.5.1 New Clothing/Textile Industry

PCE is used to remove spinning oils, lubricants and naturally occurring dirt and oils from yarn and fabric used in clothing manufacturing, and as a carrier solvent for dyes in the textile industry (Morrison and Murphy 2013). While a high percentage of PCE applied to textiles during manufacturing is expected volatize, there is potential for consumer exposure due to off-gassing from new textiles and fabrics. Chan (2014) measured PCE in indoor air in apparel stores, with a detection frequency of 30% (120 samples), and reported mean air concentration of 0.2 $\mu g/m^3$.

2.4.2.5.2 Coin Operated Dry Cleaners

Howie (1981) measured indoor air PCE concentrations in coin-operated dry cleaning facilities in the United States (6 facilities). PCE was detected in 100% of collected samples, with air concentration range from 508 to 94984 µg/m^3. EPA was not able to determine if coin operated dry cleaning machines were still in use in the United States.

2.4.2.5.3 Print Shops

Stefaniak (2000) measured PCE in area and personal breathing zone air samples, in three commercial print shops in Baltimore, MD. A total of 17 area samples and 4 personal breathing zone samples were collected, with detection frequencies of 94% and 100%, respectively. PCE concentrations in personal breathing zone samples ranged from 0.7 to 3.4 µg/m^3, and in area samples from non-detection to 21 µg/m^3.

Ryan (2002) measured PCE in indoor air in a printmaking art studio in a university building in the United States. 18 samples were collected, with reported PCE concentration mean of 0.4 µg/m^3.

Kiurski (2016) measured elevated PCE levels in a small commercial photocopy shop in Serbia, containing two copiers and a printer. PCE concentrations were attributed to the usage of photocopying equipment. A total of 225 samples were collected, with a PCE detection frequency of 64%, and measured concentration rage of 6.8 to 96341 µg/m^3.

Kowalska and Gierczak (2013) measured volatile emissions from disintegrated office equipment (11 items). PCE was detected most frequently in office equipment samples, with 68.7% detection.

2.4.2.6 Consumer Exposure Assumptions and Key Sources of Uncertainty

Overall, there is medium to high or high confidence in the consumer inhalation exposure modeling approach and results. 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 (U.S. EPA 2019b) 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 by EPA 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 (U.S. 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 (see EPAs Preliminary Information on Manufacturing, Processing, Distribution, use and Disposal: Tetrachloroethylene (2017f)). 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 parts 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 medium or medium to high confidence. The processes and inputs described for the inhalation scenarios above are also valid for the dermal exposure scenarios. While the model used for product dermal exposure estimates was the same as used for the product inhalation exposure estimates, there is overall medium (vs. high for inhalation) confidence in the model used due to the used dermal submodel. As described in Section 2.4.2.2.2, the evaluation of dermal exposures used a permeability submodel, which ignores evaporation and thus is only applicable to use scenarios for which evaporation is limited, such as during immersion or when handling a solvent-soaked rag. As a result, model results may overestimate dermal exposure when evaporation is significant, or the actual contact volume cannot be modeled using a constant bath assumption. This evaluation assumes consumer exposure under each condition of use is not chronic in nature due to the infrequent use and short duration of use for a given product. There is a medium uncertainty associated with this assumption because, although information found during EPA's systematic review process supports infrequent use and short durations of use, there is a growing consumer practice to complete projects or activities as do it yourselfers. Do it yourself activities could lead to an increased frequency of product use as well as using more than one product containing a chemical of concern within a given day. These and other factors associated with do it yourself activities could result in underestimating consumer exposure concentrations modeled in this evaluation for the do it yourself consumer.

2.4.3 Potentially Exposed or Susceptible Subpopulations

TSCA requires the risk evaluation "determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost of other non-risk factors, including an unreasonable risk to a potentially exposures of susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." 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."

During problem formulation (U.S. EPA 2018d), EPA identified potentially exposed or susceptible subpopulations for further analysis during the development and refinement of the life cycle, conceptual models, exposure scenarios, and analysis plan. In this section, EPA addresses the potentially exposed or susceptible subpopulations identified as relevant based on *greater exposure*. EPA addresses the subpopulations identified as relevant based on *greater susceptibility* in Section 3.2.5.2.

In developing the draft risk evaluation, the EPA analyzed the reasonably available information to ascertain whether some human receptor groups may have greater exposure than the general population to the hazard posed by PCE. Exposures of PCE would be expected to be higher amongst groups living near industrial facilities, groups with PCE containing products in their homes, workers who use PCE as part of typical processes, and groups who have higher age and route specific intake rates compared to the general population.

Of the human receptors identified in the previous sections, EPA identifies the following as potentially exposed or susceptible subpopulations due to their greater exposure to PCE and considered them in the risk evaluation:

Workers and Occupational Non-Users (ONUs)

EPA reviewed monitoring data found in published literature including both personal exposure monitoring data (direct exposure) and area monitoring data (indirect exposures) and identified data sources that contain measured monitoring data and or/estimated data for the various conditions of use (including import and processing of PCE). Exposure estimates were developed for users (males and female workers of reproductive age) exposed to PCE as well as non-users or workers exposed to PCE indirectly by being in the same work area of the building. Also, adolescents and female workers of reproductive age (>16 to less than 50 years old) were also considered as a potentially exposed or susceptible subpopulations

Consumers/Product Users and Bystanders Associated with Consumer Use

PCE has been identified as being used in products available to consumers. Section 2.4.2.2 provides an overview of exposure pathways considered for the consumer assessment. Furthermore, EPA identified consumers and bystanders associated with use of PCE containing consumer products as a potentially exposed and susceptible subpopulation due to greater exposure. For example, higher-intensity users (i.e., those using consumer products for longer durations and in greater amounts) were considered and evaluated. In addition, consumers are considered to include children and adults over age 11, but bystanders in the home exposed via inhalation are considered to include any age group, from infant to adult, including pregnant women and/or women of reproductive age. However, only some individuals within the general population may use these products. Therefore, those who do use these products are a potentially exposed or susceptible subpopulation due to greater exposure. Exposures for these subpopulations are considered and/or evaluated in Section 2.4.2.2.

In developing dermal exposure scenarios, EPA quantified age and sex-specific differences. For PCE, exposure scenarios that involve potentially exposed or susceptible subpopulations considered age-specific behaviors, activity patterns, and exposure factors unique to those subpopulations. EPA used the Exposure Factors Handbook (U.S. EPA 2011a) to inform body weights, intake rates, and body surface areas for children and adults. Distinct dermal exposure estimates are provided for are provided for adults (including women of reproductive age) and children (Section 2.4).

For occupational exposures, EPA assessed exposures to workers and ONUs from all PCE conditions of use (Section 2.4.1). Table 2-94 presents the percentage of employed workers and ONUs whom may experience either greater exposure or biological susceptibility within select industry sectors relevant to PCE conditions of use. The percentages were calculated using Current Population Survey (CPS) data for 2017 (U.S. BLS 2017). CPS is a monthly survey of households conducted by the Bureau of Census for the Bureau of Labor Statistics and provides a comprehensive body of data on the labor force characteristics. Statistics for the following subpopulations of workers and ONUs are provided: adolescents, men and women of reproductive age, and the elderly. For the purpose of this assessment, EPA considers “reproductive age” as age >16 to less than 50 years old.

As shown in Table 2-95, men make up the majority of the workforce in manufacturing sectors. In other sectors, women (including those of reproductive age and elderly women) make up nearly half of the workforce. Adolescents are generally a small part of the total workforce. Table 2-95 presents further breakdown on the percentage of employed adolescents by industry subsectors. As shown in the tables, they comprise only 1.2% percent of the manufacturing workforce, and only as high as 3.7% for other services such as dry cleaning that fall under a COU for PCE.

Table 2-94. Percentage of Employed Persons by Age, Sex, and Industry Sector

Age group	Sex	Manufacturing	Wholesale and Retail Trade	Professional and Business Services	Other Services
Adolescent (16-19 years)	Male	0.8%	3.0%	0.7%	1.4%
	Female	0.4%	3.2%	0.5%	1.7%
Reproductive age[a] (16-54 years)	Male	52.9%	42.8%	44.4%	35.2%
	Female	22.2%	35.4%	32.8%	38.4%
Elderly (55+)	Male	17.5%	12.3%	13.4%	13.1%
	Female	7.3%	9.6%	9.4%	13.3%

[a] The World Health Organization defines women of reproductive age as ages 15-49 (WHO 2006b)While statistics on pregnant women are not reasonably available, Labor Force Statistics from the Current Population Survey provides data on the number of employed female workers by age group, which allows for determination of the number of employed women of reproductive age. The Bureau of Labor Statistics breaks apart age groups such that age 15 is combined with children, and ages 44-54 are clustered (U.S. BLS 2017). Percentages were calculated using CPS Table 14, "Employed persons in nonagricultural industries by age, sex, race, and Hispanic or Latino ethnicity", for ages 16-64.

Table 2-95. Percentage of Employed Adolescent by Detailed Industry Sector

Sector	Subsector	Adolescent (16-19 years)
Manufacturing	All	1.2%
Wholesale and retail trade	Wholesale trade	1.4%
Professional and business services	Waste management and remediation services	0.9%
Other services	Repair and maintenance	3.1%
	Dry cleaning and laundry services	3.7%

Source: (U.S. BLS 2017). Percentage of adolescent calculated using CPS table 18b, "Employed persons by detailed industry and age."

The CPS uses 2012 Census industry classification, which was derived from the 2012 NAICS. The Census classification uses the same basic structure as NAICS but is generally less detailed. PCE conditions of use fall under the following Census industry sectors:

Manufacturing

The Manufacturing sector comprises establishments engaged in the mechanical, physical, or chemical transformation of materials, substances, or components into new products. Establishments in the sector are often described as plants, factories, or mills. For PCE, this sector covers most conditions of use that occur in an industrial setting, including: Manufacturing, Processing as a Reactant, Formulation of Aerosol and Non-Aerosol Products, the vast majority of facilities likely engaged in Vapor Degreasing (all degreaser types), Cold Cleaning, Metalworking Fluids, Adhesives, Sealants, Paints and Coatings, Other Industrial Uses, Industrial Processing Aids and Printing and Copying. This sector also covers cement manufacturing facilities that may burn waste containing PCE for energy recovery. Also – Printing and Copying worker information may also be captured under the Information sector (see below).

Wholesale and Retail Trade

The wholesale trade sector comprises establishments engaged in wholesaling merchandise, generally without transformation, and rendering services incidental to the sale of merchandise. Wholesalers normally operate from a warehouse or office. This sector likely covers facilities that are engaged in the repackaging PCE or products and formulations containing PCE. The retail trade sector comprises establishments engaged in retailing merchandise and rendering services incidental to the sale of merchandise.

Professional and Business Services

This sector comprises establishments that specialize in a wide range of services. This sector covers waste management and remediation services, which includes establishments that may handle, dispose, treat, and recycle wastes containing PCE.

Other Services

This sector comprises establishments engaged in providing services not specifically provided for elsewhere in the classification system. For PCE, this sector covers the vast majority of commercial repair and maintenance facilities that are likely to use PCE for Aerosol Applications (spray degreasing). The sector also covers the use of PCE in dry cleaning.

The EPA IRIS Assessment for PCE (U.S. EPA 2012c) also identified the developing fetus as potentially exposed, as well as infants consuming breastmilk, particularly for mothers with occupational exposure to PCE or exposure due to proximity to industrial or commercial sources (U.S. EPA 2012c). Infants fed by formula may also experience increased PCE exposure if PCE is present in drinking water supplies (U.S. EPA 2012c).

3 HAZARDS

3.1 Environmental Hazards

3.1.1 Approach and Methodology

EPA reviewed potential environmental health hazards associated with PCE. EPA identified the following sources of environmental hazard data for PCE: European Chemicals Bureau (ECB) EU Risk Assessment Report Tetrachloroethylene, Part 1 - environment (ECB 2005) and World Health Organization (WHO) Concise International Chemical Assessment Document 68; Tetrachloroethylene WHO (WHO 2006a).

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 2018b). The data quality evaluation results indicated the quality of the studies is mostly 'high' and 'moderate', and these studies were used to characterize the environmental hazards of PCE. The data evaluation results for PCE environmental hazard are summarized in Table 3-1.

3.1.2 Hazard Identification

Toxicity to Aquatic Organisms

EPA assigned an overall quality level of high, medium or low to 30 acceptable studies. These studies contained relevant aquatic toxicity data for fish, aquatic invertebrates, and aquatic plants. As shown in Table 3-1, EPA identified 10 aquatic toxicity studies as the most relevant for quantitative assessment. Four of the 10 studies were carried forward for characterizing the potential environmental risks from PCE. The rationale for selecting these studies is provided in Section 3.1.3 Weight of Scientific Evidence.

Table 3-1. Ecological Hazard Characterization of PCE for Aquatic Organisms

Duration	Test organism	Endpoint	Hazard value[1] (mg/L)	Effect Endpoint	Geometric Mean[2] (mg/L)	References	Data Quality Evaluation Ratings
Acute	Fish	LC_{50}	4.82 – 28.1	Mortality	12	(Horne et al. 1983; Call et al. 1979)	High
	Aquatic invertebrates	LC/EC_{50}	2.49 – 18.1	Immobilization	6.7	(Niederlehner et al. 1998; Richter et al. 1983; Call et al. 1980)	High
Chronic	Fish	ChV	0.5-1.4	Mortality	0.84	(Ahmad et al. 1984)	High
	Aquatic invertebrates	ChV	0.37 – 0.67	Growth	0.5	(Call et al. 1983; Richter et al. 1983; Hollister et al. 1968)	High
	Algae	EC_{50}	3.64 - >500	Biomass		(Brack and Rottler 1994; Hollister et al. 1968)	High
		NOEC/ LOEC	0.01 - 0.02	Mortality	1.4E-2	(Labra et al. 2010)	Medium

[1] Values in the tables are presented as reported by the study authors
[2] Geometric mean of definitive values only (i.e. > 48 mg/L was not used in the calculation).

Aquatic Environmental Hazards from Acute Exposures to PCE

Fish: EPA assigned an overall quality level of high for two acute (96-hour; flow-through) fish toxicity studies, which evaluated the median lethal concentrations (LC50s) of PCE to *Oncorhynchus mykiss* (rainbow trout) or *Menidia beryllina* (inland silverside) (Horne et al. 1983; Call et al. 1979). The acute 96-hour LC50 values for fish range from 4.82 mg/L (Call et al. 1979) for *O. mykiss* to 28 mg/L (Horne et al. 1983) for inland silverside *M. beryllina*. As previously identified in the Problem Formulation document, the acute 96-hour LC 50 value of 4 mg/L (Smith et al. 1991) for flagfish (*Jordanella floridae*) was determined to be a reporting error from the study.

Aquatic Invertebrates: Three studies were assigned an overall quality level of high for acute (48-hour) toxicity to aquatic invertebrates *Ceriodaphnia dubia* and *Daphnia magna*. The studies indicate the 48-hour EC/LC50 values range from 2.5 mg/L (Niederlehner et al. 1998) to 18 mg/L (Richter et al. 1983; Call et al. 1980). The geometric mean was calculated from the 48-hour EC50 and LC50 values as 6.7 mg/L. Other salt water aquatic invertebrate toxicities range from 96-hour LC 50 of 2.9 mg/L (Hollister et al. 1968) for mysid shrimp (*Mysidopsis bahia*) to 24-hour LC 50 of 23 mg/L (Sanchez-Fortun et al. 1997) for Brine shrimp (*Artemia salina*). The 48-hour acute toxicity to midge larvae (*Tanytarsus dissimilis*) show LC 50 of 31 mg/L and EC50 of 7.0 mg/L (Call et al. 1979).

Aquatic Environmental Hazards from Chronic Exposures to PCE:

Fish: A single chronic 32-day toxicity study on exposure of *Pimphales promelas* (fathead minnow) to PCE was assigned an overall quality level of high (Ahmad et al. 1984). The reported NOEL - LOEL values of 0.5 - 1.4 mg/l, respectively, based on growth and mortality of *P. promelas* exposure to PCE (Ahmad et al. 1984). The geometric mean was used to calculate the chronic toxicity value of 0.84 mg/L.

Aquatic Invertebrates: Three studies were assigned an overall quality level of high for chronic (28-day) toxicity to aquatic invertebrates *Daphnia magna* (Richter et al. 1983; Call et al. 1980), *Americamysis bahia* (opossum shrimp) (Hollister et al. 1968) from exposure to PCE. The *D. magna* 28-day study reported a NOEC value of 0.5 mg/L using reproduction based on measured concentrations (Richter et al. 1983; Call et al. 1980). The 28-day *A. bahia* reported NOEC value of 0.4 mg/L and LOEC of 0.7 mg/L (Hollister et al. 1968). The geometric mean was calculated from the NOEC and LOEC values to derive the chronic toxicity value of 0.5 mg/L.

Aquatic Plants: Three studies were assigned an overall quality level of high for EC_{50} endpoint (Brack and Rottler 1994; Hollister et al. 1968) and medium for NOEC/LOEC (Labra et al. 2010) from exposure to PCE. The algal toxicity 72/96-hr EC50 values were 3.6 for *Chlamydomonas reinhardtii* (Brack 1994) to greater than 500 mg/L for fresh and saltwater algae (Hollister, 1968) based on biomass and abundance. The algal species in the Hollister study were not specified. The most conservative toxicity values were reported for *Pseudokirchneriella subcapitata* (green microalgae) 72-hour study using NOEC - 1.0E-2 mg/L and LOEC - 2.0E-2 mg/L based on mortality (Labra et al. 2010). The geometric mean was calculated from the NOEC and LOEC values to derive the algal toxicity value of 1.4E-2 mg/L.

As noted in the Problem Formulation, EPA did not include PCE hazard toxicity to terrestrial mammals in this risk evaluation. Observed effects in laboratory mammals that occurred at much higher concentrations that have been measured or are predicted to occur in the environment. Additionally, as noted in Section 2.1, the bioconcentration factor and bioaccumulation potential of PCE is low. Therefore, it is unlikely that adverse effects will occur on the terrestrial mammalian exposure pathway (Eu 2001).

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 2018b).

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 2018b). While integrating environmental hazard data for PCE, 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 PCE 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 (U.S. 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 (U.S. EPA 1998).

To calculate COCs, EPA derived geometric means for each trophic level that had comparable toxicity values (e.g., multiple EC_{50}s measuring the same or comparable effects from various species within a trophic level). EPA did not use non-definitive toxicity values (e.g., EC_{50} > 48 mg/L) to derive geometric means because these concentrations of PCE were not high enough to establish an effect on the test organism.

To assess aquatic toxicity from acute exposures, data for two taxonomic groups were available: fish, and aquatic invertebrates. For each taxonomic group, data were available for multiple species, and geometric means were calculated as shown in Table 3-1. The geometric mean of the EC_{50}s and LC_{50}s for aquatic invertebrates, 6.7mg/L, represented the most sensitive toxicity value derived from each of the two 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.

To assess aquatic toxicity from chronic exposures, data for two taxonomic groups were described in the acceptable literature: fish, and aquatic invertebrates. Aquatic invertebrates were also the most sensitive taxonomic group for chronic exposures. The chronic 72-hour NOEC = 0.01 mg/L and LOEC = 2.0E-2 mg/L values were 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.

To assess the toxicity of PCE to algae, data from three species were available from studies that EPA assigned an overall quality level of high and medium. EC_{50}s measuring biomass ranged from 3.6 mg/L to >500 mg/L. A NOEC = 1.0E-2 mg/L and LOEC = 2.0E-2 mg/L was also reported. Because these values varied by greater than an order of magnitude, EPA used the NOEC/LOEC mortality endpoint for the most sensitive algal species to represent algae as a whole. These values, from one medium quality algae study, 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, PCE does not bioaccumulate in biological organisms. Therefore, EPA did not assess hazards to aquatic species from trophic transfer and bioconcentration or accumulation of PCE.

3.1.4 Concentrations of Concern (COC)

EPA calculated the COCs for aquatic species based on the environmental hazard data for PCE, using EPA methods (U.S. EPA 2013, 2012b). While there was data representing 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 PCE 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 PCE, EPA derived an acute and a chronic COC for fish and aquatic invertebrates. Algae was assessed separately and not incorporated into acute or

chronic COCs, because durations normally considered acute for other species (e.g. 48, 72 hours) can encompass several generations of algae.

After weighing the scientific evidence and selecting the appropriate toxicity values from the integrated data to calculate acute, chronic, and algal COCs, EPA applied an assessment factor (AF) according to EPA methods (U.S. EPA 2013, 2012b), when possible. An assessment factor (AF) is applied to the acute and chronic hazard endpoints for aquatic species to calculate a Concentration of Concern (COC) for use in the screening-level analysis of environmental hazards. 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. 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. For chronic COCs, an AF of 10 is used. The COC for algae, where multiple generations can be present over the course of a standard toxicity test, an AF of 10 is used. The use of these assessment factors are consistent with EPA methodology for the screening and assessment of industrial chemicals (U.S. EPA 2013, 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 PCE, EPA used the geometric mean of the EC_{50}s and LC_{50}s for aquatic invertebrates, which is the most sensitive acute value for aquatic species from the data integrated for PCE, from two studies EPA assigned overall quality ratings of high (Niederlehner et al. 1998; Call et al. 1980). The geometric mean of 6.7 mg/L was divided by the AF of five for aquatic invertebrates and multiplied by 1,000 to convert from mg/L to µg/L, or ppb.

The acute COC = (6.7 mg/L) / AF of 5 = 1.3 mg/L x 1,000 = 1,342 µg/L or ppb.

- The acute COC for PCE is 1,342 ppb.

Chronic COC

EPA derived the aquatic invertebrates chronic COC was from the lowest chronic toxicity value from the integrated data using the geometric mean of NOEC and LOEC for growth effects in opossum shrimp (Hollister et al. 1968). The geometric mean 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 = (0.5 mg/L) / AF of 10 = 5.0E-2 mg/L x 1,000 = 50 µg/L or ppb.

- The aquatic invertebrates chronic COC for PCE is 50 ppb.

EPA also derived a chronic COC for fish for comparison to the aquatic invertebrate chronic data. The fish chronic COC was derived from the most sensitive chronic toxicity value (ChV) from the integrated

data using the geometric mean of NOEC and LOEC for measuring mortality in fathead minnow from a study that EPA assigned a quality level of high (Ahmad et al. 1984). 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 = (0.84 mg/L) / AF of 10 = 0.084 mg/L x 1,000 = 84 µg/L or ppb.

- The fish chronic COC for PCE is 84 ppb.

Algal COC

The algal COC was derived from the integrated data using the geometric mean of NOEC and LOEC value for algae mortality (Labra et al. 2010). The algal toxicity value of 0.014 mg/L 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 algal COC = (1.4E-2 mg/L) / AF of 10 = 1.4E-3 mg/L x 1000 = 1.4 µg/L or ppb.

- The algal COC is 1.4 ppb.

3.1.5 Summary of Environmental Hazard

Acute and Chronic Aquatic Toxicity

EPA concludes that PCE presents a hazard for acute exposure duration in aquatic invertebrates, with acute toxicity values as low as 2.5 mg/L, based on immobilization in *Ceriodaphnia dubia* and *Daphnia magna* (Niederlehner et al. 1998) to 18 mg/L (Call et al. 1980). Acute 96-hour exposures to PCE for fish based on mortality LC_{50} toxicity values for rainbow trout of 4.8 mg/L to inland silverside of 28 mg/L (resulting in a geometric mean of 12 mg/L). For chronic exposures to fish, PCE has a hazard values as low as 0.8 mg/L. For chronic exposure to aquatic invertebrates, PCE has a chronic toxicity value of 0.5 mg/L. In algal species, where exposure durations are considered separate from chronic as they can encompass several generations of algae, PCE has a chronic toxicity value of 1.4E-2 mg/L.

Concentrations of Concern

The acute and chronic COCs derived for aquatic organisms are summarized in Table 3-2. EPA calculated the acute COC for PCE exposures in aquatic invertebrates as 1,342 ppb, based on the geometric mean of EC_{50}s and LC_{50}s from two studies that EPA assigned an overall quality level of high (Niederlehner et al. 1998; Call et al. 1980). EPA calculated the chronic COC for PCE exposures in aquatic invertebrates as 50 ppb, based on the geometric mean of NOEC and LOEC for growth from a single study that EPA assigned an overall quality level of high (Hollister et al. 1968).

For comparison with other trophic levels, EPA calculated the fish chronic COC for PCE of 84 ppb, based on the geometric mean of the NOEL and LOEL from a single study that EPA assigned an overall quality level of high (Hollister et al. 1968). As noted previously, algal hazard values from exposures to PCE, for 96-hour durations, are considered separately from other aquatic species because algae can cycle through several generations in this time frame. The algal COC of 1.4 ppb is based on the geometric mean of the NOEL and LOEL from a single study that EPA assigned an overall quality level of medium (Labra et al. 2010).

Confidence in COCs

Based on the data quality, weight of scientific evidence, and uncertainties (see Section 4.3.1), confidence in acute and chronic COCs for fish and invertebrates are high. The COC for algae is based on a single study that EPA assigned an overall quality level of medium. Additionally, algae species tend to vary widely in their sensitivity to chemical pollutants, and data were only available for three algal species and may not represent the most sensitive species at a given site. Therefore, confidence in algae COC is medium.

Table 3-2. COCs for Environmental Toxicity

Environmental Aquatic Toxicity	Hazard Value (µg/L)	Assessment Factor	COC (µg/L or ppb)
Toxicity to Aquatic Invertebrates from Acute Exposures	6,710	5	1,342
Toxicity to Aquatic Invertebrates from Chronic Exposures	500	10	50
Toxicity to Fish from Chronic Exposures	840	10	84
Algal Toxicity	14	10	1.4

3.2 Human Health Hazards

3.2.1 Approach and Methodology

EPA used the approach described in Section 1.5 to evaluate, extract and integrate PCE's human health hazard and dose-response information.

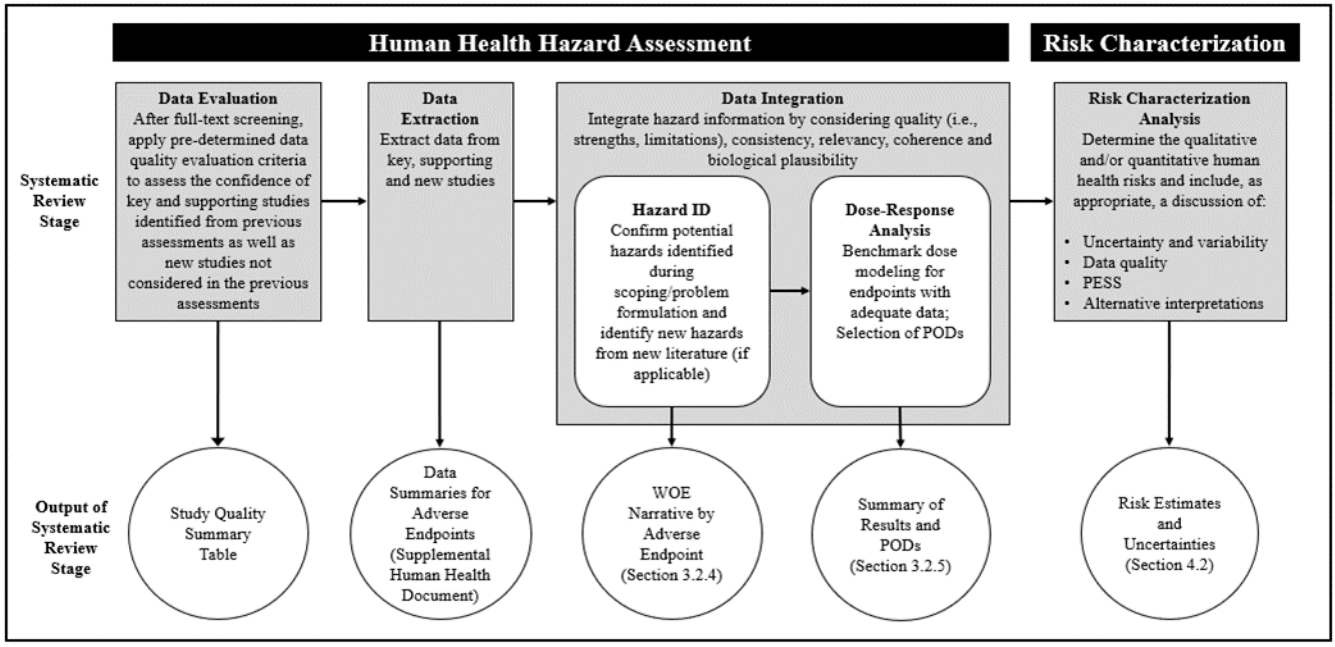

Figure 3-1. EPA Approach to Hazard Identification, Data Integration, and Dose-Response Analysis for PCE

Specifically, EPA reviewed key and supporting information from previous human health hazard assessments as well as the existing body of knowledge on PCE's human health hazards. These data sources included an existing EPA IRIS Assessment (U.S. EPA 2012c) and an ATSDR Toxicological Profile (since finalized as (ATSDR 2019)); hence, many of the human health hazards of PCE have been previously compiled and systematically reviewed.

All human health hazards of PCE previously identified in these reviews were described and reviewed in this risk evaluation, including: acute toxicity, neurotoxicity, kidney toxicity, liver toxicity, reproductive/developmental toxicity, immune and hematological effects, irritation, and cancer. EPA relied heavily on the aforementioned existing reviews along with scientific support from the Office of Research and Development in preparing this risk evaluation. Development of the PCE hazard and dose-response assessments considered EPA and National Research Council (NRC) risk assessment guidance.

Any identified new literature published since these previous assessments was screened against inclusion criteria in the PECO statement and the relevant studies (e.g., useful for dose-response)[16] were further evaluated using the data quality criteria for human, animal, and *in vitro* studies described in the

[16] Some of the studies that were excluded based on the PECO statement were considered later during the systematic review process as needed. For example, EPA reviewed mode of action information to qualitatively support the health hazard assessment.

Application of Systematic Review in TSCA Risk Evaluations document (U.S. EPA 2018b). EPA skipped the screening step (for relevance to PCE) of the key and supporting studies identified in previous assessments and entered them directly into the data evaluation step based on their previously identified relevance to the chemical(U.S. EPA 2018b). EPA skipped the screening step (for relevance to PCE) of the key and supporting studies identified in previous assessments and entered them directly into the data quality evaluation step based on their previously identified relevance to the chemical.

EPA considered studies of low, medium, or high confidence for the weight of scientific evidence (WOE) for hazard identification and dose-response analysis. Information from studies that were rated unacceptable were only discussed on a case-by-case basis for hazard ID and weight-of-scientific-evidence assessment but were not considered for dose-response analysis.

EPA has not developed data quality criteria for all types of hazard information. This is the case for toxicokinetics and many types of mechanistic data which EPA typically uses for qualitative support when synthesizing evidence. As appropriate, EPA evaluated and summarized these data to determine their utility with supporting the risk evaluation.

Following the data quality evaluation, EPA extracted the toxicological information from each relevant study. In the last step, the strengths and limitations of the data were evaluated for each endpoint and a weight-of-the-scientific evidence narrative was developed. Data for each selected hazard endpoint underwent dose-response analysis. Finally, the results were summarized, and the uncertainties were presented. The process is described in Figure 3-1. The WOE analysis included integrating information from toxicokinetics, toxicodynamics in relation to the key hazard endpoints: acute overt toxicity, liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity (including sensitization), reproductive toxicity, developmental toxicity, and cancer. EPA selected human health studies that were of high quality and relevance to move forward for dose-response analysis in order to quantitatively assess each key hazard endpoint.

Summaries for all studies considered for this draft risk evaluation, 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 results of the data quality evaluation are provided in *Draft Risk Evaluation for Perchloroethylene Data Quality Evaluation of Human Health Hazard Studies* and *Data Extraction for Human Health Hazard Studies.* (U.S. EPA 2020g).

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. The POD is a dose or concentration near the lower end of the observed range without significant extrapolation to lower doses. It is used as the starting point for subsequent dose-response (or concentration-response) extrapolations and analyses. PODs can be a no-observed-adverse-effect level (NOAEL), a lowest-observed-adverse-effect level (LOAEL) for an observed incidence, or change in level of response, or the lower confidence limit on the dose at the benchmark dose (BMDL)[17]. PODs were adjusted as appropriate to conform to the specific exposure scenarios evaluated. Section 3.2.5 describes the dose-response assessment guiding the selection of PODs for non-cancer endpoints.

[17] The benchmark dose (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.

3.2.2 Toxicokinetics

The toxicokinetics and PBPK modeling of PCE were thoroughly described in the 2012 EPA IRIS Assessment (U.S. EPA 2012e). This discussion is summarized below.

3.2.2.1 Absorption/Distribution/Metabolism/Elimination (ADME)

3.2.2.1.1 Absorption

Inhalation

Inhalation is considered to be the major exposure route, and studies on both humans and animals confirm that PCE is both rapidly and readily absorbed via pulmonary uptake (with equilibrium occurring after several hours). The blood:gas coefficient ranges from ~10-20, indicating that PCE readily moves from alveoli into the bloodstream. For the purposes of this risk evaluation, EPA conservatively assumes 100% absorption through the lungs.

Oral

For oral exposures, studies in mice, rats, and dogs demonstrate that absorption of PCE through the gut is essentially complete (i.e. 100%).

Dermal

Dermal exposure to PCE vapors is estimated to result in minimal dermal uptake compared to inhalation of those vapors (only ~1% absorbed dermally compared to inhaled). However, studies indicate that dermal absorption may be significant for direct skin application of PCE. Complete (i.e. 100%) absorption may be achieved in scenarios of impeded evaporation or complete immersion, and this risk evaluation assumes that up to 100% of the delivered dermal dose (i.e. after accounting for evaporation or in scenarios with impeded evaporation) is absorbed. Volatilization from the skin is accounted for in the occupational exposure assessment by the *Dermal Exposure to Volatile Liquids Model* based on a theoretical framework provided by Kasting and Miller (2006). The amount of liquid on the skin is adjusted by the weight fraction of PCE in the liquid to which the worker is exposed. Specific details of the dermal occupational exposure assessment can be found in Section 2.4.1.29. For the consumer risk assessment, dermal exposure is assessed using the Consumer Exposure Model (CEM; (U.S. EPA 2017a)) permeability dermal sub-model based on the ability of a chemical to penetrate the skin layer once contact occurs. The CEM permeability model assumes a constant supply of chemical, directly in contact with the skin, throughout the exposure duration. This model was applied only to consumer COUs where evaporation is inhibited, or prohibited, or full immersion of a body part occurs during use. The permeability method does NOT consider evaporation and is more representative of these COU types. For the consumer risk assessment, absorption is assessed using permeability model which uses an absorption rate as opposed to a steady-state percentage (Section 2.4.2.2.2).

Distribution

PCE is broadly distributed to all tissues and can cross both the blood:brain barrier and placenta. The highest concentrations are found in adipose tissues due to the lipophilicity of the chemical. Accordingly, PCE concentrations are higher in the brain and liver than many other tissues and it becomes concentrated in human breast milk. Skeletal muscle has been measured to contain the lowest concentration of any tissue. Long residence time in adipose tissue can result in increasing body burden with continuous or repeated exposures.

3.2.2.1.2 Metabolism

PCE is metabolized in laboratory animals and in humans through at least two distinct pathways:

1) oxidative metabolism via the cytochrome P450 (CYP [also abbreviated as P450]) mixed-function oxidase system;
2) glutathione (GSH) conjugation followed by subsequent further biotransformation and processing, either through the cysteine conjugate β-lyase pathway or by other enzymes including flavin-containing monooxygenase 3 (FMO3) and CYP3A.

The conjugative pathway is toxicologically significant because it yields relatively potent toxic metabolites, however studies in both animals and humans indicate that overall metabolism of PCE is relatively limited—particularly at higher exposures. Oxidative metabolism is the more dominant pathway in rodents, however the relative contribution of each in humans has not been determined. Available data presents a wide range of estimates for amount of PCE metabolized, depending on dose level and species (less metabolized at higher doses, and less metabolized in mice compared to rats). PBPK modeling estimated that at existing occupational regulatory levels only 1.5% of inhaled PCE would be metabolized, while at air concentrations of only 0.001 ppm a median estimate of 23-36% would be metabolized.

Oxidative Metabolism

CYP-mediated oxidative metabolism occurs predominantly in the liver, irrespective of the exposure route, and oxidative metabolites are generally responsible for PCE liver toxicity. The major oxidative metabolite is trichloroacetic acid (TCA), which is believed to derive primarily from the upstream metabolite of trichloroacetyl chloride (through hydrolysis or interaction with peptide amino groups). Dichloroacetic acid (DCA) has also been detected in urine, and DCA may form either due to further metabolism of TCA or via bioactivation of GSH conjugates. Oxalic acid is also believed to be a major urinary metabolite (at least in rats). Trichloroethanol (TCOH) may also be produced, but conflicting data suggests that detected TCOH may only be due to cross-contamination from the closely related chemical, trichloroethylene. Oxidative metabolism occurs at a faster and greater overall rate in rodents compared to humans, however the half-life of these metabolites is much greater in humans (up to 15x longer). Variability in CYP metabolic capacity is generally believed to vary by approximately 10-fold among all humans, however individual variations in *in vitro* CYP2E1 activity as high as 20-50 fold have also been reported. There is also large variability in CYP2E1 activity across different tissues. For ingested chemical, first pass through the liver would be expected to be responsible for the majority of oxidative metabolism and subsequent metabolites would travel through the blood to reach target sites. For other routes, these tissue-specific differences may result in varying downstream toxicological activity. The PBPK model is expected to account for the majority of tissue variability via oral or inhalation routes.

Conjugative Metabolism

The GSH-mediated conjugative pathway begins in the liver, with transport of the initial GSH conjugate (S-(1,2,2-trichlorovinyl) glutathione or TCVG) and its cysteine counterpart (TCVC) to the kidney target organ. While the pathway was originally demonstrated only in rodents, it has since been confirmed to exit in humans, although the relative susceptibility of humans for TCVG production compared to rodents is unclear. Transport to the kidney (primarily) results in further processing and associated renal toxicity. This toxicity is associated at least in part with the activity of β-lyases, which cleave TCVC to yield an unstable thiol, resulting in cytotoxic and mutagenic reactive metabolites. FMO3 can also produce another reactive metabolite, TCVC sulfoxide (TCVCSO), and other sulfoxide species can be produced through CYP3A metabolism of other conjugative metabolites.

Species Differences

The rate of metabolism of PCE is faster in rodents than humans resulting in higher metabolite concentrations in blood. The half-life of these metabolites is significantly longer for humans however (144 hrs in humans vs 10 hrs or less in rodents), meaning that they can impart toxicological effects over a longer period of time. TCA is the major oxidative metabolite produced in both rats and humans as indicated by it's detection in urine, however as mentioned it is detected at much higher blood concentrations (3-8 fold) in rats with a much faster half-life (>4-fold). These results are in agreement with known differences in metabolic rates in general between species, for which mice are faster than rats which are faster than humans.

Additional tissue and MOA-specific details on PCE metabolites are also provided in the Mode of Action section, Section 3.2.3.2.4

3.2.2.1.3 Elimination

PCE is primarily eliminated through pulmonary excretion of the parent compound independent of exposure route. Urinary excretion is the primary route for metabolites, although metabolites are also excreted through the lungs as a minor pathway.

Half-life of PCE from blood-rich tissues, muscle, and adipose tissue is 12-16 hours, 30-40 hours, and 55-65 hours, respectively. In rodents, as body burden increases the percentage excreted as unchanged parent compound also increases (due to decreased metabolism, see Section 3.2.2.1.2). Pulmonary excretion rate is dose-independent, related instead to ventilation rate, cardiac output, and the relative solubility of PCE in blood and tissue. In contrast, contrast, urinary excretion of metabolites is dose-dependent and rate-limited.

3.2.2.2 PBPK Modeling

The 2012 EPA IRIS Assessment (U.S. EPA 2012e) contains a Physiologically Based Pharmacokinetic (PBPK) model for PCE. The most recent analysis by Chiu and Ginsberg (2011a) improved on several earlier models. EPA has made the model code available for download via the internet. The detailed code is publicly available through EPA's HERO database (Chiu and Ginsberg 2011b).

The model structure allowed it to be used to calculate internal dose metrics for inhaled and oral exposure to PCE for mice, rats, and humans. Thus, the analysis could be used for route-to-route extrapolation or interspecies extrapolation, comparison of parent and metabolite toxicity based on a common internal dose metric, and investigation of the shape of the dose-response curve. The following dose metrics could be determined using this model:

- Daily area-under-the-curve (AUC) of PCE in blood
- Fraction of PCE intake metabolized by oxidation
- Fraction of PCE intake metabolized by GSH conjugation
- Equivalent daily production of TCA per kg body weight.

Of note, a full Bayesian uncertainty/variability analysis was not performed. Therefore, the model could not be used to represent the range of intraspecies human variability and was of limited utility for human studies not requiring route-to-route extrapolation.

The highest confidence dose metric is AUC in blood, with the main source of uncertainty for the metric being the residual difference between model predictions and the calibration/validation data (about 2-fold for each species). The next highest confidence is for estimates of PCE oxidation and TCA formation, again with approximately a 2-fold residual difference between predictions and data. There is large

interindividual variability in PCE oxidation that is not captured by the model in the absence of a Bayesian analysis. The model predicts decreasing oxidative metabolism from mice to rats to humans, meaning that humans are predicted to receive a smaller internal dose for the same applied dose compared to rodents, after accounting for body weight scaling. For cross-species extrapolation, the default assumption of equivalent air concentrations leading to equivalent internal doses appears correct based on AUC estimates.

There is greater uncertainty for estimates of GSH conjugation, especially in humans. The data suggests an approximate 2-fold range of uncertainty in rats, however there is minimal available data in mice leading to a ~60-fold range. The human estimates are extremely uncertain, with two local maxima in the model fits resulting in model predictions differing by up to 3,000-fold based on results of different optimization runs. Due to this very broad uncertainty range, the model can result in humans having either equal or greater GSH conjugation compared to rats, for which only ~1% of dosed PCE undergoes GSH metabolism.

3.2.3 Hazard Identification

3.2.3.1 Non-Cancer Hazards

The 2012 EPA IRIS Assessment (U.S. EPA 2012c) evaluated the following non-cancer hazards that may be associated with PCE exposures: the central nervous system (neurotoxicity), kidney, liver and development and reproduction. In general, neurological effects were found to be associated with lower PCE inhalation exposures than what produced other noncancer adverse effects. According to the 2012 EPA IRIS Assessment (U.S. EPA 2012c), support for an association with immune and blood effects were less well characterized. In their Toxicological Profile for PCE, ATSDR (2019) identified similar hazard concerns. The National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (U.S. EPA 2009) also identified irritation as a hazard concern. Since the EPA IRIS Assessment 13 new studies were identified and evaluated during the systematic review process. These new studies add further evidence to support the conclusions established in the EPA IRIS and ATSDR assessments (ATSDR 2019).

3.2.3.1.1 Acute Toxicity and Irritation

Data from acute exposure studies in animals and human incidents indicate that short term exposure to PCE may cause irritation and neurotoxicity and can impair cognitive function in humans (U.S. EPA 2012c). An Acute Exposure Guidance Limit (AEGL) values, established by the National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (U.S. EPA 2009), has been developed based on irritation to humans (AEGL-1), ataxia in rodents (AEGL-2), and lethality in mice (AEGL-3) (U.S. EPA 2009). Epidemiological studies since the EPA IRIS Assessment focused on chronic exposures.

There is sufficient evidence from controlled human exposure studies that acute-duration (≤ 24 hours) inhalation exposure to PCE induces symptoms of CNS depression and prolonged visual evoked potential latencies (ATSDR 2019; U.S. EPA 2012c, 2009; Altmann et al. 1990; Hake and Stewart 1977). While more limited, case reports show that CNS depression (including coma/ unconsciousness at sufficiently high doses) also occurs in humans after oral exposure to PCE (ATSDR 2019)). Sufficient information in acute-duration studies in animals exposed by inhalation or oral gavage also shows CNS depression (ATSDR 2019; U.S. EPA 2009) as well as reduced amplitude of visual evoked potentials, impaired sustained attention, prolongation of escape-directed behaviors after inhalation exposure (ATSDR 2019;

U.S. EPA 2012c; Boyes et al. 2009; Oshiro et al. 2008) and reduce operant response behavior or increased seizure threshold (ATSDR 2019)) after oral exposure.

Human controlled-exposure studies and case reports demonstrated concentration-related increases in the incidence and severity of eye and upper respiratory tract irritation (ATSDR 2019; U.S. EPA 2009). There are also reports of greater excitement and struggling in beagle dogs exposed to PCE by facemask (ATSDR 2019), however this is not adequate evidence to indicate an association with respiratory tract irritation in animals.

Data pertaining to hepatic effects in humans exposed acutely to PCE consist of only a single case report (U.S. EPA 2012c)). Dose-related hepatic effects following acute gavage administration to mice including increased serum ALT, fatty degeneration and necrosis, and cytoplasmic vacuolation (ATSDR 2019).

3.2.3.1.2 Neurotoxicity

The neurological effects of PCE in humans have been extensively studied. Findings in humans are supported by a more limited number of animal studies. The EPA IRIS Toxicological Review for PCE (U.S. EPA 2012c) provides the basis for the information below from studies published up to that time; more recent studies are also discussed. The review performed by EPA IRIS (U.S. EPA 2012c) identified visual deficits in human studies, especially diminished color discrimination, as the most sensitive endpoint of PCE exposure. With one exception, newer human studies have not materially added to the database of PCE effects on visual function; instead, these studies have focused on symptoms of neurotoxicity (Lucas et al. 2015), risks of neurodegenerative diseases (Bove et al. 2014b; Goldman et al. 2012), risks of autism spectrum disorder (Aschengrau et al. 2016a; Aschengrau et al. 2011) or risky behaviors and head injuries (Aschengrau et al. 2016a; Aschengrau et al. 2011) after prenatal or early childhood exposure. One study published since the 2012 IRIS Assessment (U.S. EPA 2012c) assessed visual function of a residential population exposed to PCE in contaminated drinking water (Getz et al. 2012). There have been no oral or inhalation repeated-exposure animal studies published after the IRIS Assessment that evaluated sensitive neurological endpoints.

Human Evidence

Visual Function

Human studies have documented an association between impairments in visual contrast sensitivity and color discrimination and PCE exposure in both occupational and residential settings (U.S. EPA 2012c). Cavalleri et al. (1994) and Gobba et al. (1998), inform the relationship between impaired color discrimination and PCE exposure. Cavalleri et al. (1994) observed a significant positive correlation between time-weighted average concentrations of PCE and the Color Confusion Index (CCI) score on the Lanthony D-15 desaturated panel test among dry cleaning workers in Italy. The 35 workers made many more mistakes in the color vision test when compared with 35 unexposed factory workers, with most errors occurring in the blue-yellow range. Exposure to PCE was measured using passive personal air sampling, yielding a time-weighted (8-hour) average concentration of 6 ppm (41 mg/m^3) for the workers; the mean exposure duration was 8.8 years. Vision testing was performed at the same time of day for workers and controls by an investigator who was blinded to exposure status. When tested two years later, color visual impairment was again significantly associated with exposure concentration among the workers; furthermore, those workers whose exposure to PCE had increased in the two-year interim exhibited a decline in performance from the initial testing, while performance was unchanged among those whose exposure decreased (Gobba et al. 1998). Schreiber at al. (2002) reported diminished

color discrimination or visual contrast sensitivity compared with unexposed referent groups among small groups of children and adults living or working in a building with a co-located dry cleaning establishment. EPA IRIS (U.S. EPA 2012c) identified potential confounders in this study, including diagnoses of learning or developmental delays among some of the exposed children, and correlations between exposure and children's ages and races.

Only one study published after the EPA IRIS Toxicological Review (U.S. EPA 2012c) examined visual function in humans exposed to PCE. Getz et al. (2012) measured color vision and visual contrast sensitivity among adult residents of Cape Cod, MA who were exposed prenatally and during early childhood to PCE-contaminated drinking water. Tests administered to the 25 exposed and 25 unexposed subjects included the Farnsworth D-15 and Lanthony D-15d for color discrimination, as well as tests of near acuity and near contrast sensitivity. The investigator who administered the tests was blinded to exposure status. A statistically significant difference in color discrimination was detected using the Farnsworth test (mean difference 0.05, 95% CI = 0.003, 0.10), but the difference observed in the Lanthony D-15d test was not statistically significant (mean difference 0.07, 95% CI = –0.02, 0.15). Contrast sensitivity at the highest spatial frequency test (18.0 cpd) was also diminished (mean difference –6.47; 95% CI = –12.33, –0.62).

Cognition

Several occupational studies of dry cleaning employees, as well as one study of individuals residing near dry cleaning facilities, have documented relationships between PCE exposure and adverse effects on visuospatial memory, attention, vigilance, and information processing speed (U.S. EPA 2012c). In one key study, a cohort of 65 dry cleaning workers in Michigan, high PCE exposure (TWA of 41 ppm or 278 mg/m^3) was associated with statistically significantly ($p<0.01$) reduced scores for pattern recognition, pattern memory, and visual reproduction tests (compared with low exposure workers whose mean exposure was 11 ppm or 75 mg/m^3 (Echeverria et al. 1995). The investigations by Echeverria et al. provided more robust evidence for the findings of Seeber et al. (1989), who reported dose-related, statistically significant effects on the threshold for perceptual speed test, digit reproduction, digit symbol, and cancellations among 101 German dry cleaning employees with low (8-hr TWA 12 ppm or 81 mg/m^3) or high (8-hr TWA 53 ppm or 359 mg/m^3) exposure to PCE (compared with 84 unexposed controls). Of note, EPA identified several shortcomings in this study, including lack of detail on methods used to select subjects, missing information related to testing procedures, differences in alcohol use between exposed and control subjects that were not accounted for in the models, and nonmonotonic dose-response relationships with some test scores. PCE exposure may also be associated with an increase in reaction time, as reported in a study of dry cleaners (Ferroni et al. 1992).

Neurodegenerative diseases

Goldman et al. (2012) examined the association between Parkinson's disease and exposure to solvents (including PCE) among discordant twin pairs. In the cohort of 99 twin pairs, each having only one twin diagnosed with Parkinson's disease, self-reported exposure (ever exposed) to PCE was associated with a large but very imprecise increased OR (10.5; 95% CI = 0.97, 113). Evaluation of each twin's cumulative PCE exposure did not materially change the findings.

In a retrospective cohort mortality study, Bove et al. (2014b) reported a nonsignificant elevation in the SMR for mortality due to ALS (Amyotrophic Lateral Sclerosis; SMR = 1.14; 95% CI = 0.70, 1.74) among PCE-exposed military personnel at Camp LeJeune (North Carolina) when compared with age, sex, race, and calendar period-specific national mortality rates. Furthermore, the hazard ratio for ALS

mortality increased with cumulative PCE exposure category (HRs of 0.69, 1.58, and 1.96 for low [>1-155 ug/L-months], medium [>155 – 380 ug/L-months], and high [>380 ug/L-months] exposures, respectively) in analyses restricted to the Camp LeJeune cohort. A borderline significant (p=0.06) positive association (β = 0.00039, 95% CI = -0.00002, 0.00080) was observed between cumulative PCE exposure (as a continuous variable) and ALS mortality in the cohort.

Neurodevelopment

Aschengrau et al. (2016a; 2011) conducted a series of studies examining neurological outcomes of early life (prenatal and early childhood) exposure to drinking water contaminated by PCE (cumulative exposures ranging from 11 to 4668 g). Individuals residing in Cape Cod, MA were exposed to PCE leaching from water distribution pipes; a model was used to estimate individual exposures to each residence from leaching. In analyses of 831 persons with prenatal and early childhood exposure compared with 547 unexposed subjects, any exposure to PCE was associated with statistically significant increased risks of engaging in risky behaviors (Aschengrau et al. 2016a). Analyses included adjustment for demographic characteristics, key risk factors for the behavioral and health outcomes under study, and nondrinking water sources of solvent exposure. Odds ratios for use of more than one major illicit drug (crack/cocaine, psychedelics, heroin, Ritalin without a prescription, and club/designer drugs) in the highest exposure groups were 1.6 (95% CI = 1.2, 2.2) for use during adolescence and 1.5 (95% CI = 1.2, 1.9) for use during adulthood. Early and heavy smoking, and frequent or heavy drinking behaviors were also increased among highly exposed subjects (ORs 1.3-1.6, with statistically significantly increased ORs for drinking, but not smoking patterns). In the same population, a significant increased risk was observed for development of bipolar disorder among highly exposed (≥ 67th percentile) subjects (RR = 2.7, 95% CI = 1.3, 5.6). Nonsignificant increased RRs were also seen for post-traumatic stress disorder (1.7, 95% CI = 0.9, 3.2 for exposure ≥ 67th percentile) and schizophrenia (2.1; 95% CI = 0.2, 20.0 for any vs. no exposure, based on 3 cases; (Aschengrau et al. 2016a).

Neuropsychological findings in a subset of the Aschengrau et al. cohort (35 exposed and 28 unexposed adults) who were willing to undergo testing showed modest, nonsignificant differences in performance on tests for visuospatial function, learning and memory, mood alteration, and attention and executive function (mean differences of -0.2 or - 0.3, with confidence intervals in the range of -0.5 to +0.1 or -0.6 to +0.1; (Aschengrau et al. 2016a). The largest magnitude of difference was observed for motor functioning (mean difference in the finger tapping test was -1.8), but the difference was imprecise (95% CI = -5.7 to +2.2). Other studies within the cohort evaluated whether PCE exposure was associated with altered brain MRI findings in a subset of the cohort (26 exposed and 16 unexposed adult subjects). There were no significant differences in MRI findings (e.g., white and gray matter volumes and white matter hypointensities) between the groups. Postulating that neurological sequelae of early PCE exposure could increase the likelihood of unintentional head injuries, Aschengrau et al. (2016b) evaluated the frequency of self-reported head injuries among members of the cohort (828 exposed and 544 unexposed). No increase in the risk of head injuries was observed for any exposure, or in the highest exposure group (RRs 0.8-1.0).

Stingone et al. (2016) evaluated the relationship between standardized test scores in math and English language arts among 3rd graders in New York City schools and modeled air concentrations of PCE (median concentration 0.68 µg/m3) and diesel particulate matter from EPA's National Air Toxics Assessment (NATA) in 1996 (assessment closest to the children's birth years) to correspond with the mothers address at time of birth. Prenatal exposure to PCE in the highest quartile was associated with lower math test scores and increased risk of failing to meet test standards for math (1.03 95% CI = 1.00,

1.06). In analyses of English language arts test results, prenatal PCE exposure was associated with decreased test scores only in the upper tail of the distribution of test scores (75th quantile and above); there was no association with failure to meet test standards. Due to the use of an exposure model based on census tract data and uncertainties surrounding the actual location of mothers during pregnancy, there was potential for exposure misclassification.

Four case-control studies of autism spectrum disorders (ASD) and prenatal exposure to hazardous air pollutants, including PCE, were identified in the literature searches (Talbott et al. 2015; von Ehrenstein et al. 2014; Roberts et al. 2013; Kalkbrenner et al. 2010). Three of the studies used modeled air concentrations of toxicants at the place of maternal or birth residence based on EPA's NATA, while von Ehrenstein et al. (2014) used measured air concentrations from monitoring stations within 5 km of the subjects' residences (Los Angeles County CA). Two studies (Roberts et al. 2013) and (von Ehrenstein et al. 2014) reported significant positive associations between the odds of ASD and PCE exposure. Roberts et al. (2013) reported an OR of 1.60 (95% CI = 1.07, 2.41) comparing the highest to lowest quintiles of PCE exposure in a case-control study nested within the Nurses' Health Study II. In the study by von Ehrenstein et al. (2014), significantly increased ORs were observed for an interquartile range increase in exposure concentration across the pregnancy (OR = 1.40, 95% CI = 1.09, 1.80 for stations within 5 km of the residence and OR =1.61, 95% CI = 1.14, 2.26 for stations within 3.5 km). Stratification by ASD severity and by gender showed stronger associations for milder ASD and in males. Kalkbrenner et al. (2010) and Talbott et al. (2015) did not report significant associations between ASD and PCE exposure in case control studies in NC and WV or PA (respectively).

Clinical Signs of Neurotoxicity

Lucas et al. (2015) observed no significant differences ($p \geq 0.01$) in the prevalence of self-reported symptoms of neurotoxicity (e.g., fatigue at end of day, difficulty sleeping) when comparing 50 dry cleaning workers with exposure to PCE with symptoms reported by 95 workers who were not exposed. The median airborne concentration of PCE was 7 ppm (47 mg/m^3) (range 0.22-33 ppm) in the dry cleaning establishments, and workers had blood levels of PCE ranging between 11.8 and 544 µg/L (median 73.6 µg/L).

Animal Evidence

Animal studies provide support for the effects seen in humans, but the database is much more limited. Effects recorded in studies of rats, mice, and gerbils include clinical signs of neurotoxicity, neurophysiological changes, and alterations in brain chemistry or brain weight (ATSDR 2019; U.S. EPA 2012c). Other studies reported decreases in brain fatty acid and DNA content, alterations in taurine and glutamine content, and decreased brain weight in gerbils and impaired nociception in rats (U.S. EPA 2012c).

Limited information is reasonably available on developmental neurotoxicity in animals exposed to PCE, however existing data suggests that gestational exposure can impair neurobehavior, motor performance, and neurotransmitter signaling (U.S. EPA 2012c).

No studies examining sensitive neurological endpoints in adult animals were published after the EPA IRIS Toxicological Review (U.S. EPA 2012c). No clinical signs of neurotoxicity were noted in female Sprague-Dawley rats exposed to PCE concentrations up to 1000 ppm (6783 mg/m^3) for four weeks in a study focused on immunotoxicity (Boverhof et al. 2013).

3.2.3.1.3 Kidney Toxicity

Human Evidence

Most of the available epidemiological studies, conducted in populations of dry cleaning workers, examined markers of kidney toxicity without including standard tests for kidney function (U.S. EPA 2012c; Mutti et al. 1992). Based on the observed increases in urinary RBP, β2-glucuronidase, lysozyme, and glutamine synthetase, EPA believes that PCE has its primary effect on the proximal tubules, as these are markers of proximal tubular injury. Other markers of tubular injury, including N-acetyl glucuronidase (NAG) and alanine aminopeptidase (AAP) were not associated with exposure (U.S. EPA 2012c), however NAG is a relatively insensitive measure of tubular dysfunction, and AAP was assessed in only one study. One epidemiological study published after the EPA IRIS Toxicological Review (U.S. EPA 2012c) examined non-cancer renal toxicity and found that PCE was not significantly associated with chronic renal diseases (Silver et al. 2014).

Animal evidence

Animals exposed to PCE by inhalation exhibit renal effects such as increased kidney weights, and tubular histopathology (ATSDR 2019; U.S. EPA 2012c). Effects have been reported in both male and female rats and male and female mice. In a multigeneration study of Alpk:APfSD rats exposed for ~19 weeks, renal effects including minimal chronic progressive glomerulonephropathy and increased pleomorphism in proximal tubular nuclei were seen at 1000 ppm (6783 mg/m^3; the highest concentration tested) (Tinston 1994). With two years of exposure to 200 ppm (1357 mg/m^3), male and female rats showed increased relative kidney weights and karyomegaly of the proximal tubules (JISA 1993; NTP 1986b). In a four-week immunotoxicity study published after the EPA IRIS Toxicological Review (U.S. EPA 2012c), no changes in kidney weight or histology were observed in female Sprague-Dawley rats exposed by whole-body inhalation to PCE concentrations up to 1000 ppm (6783 mg/m^3; (Boverhof et al. 2013)).

Mice exposed to 609 ppm (4131 mg/m^3) for 13 weeks exhibited histopathology changes (not further described) in the proximal tubules; at 200 ppm (1357 mg/m^3) for 13 weeks, karyomegaly of the renal tubular epithelial cells was observed (JISA 1993; NTP 1986b). Chronic (2 years) inhalation exposure resulted in nephrosis (karyomegaly and cytomegaly of the proximal tubules) in both sexes of B6C3F1 mice exposed to 100 ppm (678 mg/m^3; the lowest concentration tested) (NTP 1986b) and karyomegaly with atypical dilation of the proximal tubules in male and female hybrid mice exposed to 250 ppm (1696 mg/m^3; (JISA 1993).

After 78 weeks of exposure to doses ≥ 386 mg/kg-day (mice) or ≥ 475 mg/kg-day (rats) administered by gavage in corn oil, both sexes of Osborne-Mendel rats and B6C3F1 mice exhibited toxic nephropathy, with higher incidences in rats than mice (NCI 1977). Mixed evidence including both positive and negative findings for signs of kidney toxicity were observed in other mice studies (U.S. EPA 2012c), while increased kidney weight, urinary markers of damage, and histopathology was reported in rats (Jonker et al. 1996).

A group of studies in F344 rats showed accumulation of α2u-globulin and hyaline droplets in the proximal tubules of male rats exposed to PCE by gavage in corn oil for 10 days to four weeks (U.S. EPA 2012c). These changes were correlated with cell proliferation, formation of granular tubular casts, and tubular cell regeneration, suggesting the involvement of male rat-specific α2u-globulin accumulation in the mode of action for some renal effects of PCE. However, the kidney effects seen in female rats and in mice of both sexes show that other mechanisms (e.g., peroxisome proliferation and/or cytotoxicity

mediated by reactive metabolites produced from glutathione conjugation in the kidney; see Section 3.2.3.2.4) also play a role in the renal toxicity of this compound.

3.2.3.1.4 Liver Toxicity

Human evidence

There is limited information on the hepatic effects of PCE in humans, with conflicting evidence across several occupational studies of dry cleaning workers. Sonographic changes in the liver and alterations in hepatic enzyme levels in serum (compared with unexposed workers) were noted in two studies of dry cleaners with exposure to PCE; however other studies noted no differences in enzyme levels (U.S. EPA 2012c). Exposure levels in the negative studies were comparable to those in the ones reporting effects, but workers in the studies reporting effects had been exposed for much longer (12-20 yrs vs 3-6 yrs in negative studies. In Silver et al. (2014), the only human study of PCE published after EPA IRIS (U.S. EPA 2012c) that examined noncancer liver effects, there was a statistically significant deficit of cirrhosis and chronic liver disease in male workers at a microelectronics and business machine facility.

Animal evidence

Liver toxicity (i.e., necrosis, vacuolation, etc) has been reported in multiple animal species by inhalation and oral exposures to PCE, with the mouse typically being more sensitive than the rat. The liver effects are characterized by increased liver weight, necrosis, inflammatory cell infiltration, triglyceride increases proliferation, cytoplasmic vacuolation (fatty changes), pigment in cells, oval cell hyperplasia and regenerative cellular foci (U.S. EPA 2012c).

In mice exposed to PCE by oral gavage, increased serum ALT levels, increased liver weight, hepatocellular hypertrophy, fatty degeneration and necrosis, and regenerative repair/increased DNA synthesis were observed after exposure to doses of 20 - 2000 mg/kg-day for 6 weeks (Buben and O'Flaherty 1985). Rats exposed orally to 600 or 2,400 mg/kg-day PCE for 32 days showed increased relative liver weight as well (Jonker et al. 1996). In inhalation studies of PCE, both mice and rats exhibited hepatic effects, but mice appear to be more sensitive. Mice displayed increases in palmitoyl CoA, peroxisome proliferation, mitochondrial proliferation, increased relative weight, centrilobular lipid accumulation/fatty degeneration, and liver necrosis/degeneration. Effects observed in rats were limited to increased liver weight after subchronic exposure and spongiosis hepatis and hyperplasia following chronic exposure (U.S. EPA 2012c). In rats, increased liver weight was observed after 90 days of continuous exposure, while spongiosis hepatis and hyperplasia were noted to occur at increased incidences after 110 weeks of exposure (U.S. EPA 2012c; JISA 1993).

A four-week inhalation immunotoxicity study in rats (Boverhof et al. 2013) that was published after EPA IRIS (U.S. EPA 2012c) also reported hepatic effects. Female Sprague-Dawley exposed whole-body to 1000 ppm (6783 mg/m^3) exhibited increased relative liver weights (in conjunction with decreased body weight at this exposure level) and an increased incidence of centrilobular hepatocellular hypertrophy. At lower exposure levels, no biologically significant hepatic effects were noted.

3.2.3.1.5 Reproductive/Developmental Toxicity

The EPA IRIS Assessment for PCE (U.S. EPA 2012c) evaluated the developmental and reproductive toxicity of PCE in humans and animals.

Human evidence

Reproductive

Studies of PCE exposure in humans have evaluated several reproductive outcomes including effects on menstrual disorders, semen quality, fertility, time to pregnancy, and risk of adverse pregnancy outcomes including spontaneous abortion, low birth weight or gestational age, birth anomalies, and stillbirth (U.S. EPA 2012c).

Sperm concentration, morphology and motility were examined in California men who worked as dry cleaners (n = 34) compared with aged matched laundry workers (n= 48) (Eskenazi et al. 1991). The three measures of exposure in this study were dry cleaners vs. laundry workers, exhaled breath concentrations of PCE and an exposure score assigned by an industrial hygienist. Clinically relevant changes in sperm concentration, morphology and motility were not associated with any measure of PCE exposure. Fertility rates were examined among wives of dry cleaners and laundry workers in this study; however, the small sample size in this study precluded a determination of findings.

The potential association between PCE exposure and time to pregnancy was evaluated in several studies including a Danish case-control study of couples treated for infertility, a retrospective time-to-pregnancy study in Finnish women, and a Finnish case-control study (U.S. EPA 2012c). Some evidence of an association was identified in these studies, however the presence of confounders, absence of PCE-specific data in all values, and possibility of bias diminish the impact of the results.

Developmental

The epidemiological evidence for developmental effects associated with PCE exposure is suggestive based on several studies of maternal occupational exposure to PCE that suggest an increased risk of spontaneous abortion at high concentrations (Olsen et al. 1990; Kyyronen et al. 1989). In addition, drinking water studies have suggested associations between PCE exposure and pre-term birth, low birth weight, eye and ear anomalies, and oral cleft defects (U.S. EPA 2012c).

Animal evidence

Data from animal studies identified various manifestations of developmental toxicity including increased mortality and decreased body weight in the offspring of rodents exposed via inhalation.

Reproductive

A multi-generation study (Tinston 1994) exposed rats to 0, 100, 300, or 1,000 ppm (0, 678, 2035, 6783 mg/m3) PCE, 6 hours/day, 5 days/week, for 11 weeks prior to mating and then for 6 hours/day during mating and through GD 20. First generation dams and litters were exposed from PND 6 through PND 29 but were not exposed from GD 21 through PND 5. This study did not evaluate estrous cyclicity, sperm parameters, age to sexual maturation or enhanced reproductive organ histopathology. The only significant reproductive effect reported in this study was reduced testes weight in F1A and F1 males at 1000 ppm (6783 mg/m^3). Sperm abnormalities were not observed in rats exposed to 100 or 500 ppm (678 or 3391 mg/m^3), 7 hours/day for 5 days (measured at 1, 4 and 10 weeks after the last exposure). Sperm head abnormalities were increased in mice exposed to 500 ppm (3391 mg/m^3) PCE at 4 weeks only (Beliles et al. 1980). The temporal pattern of this effect suggests that spermatocytes and/or spermatogonia may be sensitive to PCE exposure. Female reproductive toxicity was also observed based on reduced fertilization of oocytes from exposed female rats (U.S. EPA 2012c).

Developmental

Animals studies generally support the findings from the epidemiological literature for developmental effects associated with PCE. Inhalation exposure to PCE resulted in increases in pre- and post-

implantation losses, increased incidence of total malformations, decreased fetal weight, increased incidence of skeletal retardations or delayed ossification, and/or decreased postnatal survival in rats (U.S. EPA 2012c; Carney et al. 2006), increased incidence of visceral malformations or decreased fetal weight and delayed ossification in mice, and increases in abortions, total litter resorptions, post-implantation losses, and the incidence of malformations in rabbits (U.S. EPA 2012c).

3.2.3.1.6 Immune System and Hematological Effects

Immune System Effects

Human Evidence

The association between PCE exposure and alterations in lymphocyte subpopulations, immunoglobulin and cytokine levels, and other markers of inflammation has been indicated in dry cleaning workers and in children in Germany. Studies of the relationship between serum cytokine and IgE levels in infants or toddlers and volatile organic compounds in the children's bedroom air reported no association with IgE but did report reduced interferon-γ levels for PCE exposure above the 75th percentile (U.S. EPA 2012c). No relevant studies were identified that were published after the EPA IRIS Assessment (U.S. EPA 2012c).

There is conflicting data on whether there is a link between increasing PCE exposure and asthma symptoms. While there is limited evidence of exacerbation of asthma symptoms, other data found no association with either ambient or exhaled concentrations after adjustment for co-exposure to criteria pollutants (U.S. EPA 2012c).

A number of studies have been conducted to evaluate the potential link between systemic autoimmune conditions and exposure to solvents as a category, however limited data is available to evaluate whether PCE exposure alone is associated with these conditions. Case reports and population based studies have examined incidences of sclerosis, localized scleroderma, rheumatoid arthritis, and other conditions without any statistically significant associations obtained (U.S. EPA 2012c).

Animal Evidence

There is conflicting limited data from animal studies concerning effects on the immune organs of thymus and spleen (U.S. EPA 2012c). Two animal studies published after EPA IRIS (U.S. EPA 2012c) examined immune system effects (Boverhof et al. 2013; Seo et al. 2012). Seo et al. (2012) evaluated potential immune adjuvant effects of PCE in ICR mice exposed to 0.01 and 1 mg/L in drinking water for 2 or 4 weeks. Twenty-four hours before assessment (at 2 or 4 weeks), mice were sensitized by intradermal injection with anti-dinitrophenol (DNP) IgE antibody. At assessment, mice were challenged with a solution of Evans blue and anti-DNP IgE antibody via intravenous injection; after 30 minutes, the passive cutaneous anaphylaxis (PCA) reaction was measured by removal of skin dyed blue and quantification of pigment. The PCA reaction was significantly increased at 0.01 and 1 mg/L by 2.1- and 2.4-fold, respectively, at 4 weeks. No significant immune adjuvant effect was observed at 2 weeks.

Boverhof et al. (2013) did not observe immunotoxicity effects in female Sprague-Dawley rats (16/group) exposed whole-body to PCE concentrations up to 1000 ppm (6783 mg/m^3) for 4 weeks (6 hours/day, 5 days/week). No exposure-related changes were noted in total protein concentration, LDH enzyme activity, or leukocyte differential cell distribution in bronchoalveolar lavage fluid. In addition, treatment did not alter the number of spleen cells, or spleen or thymus weight or histology, and there were no treatment-related changes in immune reaction in the SRBC antigen assay.

Hematological Effects

Human Evidence

In a single study, decreased erythrocyte counts and hemoglobin levels and increased total white cell and lymphocyte counts were indicated in PCE-exposed dry cleaning workers (U.S. EPA 2012c). Among human studies published after the EPA IRIS Toxicological Review (U.S. EPA 2012c), no information pertaining to hematological effects was identified.

Animal Evidence

Animal studies showing effects on hematological parameters are restricted to mice with evidence of diminished erythropoiesis and increased leukocytes (U.S. EPA 2012c). PCE exposure resulted exhibited a temporal increase in reticulocytes and a small reduction in erythroid committed cells in the bone marrow as well as increased spleen weight with hemosiderin deposits and red pulp congestion and increased serum LDH isozyme I (ATSDR 2019). When NMRI mice were exposed to PCE in drinking water for 7 weeks starting at 2 weeks of age, Hemolytic anemia with evidence of splenic involvement was observed in mice, with no evidence that hepatic toxicity contributed to the effect (U.S. EPA 2012c).

Hematologic effects were not reported in rat studies reviewed by EPA IRIS (U.S. EPA 2012c). In the 4-week rat study by Boverhof et al. (2013) that was published after the EPA IRIS Toxicological Review (U.S. EPA 2012c), no exposure-related changes to hematological parameters were observed at exposure concentrations up to 1000 ppm (6800 mg/m^3).

3.2.3.2 Genotoxicity and Cancer Hazards

EPA has identified several human studies published subsequent to the 2012 IRIS assessment of PCE and has evaluated these studies as well as key and supporting studies from the IRIS assessment (U.S. EPA 2012c) according to the data quality criteria published in (U.S. EPA 2018b). The key and supporting studies that were evaluated include the studies that were considered for dose-response modeling and heavily considered in the overall IRIS assessment (U.S. EPA 2012c). The full list of studies evaluated for data quality is identified in the supplemental file *Draft Risk Evaluation for Perchloroethylene Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal Studies* (U.S. EPA 2020l).

A summary of genotoxicity studies is also included here. Note that EPA has not re-evaluated genotoxicity studies for quality but is relying on previous assessments, such as the IRIS assessment conclusions. A discussion of these studies follows.

3.2.3.2.1 Genotoxicity

(U.S. EPA 2012c), (IARC 2014), and (ATSDR 2019) provide comprehensive reviews on the genotoxicity of PCE. The discussion of PCE genotoxicity here is based on these previous assessments, supplemented by information from a few individual genotoxicity studies (Everatt et al. 2013; Irving and Elfarra 2013; Tucker et al. 2011).

***In vivo* human**

A handful of cross-sectional studies evaluating genotoxicity endpoints in exposed workers suggested that PCE may induce increases in micronuclei and DNA damage. Significant increases in the frequency of micronuclei and in DNA damage (mean tail length by comet assay) were observed in human lymphocytes from dry cleaning workers (Everatt et al. 2013). The frequency of chromosomal aberrations was not significantly different between workers and controls, but regression analysis of these results in the exposed group showed significant positive associations with PCE exposure duration and

frequency (Everatt et al. 2013). A recent study by Azimi et al. published after the conclusion of the TSCA literature search (as cited in (ATSDR 2019)) provided some support for the finding of DNA damage reported by (Everatt et al. 2013). Azimi et al. observed significant increases in comet assay tail length, percent DNA in tail, and tail moment in 33 dry cleaners employed for at least 3 months (median duration 8 years), when compared with 26 controls; exposure levels were not reported. (Tucker et al. 2011) observed statistically significant increases in the frequencies of acentric fragments and in a group of dry cleaning workers exposed for at least 1 year compared to controls, but no statistically significant difference was observed for chromosomal translocations. A previous study of these subjects reported reductions in oxidative DNA damage in leukocytes from exposed workers compared with controls, and there was no statistically significant increase in sister chromatid exchanges observed in studies on workers compared to ONUs or controls (U.S. EPA 2012c).

***In vivo* animal**

Few in vivo animal studies of PCE genotoxicity have been performed, and the results of the available studies are inconclusive. A marginal but dose-related increase in DNA damage, as measured by comet assay tail intensity, was reported to occur in hepatocytes, but not kidney cells of mice given PCE orally and the significance of this results has been questioned (U.S. EPA 2012c). In an earlier study, single strand DNA breaks were reported in mouse liver and kidney (but not lung) after intraperitoneal injection of PCE, but the observed effect was no longer apparent after 24 hours. No DNA strand breaks were observed in the kidneys of male rats given PCE orally for a week. No increase in oxidative DNA damage was reported in urine, lymphocytes, or liver of rats exposed by intraperitoneal injection, but there was significant morbidity and mortality among the animals at the higher doses (U.S. EPA 2012c).

In one study investigating micronucleus induction, no increase in the frequency of micronuclei was observed in reticulocytes or hepatocytes after intraperitoneal injection of PCE before partial hepatectomy, while an increase in micronuclei was seen in hepatocytes when treatment occurred after partial hepatectomy (ATSDR 2019). Examinations for DNA binding in rats and mice after intraperitoneal exposure to radiolabelled PCE showed DNA labelling in mouse liver and stomach and, at lower levels, in mouse kidney and rat stomach. An earlier study using a less sensitive method showed no DNA binding in mouse liver after oral or inhalation exposure (U.S. EPA 2012c).

***In vitro* mutagenicity**

A test for gene mutations in mouse lymphoma L5178Y cells was negative both with and without metabolic activation (U.S. EPA 2012c). In vitro non-mammalian testing for mutagenicity suggests that PCE itself is not mutagenic, in contrast to some oxidative and conjugated metabolites of PCE. PCE has been extensively tested for forward and reverse mutations in Salmonella typhimurium, Escherichia coli, and Saccharomyces cerevisiae, both with and without metabolic activation. In the preponderance of tests, the results were unequivocally negative, except for one strong exception (ATSDR 2019; IARC 2014; U.S. EPA 2012d).

In that exception study, a clear positive response was observed in *S typhimurium* TA100 with metabolic activation and supplied glutathione (GSH), with an even stronger response when purified GSH S-transferase was also added. These results suggest that metabolites of PCE in the glutathione conjugation pathway are mutagenic. Support for this finding is seen in testing of PCE metabolites for mutagenicity. Ames testing of TCVG yielded positive results with metabolic activation, and equivocal or negative results without activation (U.S. EPA 2012c). However, positive results were observed in Ames testing of TCVC (U.S. EPA 2012c), NAcTCVC (N-acetylated TCVC) (U.S. EPA 2012c), and TCVC sulfoxide (Irving and Elfarra 2013) without metabolic activation. The mutagenicity of NAcTCVC in Salmonella is

believed to result from bacterial deacetylation to TCVC (U.S. EPA 2012c). Irving et al. (2013) showed that TCVC was a more potent mutagen than TCVC sulfoxide, but concluded that the latter was a definite, albeit weak, mutagen.

Oxidative metabolites of PCE have also shown some evidence for mutagenic activity. Trichloroacetyl chloride exposure increased revertants in *S. typhimurium* TA100 with or without activation in one study but not in another (U.S. EPA 2012c). In addition, PCE oxide was positive for reverse mutations in *S. typhimurium* TA1535 without activation, but not in *E. coli* WP2uvrA. Testing of the oxidative metabolite trichloroacetic acid (TCA), is ambiguous because interpretation of TCA in vitro test results is complicated by pH changes induced by the compound (U.S. EPA 2012c).

PCE has been tested for gene conversion, mitotic combination, and reverse mutation in *S. cerevisiae*. Positive results were observed only when log-phase cultures, in which xenobiotic metabolism is stimulated, were used. When stationary cultures were used, exposure did not induce gene conversion, mitotic combination, or reverse mutation (IARC 2014). In growing cells of the D61.M strain, PCE exposure, both with or without metabolic activation, induced aneuploidy (IARC 2014). No evidence for sex-linked recessive lethal mutations was observed in tests of Drosophila melanogaster exposed to PCE by feeding, inhalation, or injection (U.S. EPA 2012c).

***In vitro* Micronuclei, SCEs and Chromosomal Aberrations**

In mammalian cell systems tested in vitro, no evidence for SCEs or chromosomal aberrations was observed in Chinese hamster ovary cells, Chinese hamster lung cells, or human lymphocytes. Assays for induction of micronuclei in vitro yielded mixed results. Induction of micronuclei were reported in Chinese hamster ovary cells exposed to PCE without metabolic activation, but not in Chinese hamster lung cells. Experiments in metabolically enhanced cells yielded positive results for micronucleus induction. Increases in micronuclei were seen in human AHH-1 lymphoblastoid cells (which have high GST activity) and in daughter cell lines that express human CYP2E1 (h2E1 cells) or CYPs 1A2, 2A6, 3A4, 2E1, and microsomal epoxide hydrolase (MCL-5 cells) (U.S. EPA 2012c).

***In vitro* DNA damage and morphological cell transformation**

Few experiments examining DNA damage in cell systems in vitro after exposure to PCE have been performed. Equivocal results were reported in tests of human WI38 fibroblasts for unscheduled DNA synthesis: low doses yielded results comparable to the positive control, while high doses were negative, although the positive control response was weak and cytotoxicity was observed at high doses (U.S. EPA 2012c). In other studies of unscheduled DNA synthesis in rat and mouse hepatocytes and human lymphocytes and fibroblasts, PCE did not yield positive results (U.S. EPA 2012c). A more recent study reported no increase in 8-OHdG (a measure of oxidative DNA damage) or γ-H2AX levels (indicative of double strand DNA breaks) in HepG2 cells exposed to PCE (Deferme et al. 2015); however, the capacity of HepG2 cells to metabolize PCE is unknown.

PCE exposure resulted in morphological cell transformation when RLV/Fischer rat embryo cells were exposed for 2 days, but not when BALB/c-3T3 cells were exposed for 3 days followed by a 30-day incubation period (U.S. EPA 2012c).

3.2.3.2.2 Carcinogenicity Epidemiological Studies

(U.S. EPA 2012c) performed a thorough review of the epidemiological data pertaining to carcinogenicity of PCE available from studies conducted through 2011. This review concluded that there was a pattern of evidence associating PCE exposure with several types of cancer, specifically bladder

cancer, non-Hodgkin's lymphoma (NHL), and multiple myeloma (MM), and that more limited data supporting a suggestive effect were available for cancer at other sites, including esophageal, kidney, lung, liver, cervical, and breast cancer.

Descriptions of the data supporting these conclusions can be found in the IRIS Toxicological Review for PCE (U.S. EPA 2012c). Newer epidemiological studies not available at the time of the IRIS review are summarized in Table 3-3 along with the outcome of EPA's data quality evaluation (U.S. EPA 2020k). A detailed description of all epidemiological data can be found in Appendix 5.3.68F.1.11.

7030 **Table 3-3. Summaries of Newer Epidemiologic Cancer Studies Published after the 2012 IRIS Toxicological Review**

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Cause-specific mortality: kidney cancer, Hodgkin's lymphoma, Leukemias, ALS	Camp Lejeune, North Carolina cohort; n=154,932 median age, start of follow-up: 20 median age, end of follow-up: 49 Camp Pendleton, California cohort n=154,969 median age, start of follow-up: 20 median age, end of follow-up: 49 exposure period: 1975-1985; mortality follow-up period: 1979-2008	Chemical name: Tetrachloroethylene (PCE); exposure matrix: estimated monthly average PCE concentration in Tarawa Terrace water system (1975-1985) Mean: 75.7 ug/L, Median: 84.9 ug/L, Range: 0-158.1 ug/L; estimated monthly average PCE concentration in Hadnot Point water system (1975-1985) Mean: 15.7 ug/L, Median: 15.4 ug/L, Range: 0-38.7 ug/L); Duration: On average an individual in the Camp Lejeune cohort resided at the base for 18 months.	Positive, non-significant associations observed between cumulative exposure to PCE and mortality due to kidney cancer.	(Bove et al. 2014b)	High
Diffuse large B-cell lymphoma	Georgia population (2000 census)	Geocoded toxic release sites data for Perc from 1988-1998 EPA's TRI	Significantly decreased risk for diffuse large B-cell lymphoma with increasing mean distance (per 1 mile) to Perc TRI sites.	(Bulka et al. 2016)	Medium
Mortality from lymphatic and haematopoietic cancer	1704 dry cleaning workers in four US cities (San Francisco/Oakland, Chicago, Detroit, and New York)	Employment in a shop using Perc, mean (sd) years of employment for exposed workers 6.2 (5.0)	Significant elevated SMRs were observed for all cancers, esophageal cancer, and trachea, bronchus, and lung cancer. SMRs were significantly lower for liver cancer. No significant association was found for kidney cancer, lymphatic and haematopoietic cancer, and bladder cancer.	(Calvert et al. 2011)	Medium

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Diagnosis of cancer in oral cavity, oropharynx, hypopharynx, oral cavity, and larynx (detailed list of codes in text)	Case-control, women only, 296 cases, 775 controls, diagnosed 2001-2007, general population, 18-85 years, subset of ICARE cohort	Perc, exposure qualitatively stated, modeled as cumulative exposure index (CEI)	Statistically significant positive association between Perc and head/neck cancers in ever/never analysis; null association in continuous cumulative exposure assessment	(Carton et al. 2017)	Medium
Cancers of the bladder, prostate, colon, stomach, rectum, kidney, pancreas, esophagus, and liver, as well as melanoma and non-Hodgkin's lymphoma.	3730 male, Canadian patients aged 35 to 70 years diagnosed 1979-1985 in 18 largest Montreal hospitals; 533 controls from electoral lists in Quebec. A second control group consisted of the population controls together with patients with cancers at sites distal to the primary cancer being assessed.	PERC exposure determined from self-reported job history categorized by chemists and industrial hygienists based on degree of confidence, frequency, and relative levels (not quantitative)	Significant increase in the OR for prostate cancer associated with Perc exposure (substantial), non-significant OR for all other cancers	(Christensen et al. 2013)	Medium
Breast cancer incidence	920 incident breast cancer cases, 1293 controls, Cape Cod, Massachusetts, 1983-1993,	Water distribution modeled exposure to Perc-lined public water distribution pipelines	Perc was not significantly associated with breast cancer, but there was a modest increase in risk in women with high perc exposure	(Gallagher et al. 2011)	Medium
Bladder cancer	113,343 cases and 566,715 matched controls from the Nordic Occupational Cancer (NOCCA) project (through 2005)	Perc exposure estimated via linkage between occupational codes and Nordic Occupational Cancer (NOCCA) project job exposure matrix (JEM)	No significant trend in risk with increasing Perc exposure, significant increase in hazard ratio was only observed in the mid exposure group	(Hadkhale et al. 2017)	Medium

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Neuroblastoma	Children (75 cases, 14602 controls), ages <6 born in 1990-2007 in California within 5 km of exposure monitoring stations, cases from California Cancer Registry	Perc (0.186 ppbV) in ambient air, pollution monitoring stations used to estimate maternal exposure during pregnancy from birth certificate address	Non-significant positive association between Perc and neuroblastomas per interquartile increase in exposure at 5km radius	(Heck et al. 2013)	Medium
Astrocytic brain cancer risk	Men in southern Louisiana, United States, exposed from 1978 - 1980; in northern New Jersey and Philadelphia, Pennsylvania, United States, exposed from 1979 - 1981 (n=620, 300 cases, 320 controls)	Tetrachloroethylene, low exposure (1)	Chi trend= -0.65. Exposure not significantly associate with astrocytic brain cancer	(Heineman et al. 1994)	Medium
Cancer mortality	Lockheed Martin aircraft manufacturing factory workers in Burbank, California (employed after January 1, 1960; followed up through December 31, 2008)	Years of exposure to Perc based on job histories and industrial hygiene surveys	No significant trend for any specific cancer or total cancer by increasing years of exposure.	(Lipworth et al. 2011)	High
Lung cancer	Investigation of occupational exposure and environmental causes of respiratory cancers (ICARE) study subjects, population-based case-control study in France 2001-2007 (2274 men cases and 2780 men controls)	Cumulative Exposure Index (CEI) based on self-reported job histories and probability, intensity, and frequency of exposure to Perc based on jobs	Perc was not significantly associated with lung cancer in men.	(Mattei et al. 2014)	Medium

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Mycosis fungoides (MF)	100 patients with Mycosis Fungoides and 2846 controls, 35-69 years of age, from Denmark, Sweden, France, Germany, Italy, and Spain, 1995-1997	Occupational exposure to Perc assessed with job exposure matrix	A positive, non-significant association was observed between Mycosis Fungoides and male subjects with exposure to Perc >= median of control exposure vs. unexposed male subjects	(Morales-Suárez-Varela et al. 2013)	High
Brain cancer: glioma and meningioma cases	489 glioma cases, 197 meningioma cases, and 799 controls from three USA hospitals in Arizona, Massachusetts and Pennsylvania	Occupational exposure to Perc via self-reported occupational history and industrial hygienist assigned level of exposure	Perc was not significantly associated with glioma or meningioma	(Neta et al. 2012)	High
Cancer of the liver	15 million people participating in a decennial census in Denmark, Finland, Iceland, Norway, and Sweden. Aged 30-64 in years 1960-1990.	Employment in dry cleaning and/or laundering during time period of predominant Perc use	Significantly elevated SIRs were observed in women for stomach, liver, cervical, oral cavity, and lung cancers. No association was found for kidney, bladder, and non-Hodgkin's lymphoma cancer incidence in women.	(Pukkala et al. 2009)	Medium
Diagnosis of kidney cancer	General population case-control study of kidney cancer (1217 cases; 1235 controls). Detroit (2002 - 2007) and Chicago (2003).	Job exposure matrix was used to determine years exposed, average weekly exposure and cumulative hours exposed. to perc	Increased risk of kidney cancer for high intensity exposure group; OR 3.0 (1.3 - 7.4) for 3rd tertile (>1820 hours) vs. unexposed for cumulative hours exposed. No significant associations observed in for other levels of perc exposure.	(Purdue et al. 2017)	High

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Mortality from multiple myeloma	Aircraft maintenance workers (n = 14,457; 10,730 men and 3725 women) at Hill Air Force Base (Utah, USA), for at least one year from 1952-1956, and followed up through 2000	Occupational exposure to Perc (yes/no) based on job-exposure matrix; no quantitative assessment available	Positive association between mortality from multiple myeloma and occupational exposure to Perc compared to no exposure (statistically significant for females, non-statistically significant for males)	(Radican et al. 2008)	Medium
Childhood cancers, neural tube defects, oral clefts,	Children born to mothers with exposure to contaminated drinking water at Camp Lejeune: 51 cases and 526 controls	Perchloroethylene (perc) in drinking water during 1st trimester of pregnancy; modelled exposure high (>=44 ppb), low (<44 ppb)	Positive, non-significant associations observed between childhood cancers and any, high or low 1st trimester exposure to perc compared to unexposed).	(Ruckart et al. 2013)	High
Age of diagnosis of breast cancer (male only).	Case-control, male Marines born before 1969, diagnosed 1995-2013, with identifiable tour dates/locations	Perc, residential drinking water at Camp Lejeune, cumulative exposure >159 ppb	Non-significant positive association between Perc exposure and breast cancer diagnosis and age of diagnosis	(Ruckart et al. 2015)	High
Glioma	Non-farm workers from the Upper Midwest Health Study (798 cases and 1141 controls from Iawa, Michigan, Minnesota, and Wisconsin 1995-1997)	Perc (tetrachloroethylene) use (self-reported occupational history through 1992, bibliographic database of published exposure)	Perc was associated with a significant decrease in gliomas.	(Ruder et al. 2013)	High

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Total lymphoma, HL, B-NHL, T-NHL, B-NHL subentities (DLBCL, FL, CLL, multiple myeloma, marginal zone lymphoma)	710 participating cases (matched to 710 controls) with malignant lymphoma among men and women aged 18 to 80 years in 6 regions in Germany	Cumulative occupational exposure to Perc [ppm*years] based on intensity, the frequency, and duration of Perc exposure (0 to >78.8 ppm*years)	Perc was not significantly associated with malignant lymphoma or any specific type of lymphoma; however, there was an increase (non-significant) in risk of total lymphoma in the highest exposure group (>78.8 ppm*years).	(Seidler et al. 2007)	High
Kidney, bladder, liver, NHL, overall cancer incidence	Swedish national cohort of dry cleaning and laundry workers (n = 10,389) assembled in 1984 followed up for new cases of cancer by matching with the Swedish cancer register from 1985 to 2006	Occupation as dry cleaners and laundry workers exposed to perchloroethylene; exposure levels in the 1970s were of the order of 100–200 mg/m3 (15–30 ppm)	Non-significant elevated risk of Hodgkin's lymphoma, kidney and liver cancer, significantly elevated risk of Non-Hodgkin's lymphoma and lung cancer; no elevated risk of bladder cancer	(Seldén and Ahlborg 2011)	Medium
Kidney cancer incidence	Greater Montreal metropolitan area. Case-control study of occupationally-exposed men aged 35 to 70 year old (4263 cases, 533 population controls; also hospital and cancer controls).	Any or substantial exposure	ORs were not significantly elevated for PCE exposure and kidney cancer (no quantitative data were provided).	(Siemiatycki 1991)	Medium

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Bladder and other urinary cancer mortality	National Institute for Occupational Safety and Health (NIOSH) Cohort, 34494 workers at NY microelectronics and business machine facility, 2009, 52-65yrs	Cumulative Perc exposure score based on department-exposure matrix	Perc was not significantly associated with bladder and other urinary cancers mortality.	(Silver et al. 2014)	Medium
Testicular cancer	National Institute for Occupational Safety and Health (NIOSH) Cohort, 34494 workers at NY microelectronics and business machine facility, 2009, 52-65yrs	Cumulative Perc exposure score based on department-exposure matrix	Perc was not significantly associated with testicular cancer incidence.	(Silver et al. 2014)	Medium
Acute myeloid lymphoma	Cases of acute myeloid leukemia (n=14,337) diagnosed between 1961 and 2005, and controls (n=71,027) matched by age, sex, and country identified from the Nordic Occupational Cancer Study cohort	Cumulative Perc exposure estimated using job exposure matrix, Median (ppm-yr) 12.1	No significant increase in acute myeloid leukemia risk was observed with low, moderate, or high exposure to Perc, compared to referent group when hazard ratios were calculated using a 10-year lag (p-value = 0.39). Findings for analysis stratified by sex or age were not reported	(Talibov et al. 2014)	High

Outcome/ Endpoint	Study Population	Exposure	Results	Reference	Data Quality Evaluation
Cancer diagnosis: liver/biliary, kidney, bladder, pancreas, lung, cervix, Hodgkin's lymphoma, and non-Hodgkin's lymphoma	Adults working in the Sweden during the 1960 and 1970 census, including 31,418 women and 15,515 men working as launderers, dry cleaners, or pressers	Occupation as a dry cleaner, launderer, or presser served as surrogate for Perc exposure	Increased incidence of Hodgkin's disease (significant), lung (significant), cervix (significant), liver/biliary passages, kidney, and bladder cancer, all other outcomes were non-significant	(Travier et al. 2002)	High
Lung cancer	Lung cancer cases and randomly selected population-based controls frequency matched by sex and age in Montreal Canada	Perc exposure (any or substantial) was assessed by a team of industrial chemists and hygienists based on self-reported job histories	Increase in OR for any exposure or substantial exposure to Perc, results were only significant for any exposure in Study I and in the pooled analysis	(Vizcaya et al. 2013)	Medium
Liver and kidney cancer, non-Hodgkin's lymphoma (NHL) and multiple myeloma (MM)	All subjects aged 30–64 years who participated in 1960 through 1990 censuses in Finland, Iceland, Norway and Sweden; five matched controls per case	Job-exposure matrix, intensity × prevalence of perchloroethylene exposure (90th percentile: 0.05 units)	A positive, non-significant association was observed between high cumulative perchloroethylene exposure (intensity × prevalence) and kidney cancer in men and women.	(Vlaanderen et al. 2013)	High
Renal pelvis cancer, bladder cancer	Employed Swedish residents (1,014 and 360 renal pelvis cancers and 18,244 and 3,347 bladder cancers among men and women, respectively)	Occupation type (workers in laundry, ironing, dyeing) or industry	Non-significant excess risk of renal pelvis cancer among men working in laundry, ironing, dyeing industry.	(Wilson et al. 2008)	Medium

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3.2.3.2.3 Carcinogenicity Animal Studies

(U.S. EPA 2012c) performed a review of the animal toxicity data pertaining to carcinogenicity of PCE from studies conducted through 2011. No additional animal cancer studies were located in U.S. EPA's current systematic review. A summary of the database reviewed by (U.S. EPA 2012c) for each cancer is provided as follows. Full study details are provided in Appendix F.2.

Liver

Hepatocellular adenomas and carcinomas exhibited a dose-related increase in male and female B6C3F1 mice exposed by inhalation to PCE at 100 or 200 ppm for 103 weeks, with significant increases in incidence of hepatocellular carcinoma and combined hepatocellular adenomas or carcinomas observed at both exposure concentrations (NTP 1986a). A dose-related increase in hepatocellular adenomas or carcinomas was also observed in male and female Crj:BDF1 mice in a 2-year inhalation study, with increases achieving statistical significance in both sexes at 250 ppm (JISA 1993). A significant increase in the combined incidence of hemangiosarcomas or hemangiomas, occurring in the liver, spleen, fat, subcutaneous skin, and heart, was observed in male mice at 250 ppm (JISA 1993). In an oral study, the incidence of hepatocellular carcinoma was significantly increased in male and female B6C3F1 mice administered time-weighted average doses of 536 or 1,072 mg/kg-day in males and 386 or 772 mg/kg-day in females for 78 weeks, with a decreased time to first tumor in treated male and female mice, compared to controls (NCI 1977).

Kidney

Renal tubular adenomas and adenocarcinomas were observed in male, but not female, F344/N rats exposed to PCE by inhalation at 200 or 400 ppm for 103 weeks (NTP 1986a); although incidence was low, the rarity of renal tubular carcinomas in this strain of rat, in combination with the proliferative lesions (renal tubular cell hyperplasia) observed in male rats and one female rat, suggest that these findings are biologically significant.

Blood

A dose-related increase in the incidence and severity of MCL was observed in male and female F344/N rats exposed to PCE by inhalation at concentrations up to 400 ppm for 103 weeks, with decreased time to onset in exposed females (NTP 1986a). The incidence of advanced stage MCL was significantly increased in both sexes at 400 ppm (NTP 1986a). (JISA 1993) also observed a positive dose-related trend in the incidence of MCL in male and female F344/DuCrj rats exposed by inhalation for 2 years, reaching statistical significance in males only at 600 ppm. The time to first occurrence of MCL was reduced in exposed female rats, relative to controls (JISA 1993).

Brain

A slight, but biologically significant, increase in brain gliomas was observed in male and female F344/N rats exposed to PCE by inhalation at 400 ppm for 103 weeks (NTP 1986a). The fact that this is a rare tumor type, along with a decreased time to first tumor in exposed rats, support the biological significance of this finding.

Testis

F344/N rats exposed to PCE vapors at 200 or 400 ppm for 103 weeks exhibited a significant positive dose-related trend in the incidence of testicular interstitial cell tumors (NTP 1986a).

3.2.3.2.4 Mode of Action

Liver

Modes of action considered by (U.S. EPA 2012c) for liver cancer induced by PCE in mice include: (1) genotoxicity; (2) epigenetic changes (altered DNA methylation); (3) cytotoxicity and oxidative stress; and (4) peroxisome proliferator-activated receptor (PPAR) activation/peroxisome proliferation. Based on their review of available data, both (U.S. EPA 2012c) and (IARC 2014) determined that multiple modes of action were likely responsible for liver tumors induced by PCE. A number of newer publications (Luo et al. 2018b; Luo et al. 2018a; Cichocki et al. 2017; Luo et al. 2017; Zhou et al. 2017; Lacey et al. 1999) examining toxicokinetic and toxicodynamic responses in the livers of mice exposed to PCE and the related compound, trichloroethylene, provide additional insight into the modes of action for PCE liver cancers in mice.

Much of the research on liver carcinogenicity associated with PCE exposure has focused on the role of the metabolite TCA. Further information on modes of action for TCA hepatocarcinogenicity can be found in the (U.S. EPA 2011b) Toxicological Review for TCA.

Role of metabolism

Available information on the metabolism of PCE in the liver suggests that the oxidative metabolism is likely the dominant pathway, with glutathione conjugation occurring to a much lesser degree (U.S. EPA 2012c). Metabolism through the oxidative pathway was ~30-fold higher than through the conjugation pathway in male mice of three strains after single oral doses of 1,000 mg/kg PCE (Luo et al. 2018b). The primary oxidative metabolite of PCE is trichloroacetyl chloride (TCAC) which is subsequently hydrolyzed to TCA. Dechlorination of TCA could yield dichloroacetic acid (DCA); however, most of the DCA excreted after exposure to PCE is believed to be produced in the kidney as an end product of β-lyase metabolism (reviewed by (Guyton et al. 2014). Initially, oxidative metabolism of PCE was believed to be mediated primarily by CYP2E1. However, (Luo et al. 2018a) observed TCA formation in the livers of CYP2E1 knock-out mice (albeit at lower levels than in wild-type), showing that other CYPs can also metabolize PCE to TCA.

Metabolites of the glutathione conjugation pathway also occur in the liver. In C57BL/65J mice given a single dose of 100, 300, or 1,000 mg/kg PCE, dose-dependent increases in the concentrations of S-(1,2,2-trichlorovinyl) glutathione TCVG and N-acetyl-S-(1,2,2-trichlorovinyl)-L-cysteine (NAcTCVC) in the liver were seen, and the concentrations were higher in the liver than in kidney or serum in these animals (Luo et al. 2017). At 1,000 mg/kg, but not lower doses, S-(1,2,2-trichlorovinyl)-L-cysteine (TCVC) was also detected in the liver (Luo et al. 2017), likely because oxidative metabolism was saturated at this dose.

Genotoxicity in the liver

Individual studies of PCE genotoxicity are discussed above under Genotoxicity. As discussed in that section, PCE shows little to no genotoxic activity in the absence of metabolic activation. Several metabolites resulting from both the oxidative and conjugation pathways have shown some indication of mutagenic activity in vitro, including TCAC, TCVG, TCVC, TCVC sulfoxide (TCVCS), NAcTCVC, and PCE oxide. Among these, TCVG and NAcTCVC have been detected in the livers of C57BL/65J mice. The primary metabolite in the liver, TCA, has shown little to no genotoxic activity in vitro, but testing of this compound is confounded by the pH changes it induces. In vivo studies examining genotoxicity have shown negative or equivocal effects (i.e. modest increases in DNA damage and DNA binding in mouse) (U.S. EPA 2012c). There is also general positive epidemiological evidence (not kidney-specific) of genotoxicity from chronic PCE exposure in humans (Section 3.2.3.2.1).

Epigenetic changes

Changes in the methylation of DNA have been shown to occur early in the development of most tumors (U.S. EPA 2012c). There are no studies examining mouse liver DNA methylation or other epigenetic changes after exposure to PCE. A role for DNA hypomethylation in the hepatocarcinogenicity of PCE has been postulated based on observations of hypomethylation, especially in the proto-oncogenes c-myc and c-jun, in mouse liver after exposure to the metabolites TCA and DCA (IARC 2014; U.S. EPA 2012c). Notably, c-myc DNA hypomethylation occurred earlier than increases in liver cell proliferation (U.S. EPA 2012c).

Cytotoxicity and oxidative stress

Studies in mice and rats exposed for at least 4 weeks provide clear evidence for the hepatotoxic effects of PCE (see Section 3.2.3.1.4), and demonstrate that mice are more sensitive to these effects than are rats. In mice, oral exposure to PCE has resulted in increased serum alanine aminotransferase (ALT) levels, increased liver weight, hepatocellular hypertrophy, fatty degeneration and necrosis, and regenerative cell proliferation/increased DNA synthesis (U.S. EPA 2012c), while inhalation exposure induced peroxisome proliferation, mitochondrial proliferation, increased relative liver weight, centrilobular lipid accumulation/fatty degeneration, necrosis, and degeneration (U.S. EPA 2012c). A more recent study of male mice from 45 mouse strains given a single oral dose of PCE (1,000 mg/kg) showed a range of hepatic effects at sacrifice within 24 hours postdosing; most strains showed significant increases in liver triglycerides, and about one-third of the strains exhibited hepatosteatosis of varying severities (Cichocki et al. 2017). PCE-induced accumulation of triglycerides in the liver appears to require the presence of CYP2E1, as knock-out mice did not show this effect after 5 days of oral exposure while wild-type mice and those expressing humanized CYP2E1 did.

In the one study that examined the relationship between hepatocyte toxicity and regenerative cell proliferation in mice (U.S. EPA 2012c), toxicity (manifested as increased plasma ALT) was evident within 24 hours of exposure at all three dose levels (150, 500, and 1,000 mg/kg-day for 30 days). DNA synthesis was increased at all doses after 7 days of exposure (the earliest time point measured), and histopathologic evidence of regenerative repair was seen after 30 days of exposure to the two higher doses (U.S. EPA 2012c), demonstrating that hepatocyte injury occurred early and may have preceded cell proliferation.

In addition to regenerative cell proliferation, other sequelae of hepatotoxicity, including inflammation and oxidative stress, could play a role in liver tumors induced by PCE. In humans, fatty liver resulting from a high-fat diet is thought to increase oxidative stress, leading to genetic instability and release of inflammatory mediators that contribute to the induction of hepatocellular carcinoma (reviewed by (Takakura et al. 2019)). As discussed above, hepatic triglyceride accumulation and fatty degeneration are hallmarks of PCE exposure in mice. Limited data pertaining to the role of oxidative stress in PCE-induced mouse liver toxicity or carcinogenicity are available, showing that administration of the antioxidants vitamin E and taurine mitigated hepatic effects (increases in liver to body weight, alterations in glycolytic and gluconeogenic enzyme and ATPase activities, and/or hepatocyte degeneration and necrosis) in Swiss mice exposed to 3,000 mg/kg-day PCE for 15 days (U.S. EPA 2012c).

Deferme et al. (2015) reported no increase in oxygen radical formation (measured by electron spin resonance spectroscopy) in HepG2 cells exposed to 2 mM PCE in vitro for up to 72 hours. Consistent with this result, (Deferme et al. 2015) did not observe a significant induction of genes related to

oxidative stress after PCE exposure in this system. However, in B6C3F1 mice exposed via gavage, a dose-related upregulation of genes involved in oxidation/reduction was observed after exposure to PCE (Zhou et al. 2017).

PPAR activation

PPARα is a ligand-activated transcription factor involved in the regulation of hepatic lipid metabolism. In response to fasting, PPARα activation in mammals leads to upregulation of genes involved in fatty acid β-oxidation, mitochondrial β-oxidation, gluconeogenesis, and autophagy, all aimed at providing the fasted body with adequate glucose (reviewed by (Preidis et al. 2017)). Activation of the PPARα receptor as a mechanism for hepatocarcinogenesis is proposed to operate through perturbations in cell proliferation and apoptotic pathways, leading to clonal expansion of initiated cells (U.S. EPA 2012c).

In laboratory animals exposed to PCE, several effects indicative of PPARα activation have been observed, including increases in the number and size of liver peroxisomes (U.S. EPA 2012c), increased expression of CYP4A peroxisomal marker enzymes (Cichocki et al. 2017; Zhou et al. 2017; Philip et al. 2007), and increased hepatic levels of palmitoyl coenzyme A oxidase (PCO, also known as acyl CoA oxidase) (U.S. EPA 2012c). Studies comparing results in rats and mice have shown greater increases in PCO in the livers of mice exposed to PCE than in rat livers after exposure to the same doses (U.S. EPA 2012c). In vitro testing indicates that activation of mouse and human PPARα after exposure to PCE is likely mediated primarily by the metabolites, TCA and/or DCA, as PCE itself was essentially inactive (U.S. EPA 2012c).

(U.S. EPA 2012c) also reviewed the dose-response and temporal concordance between PPARα activation and cell proliferation in SW mice exposed to PCE. The original study showed that cell proliferation occurred at lower doses (≥150 mg/kg-day after 7 days after exposure) and persisted longer (14-30 days after exposure at 500 and 1,000 mg/kg-day) than increased expression of PPARα marker CYP4A (1,000 mg/kg-day and only after 7 days of exposure). The study authors suggested that their findings argued against a significant role of PPARα activation in PCE-induced liver carcinogenicity. Citing other studies in mice and rats, (U.S. EPA 2012c) noted that PCE induces a modest peroxisome proliferating response in both species, but only mice develop liver tumors, indicating a lack of concordance between peroxisome proliferation and occurrence of liver tumors across species.

Several notable papers probing the role of PPARα activation in mouse liver after PCE exposure were published after the literature searches were performed for the (ATSDR 2019), (IARC 2014), and (U.S. EPA 2012c) reviews. In a study comparing mouse liver and kidney transcriptomic responses to equimolar oral doses of trichlorethylene and PCE, (Zhou et al. 2017) observed dose-related upregulation of genes involved in PPARα signaling, fatty acid metabolism, and oxidation/reduction in the livers of male B6C3F1 mice exposed to PCE. Genes related to the ATP binding cassette (ABC) family of transporters were also upregulated by PCE; some of these transporters are involved in transportation of cholesterol and lipids, and some are expressed exclusively in peroxisomes. Genes in mitochondria-related pathways and nucleotide metabolism pathways were downregulated. The dose-related alterations in gene expression were correlated both with external PCE dose and hepatic levels of TCA. While gene expression changes related to PPARα signaling were common to both trichloroethylene and PCE, effects on genes related to ABC transporters, mitochondrial pathways, and nucleotide metabolism were unique to PCE (Zhou et al. 2017).

Cichocki et al. (2017) published a seminal paper examining mouse strain variability in toxicokinetic and toxicodynamic responses to PCE exposure. Male mice of 45 strains (Collaborative Cross) received a single oral dose of 1,000 mg/kg PCE and were sacrificed at several time points up to 24 hours after dosing. In this study, variability in liver TCA levels after exposure spanned almost an order of magnitude. In addition, the toxicodynamic response to PCE varied: some strains exhibited significantly lower body weight (as much as 15%); only a few showed significant differences in liver to body weight ratio. Most strains showed significant increases in liver triglycerides with concomitant decreases in serum triglycerides, and about one-third exhibited hepatic steatosis. Similarly, most strains showed increased hepatic expression of PPARα markers CYP4A10 and Acox1 (the gene that encodes acyl CoA oxidase or PCO); however, the degree of upregulation varied almost 600-fold across the strains. (Cichocki et al. 2017) noted that none of the significant effects of PCE on hepatic endpoints (including CYP2E1 protein and triglyceride levels, expression of PPARα responsive genes, and histopathology changes) was correlated with hepatic TCA levels across the tested strains. The reason why dose-related gene expression changes were correlated with hepatic TCA levels in male B6C3F1 mice (Zhou et al. 2017) but not correlated across the strains tested by (Cichocki et al. 2017) is unclear, but could include strain differences in CYP isozyme activities and saturation as well as toxicodynamic differences across the strains.

Two studies of PPAR knock-out mice and mice expressing humanized PPARα exposed to the closely related compound trichloroethylene provide insight into the role of PPARα activation in PCE-induced liver effects in mice. PCE and trichloroethylene share the common metabolite TCA, which is believed to play a role in the hepatic toxicity and carcinogenicity of both compounds. (Ramdhan et al. 2010) compared the effects of trichloroethylene exposure via inhalation at 1,000 or 2,000 ppm (8 hours/day) for 7 days in male Sv/129 wild type mice, PPARα(-/-) knock-out mice, and mice modified to express human PPARα cDNA (hPPARα). Hepatic effects of trichloroethylene exposure that did not differ significantly among the three strains included increased liver weight, increased plasma aspartate aminotransferase (AST) and ALT, and histopathology evidence of liver necrosis. Hepatic inflammation was observed at the highest exposure in all strains (and not in controls) but was of lesser severity in both PPARα-null and hPPARα mice. Only wild type mice exhibited a significant increase in hepatocyte proliferation, and only at the highest exposure. In contrast, only PPARα-null and hPPARα mice exhibited significant increases in liver triglycerides (at both exposure levels in hPPARα mice, and at the highest exposure only in PPARα-null) and hepatic steatosis (at both exposure levels in both strains). No change in hepatic triglycerides or steatosis was seen in wild-type mice. Both wild-type and hPPARα mice exhibited upregulation of PPARα target genes, while PPARα-null mice did not. Interestingly, urinary excretion of TCA was significantly lower (by about half) in PPARα-null mice compared with wild type and hPPARα mice, indicating that toxicokinetics may explain some of the differences in effects.

To investigate the role of toxicokinetics, (Yoo et al. 2015) administered trichloroethylene by gavage (400 mg/kg) to male and female mice (129S1/SvImJ, PPARα-null, and hPPARα) once or 5 days/week for 4 weeks and measured metabolite levels in liver, kidney, and serum, and their relationship to PPARα activation. Marked sex-related differences in tissue levels of trichloroethylene, trichloroethanol (TCOH), and TCA were observed after single or repeat dosing, with males exhibiting significantly higher metabolite levels in liver, kidney, and serum. No differences between the strains were seen in levels of TCOH in the liver, kidney, or serum, or in levels of TCA in serum after single or repeat dosing. After both single and repeat dosing, TCA levels in the liver were significantly lower in PPARα-null and hPPARα mice of both sexes compared with wild-type mice; in addition, with repeat dosing, the level of

hepatic TCA in hPPARα males was significantly lower than in PPARα-null males. Despite much lower levels of TCA, trichloroethylene-treated hPPARα mice of both sexes showed induction of CYP4A10 (a marker of PPARα activation) expression in the liver, and the mRNA levels were comparable to those seen in wild-type mice.

Summary

In summary, PCE appears to induce liver tumors in mice through multiple, potentially interdependent modes of action mediated largely by metabolites, including mutagenicity, epigenetic changes, cytotoxicity and oxidative stress, PPARα activation, and possibly also through other changes in gene expression. TCA appears to be an important hepatic metabolite but is probably not the only metabolite involved in hepatic effects of PCE. Available data show that the metabolism of PCE in the liver varies by sex, strain, and CYP2E1 and PPARα genotypes, and that several PCE metabolites are genotoxic. Based on limited data on PCE and studies of the related compound trichloroethylene, PPARα activation is probably not a necessary event for PCE-induced liver tumors but may influence both the metabolism and the nature of the hepatic effects induced. In addition to PPARα activation, PCE exposure also upregulates genes involved in ABC transporters, and downregulates nucleotide metabolism and mitochondrial-related genes. The relationship, if any, of these changes to the mode(s) of action for PCE liver carcinogenicity is unknown.

Kidney

(U.S. EPA 2012c) considered four potential modes of action for PCE-induced kidney cancers in rats: (1) genotoxicity; (2) α2u-globulin accumulation; (3) PPARα agonism/peroxisome proliferation; and (4) cytotoxicity not related to α2u-globulin accumulation. (U.S. EPA 2012c) considered it likely that several mechanisms contribute to renal carcinogenesis, but found evidence insufficient to draw further conclusions, whereas (IARC 2014) concluded that genotoxicity resulting from PCE metabolites in the kidney was the most likely mechanism for kidney cancers based on data available at the time of their review.

Role of metabolism

(Irving and Elfarra 2013) reviewed the available literature and concluded that the nephrotoxicity and nephrocarcinogenicity of PCE are mediated primarily through β-lyase-dependent bioactivation of the cysteine S-conjugate metabolite TCVC. The steps involved are as follows: PCE is conjugated to GSH in the liver to form TCVG; TCVG is processed into the cysteine conjugate (TCVC) in the kidney, bile duct epithelium, intestinal lumen, or bile canalicular membrane of hepatocytes; TCVC enters the circulatory system and is translocated to the kidney; and β-lyase acts on TCVC to form dichlorothioketene, a reactive electrophilic sulfur species. While TCVC has been found to be mutagenic in the Ames Salmonella mutagenicity assay, the addition of an inhibitor of β-lyase to the test system has been found to reduce the mutagenicity of TCVC, suggesting that the β-lyase-derived metabolites are primarily responsible for the mutagenicity of TCVC.

TCVC may be N-acetylated in the kidney to form the mercapturic acid, NAcTCVC (Luo et al. 2019). Both TCVC and NAcTCVC may be further metabolized to form reactive sulfoxides (Luo et al. 2019). TCVCS has been observed to have greater nephrotoxicity than TCVC (Elfarra and Krause 2007); however, the mutagenic activity of TCVCS in Salmonella is 30-fold lower than that of TCVC (Irving and Elfarra 2013).

In a study comparing glutathione-pathway metabolites of PCE in male mice of 45 different strains administered PCE as a single gavage dose of 1,000 mg/kg, area under the kidney tissue concentration-time curves (AUC) estimates for TCVG, TCVC, and NAcTCVC varied by at least 29-fold across the strains (Luo et al. 2019), demonstrating marked variability in the metabolism of PCE. Tissue concentrations of metabolites of the GSH pathway (liver TCVG, serum TCVG, liver NAcTCVC, and kidney NAcTCVC) were found to be significantly correlated with increased kidney levels of Kim-1 (kidney injury molecule-1), a protein marker of proximal tubular injury (Luo et al. 2019), supporting a link between this metabolic pathway and kidney toxicity.

PCE is also subject to oxidation, yielding TCA. Zhou et al. (2017) found quantifiable concentrations of TCA in the kidneys of mice at single gavage doses of 300 mg/kg and higher. TCA levels in the kidney were highly correlated with dose-related gene expression changes, including those related to peroxisomal fatty acid β oxidation, in the kidney.

Genotoxicity in the kidney

As discussed above under Section 3.2.3.2.1, several metabolites of PCE are genotoxic, while the parent compound itself shows little to no genotoxic activity in the absence of metabolic activation. The evidence for genotoxicity of the primary renal metabolites of PCE is stronger than that for hepatic metabolites, as reflected in the IARC conclusion that genotoxicity was the likely mode of action for the renal tumors. Specifically, the renal metabolites TCVG, TCVC, TCVCS, and NAcTCVC have all shown mutagenic activity in vitro. The mutagenicity of TCVG appears to depend on further metabolism via cysteine conjugation, while NAcTCVC is mutagenic following deacetylation (U.S. EPA 2012c), suggesting that conversion to TCVC may be necessary for the mutagenic activity of these two compounds. TCVC is mutagenic without metabolic activation in cell systems with β-lyase activity, and the mutagenic action is blocked by inhibition of β-lyase (Irving and Elfarra 2013), indicating that β-lyase-derived metabolites appear to be primarily responsible for the mutagenicity of TCVC. Species- and sex-related differences in the activities of β-lyase and other enzymes in the glutathione pathway may explain the sex- and species-specific renal carcinogenicity of PCE. As noted earlier, metabolic differences among strains resulted in at least 29-fold differences in AUC estimates for TCVG, TCVC, and NAcTCVC in the kidneys of male mice of 45 strains exposed to PCE (Luo et al. 2019). There is also general positive epidemiological evidence (not kidney-specific) of genotoxicity from chronic PCE exposure in humans (Section 3.2.3.2.1).

A2u-Globulin accumulation

Accumulation of α2u-globulin was considered as a mode of action for PCE-induced kidney cancer. This mode of action is unique to the male rats because female rats and other mammalian species do not accumulate α2u-globulin in the kidney. (U.S. EPA 2012c) hypothesized the following sequence of key events: excessive accumulation of α2u-globulin-containing hyaline droplets in renal proximal tubules, cytotoxicity and single-cell necrosis of tubule epithelium, sustained regenerative tubule cell proliferation, development of intralumenal granular casts containing sloughed cellular debris associated with tubule dilatation and papillary mineralization, foci of tubule hyperplasia in convoluted proximal tubules, and formation of renal tubule tumors.

Evidence of hyaline droplet nephropathy has been observed in male rats exposed to PCE (Bergamaschi et al. 1992; Green et al. 1990; Goldsworthy et al. 1988). Male F344 rats administered PCE via gavage at 1,000 mg/kg-day for 10 days showed increases in α2u-globulin, protein droplet accumulation, crystalloid accumulation, and cell replication in proximal tubules (Goldsworthy et al. 1988). The

increased cell replication, which was correlated with α2u-globulin accumulation and occurred in the same segment of the proximal tubule, is suggestive of a link between α2u-globulin accumulation and kidney tumors (U.S. EPA 2012c). Accumulation of α2u-globulin was also observed in the kidneys of male rats exposed by gavage to PCE at 500 mg/kg-day for 4 weeks (Bergamaschi et al. 1992). (Green et al. 1990) observed increased hyaline droplets in the proximal tubules of male rats exposed by gavage to PCE at 1,500 mg/kg-day for 42 days, as well as in male rats exposed by inhalation to PCE at 1,000 ppm for 10 days. Formation of granular tubular casts and evidence of tubular cell regeneration were also observed in rats dosed with PCE at 1,500 mg/kg-day for 42 days (Green et al. 1990). However, accumulation of α2u-globulin was not observed in the kidneys of male rats exposed by inhalation to 400 ppm for 6 hours/day for 28 days (Green et al. 1990), although (U.S. EPA 2012c) notes that recovery may have occurred during the 18-hour period between the final exposure and sacrifice. It is also possible that a longer exposure at this concentration might be required for accumulation of α2u-globulin.

(U.S. EPA 2012c) noted that α2u-globulin accumulation in response to PCE exposure has only been observed at doses higher than those associated with kidney tumors. In addition, non-neoplastic kidney lesions are not exclusively observed in male rats, as they have also been observed in female rats and male and female mice, in which α2u-globulin accumulation does not occur. In addition, nephrotoxicity has been observed in male and female rats and mice without hyaline droplet formation. (U.S. EPA 2012c) concluded that there are insufficient data to demonstrate that PCE-induced renal cancers are caused by α2u-globulin accumulation.

PPARα agonism/peroxisome proliferation

Another possible mode of action for kidney cancer examined by (U.S. EPA 2012c) is PPARα agonism/peroxisome proliferation. The following steps are hypothesized: activation of the PPARα receptor by one or more reactive metabolites of PCE (e.g., TCA), resulting in alterations in cell proliferation and apoptosis, followed by clonal expansion of initiated cells (U.S. EPA 2012c).

In an in vitro study, PPARα derived from humans and mice was found to be activated by PCE metabolites dichloroacetate and trichloroacetate, although not by PCE itself (Maloney and Waxman 1999).

In vivo, the activity of PCO, a marker for peroxisomal β-oxidation, was found to be increased (1.2 to 1.6-fold) in pooled kidneys of mice exposed to PCE by inhalation (6 hours/day) at 200 ppm for 28 days or 400 ppm for 14-28 days, significantly increased (1.3-fold) in male rat kidneys at 200 ppm at 28 days but not at 400 ppm, and significantly increased (1.2 to 1.6-fold) in female rat kidneys at 200 ppm at 28 days or 400 ppm at 14-28 days; however, there was no effect on renal catalase activity in rats or mice and no peroxisome proliferation was observed in rat or mouse kidney at microscopic examination (Odum et al. 1988). PCO activity was also increased in the kidneys of male rats (1.7-fold, not significant) and male mice (2.3-fold, significant) administered PCE by gavage at 1,000 mg/kg-day for 10 days (Goldsworthy and Popp 1987). In addition, mice treated with a single dose of 1,000 mg/kg PCE showed increased mRNA expression of PPARα-responsive genes in kidney tissue (Luo et al. 2019). Similarly, by measuring gene expression in the kidney, (Zhou et al. 2017) observed dose-dependent induction of genes associated with peroxisomal fatty acid β-oxidation pathways in a manner in mice administered a single dose of PCE.

Overall, only modest effects on PPARα-activation, as indicated by peroxisomal enzyme activity, have been observed after PCE exposure at doses exceeding those associated with kidney tumors (Odum et al.

1988; Goldsworthy and Popp 1987). (U.S. EPA 2012c) concluded that there is no evidence for PCE (or other compounds) that causally links PPARα-activation to kidney tumorigenesis.

Cytotoxicity not related to α2u-globulin accumulation

(U.S. EPA 2012c) also examined renal cytotoxicity as a possible mode of action for kidney cancer. It was suggested that sustained cytotoxicity and necrosis cause activation of repair processes and cellular regeneration that may lead to renal neoplasms. Reactive metabolites of PCE, including TCVC and TCVG, produced upon glutathione conjugation are known to result in kidney toxicity (U.S. EPA 2012c). TCVC has been observed to cause dose-related cytotoxicity, measured by release of lactate dehydrogenase, in a porcine renal cell line (Vamvakas et al. 1989a) and in renal proximal tubule cells isolated from male rats (Vamvakas et al. 1989b). 1,2,2-trichlorovinylthiol, an unstable thiol produced by cleaving TCVC, may give rise to a highly reactive thioketene, which can form covalent adducts with cellular nucleophiles (U.S. EPA 2012c; Vamvakas et al. 1989b). In another in vitro study, (Lash et al. 2002) observed that PCE and its TCVG metabolite caused increased acute renal cytotoxicity in isolated renal cortical cells from rats with the effect being greater in cells isolated from males, as compared to females. In addition, TCVC was found to cause acute cytotoxicity in primary cultures of proximal tubular cells from rat and human kidneys (IARC 2014).

Observed signs of non-neoplastic kidney toxicity in rodents exposed to PCE in vivo have included: karyomegaly of the proximal tubules in male and female rats and mice (Jonker et al. 1996; JISA 1993; NTP 1986a), tubular cell hyperplasia in male and female rats (NTP 1986a), nephrosis (non-inflammatory degenerative kidney disease) in female mice (NTP 1986a), casts in male and female mice (NTP 1986a), atypical tubular dilation of the proximal tubules in male and female rats and mice (JISA 1993), changes in urinary markers related to kidney function (total protein and N-acetyl-β-glucosaminidase) in female rats (Jonker et al. 1996), glomerular nephrosis and degeneration in male and female mice (Ebrahim et al. 1996), exacerbation of chronic renal disease in male rats (JISA 1993), and toxic nephropathy in male and female rats and mice (NCI 1977). Male rats exposed to TCVC or TCVCS, metabolites of PCE, by a single intraperitoneal injection showed visible acute renal tubular necrosis, intratubular casts and interstitial congestion and hemorrhage (TCVCS only), increased urinary glucose concentration and ɣ-glutamyl transpeptidase activity, and increased blood urea nitrogen (TCVCS only), with TCVCS exhibiting greater nephrotoxicity than TCVC (Elfarra and Krause 2007).

Although nephrotoxicity has been observed in both sexes of rats and mice, renal tubular neoplasia have been observed only in male rats (NTP 1986a). In addition, signs of non-neoplastic kidney damage were observed in rats and mice of both sexes in the early stages of the (NTP 1986a) inhalation study, suggesting that animals of both species and sexes surviving to scheduled termination had sustained nephrotoxicity for the majority of the study period; however, neoplasms were only observed in male rats. This is inconsistent with nephrotoxicity being the primary mode of action for kidney neoplasms.

In humans, symptoms of renal dysfunction, including proteinuria and hematuria, have been observed in patients administered PCE via inhalation as an anesthetic (IARC 2014). One study found an increased incidence (>2.5-fold) of end-stage renal disease in dry cleaning workers exposed to PCE by inhalation. Urinary markers of renal damage were found to be altered in dry cleaning workers by Mutti et al. (1992); effects included increased prevalence of abnormal values for brush-border antigens, a higher geometric mean concentration of brush-border antigens, and a higher concentration of tissue non-specific alkaline phosphatase in urine. In addition, dry cleaning workers were observed to have significantly increased urinary concentrations of β-glucuronidase and lysozyme, indicators of kidney

function (IARC 2014). Effects on urinary indicators of renal tubule function, including significantly increased prevalence of abnormal values of retinol-binding protein (Mutti et al. 1992) and a higher geometric mean concentration of retinol-binding protein (IARC 2014) were observed in two of six studies of dry cleaning workers.

Summary

In summary, available data provide evidence for mutagenicity as a likely mode of action for renal carcinogenicity induced by PCE, while data supporting other candidate modes of action are more limited and have unclear causal links to tumorigenesis.

Blood

There is no specific information pertaining to potential modes of action for PCE-induced hematopoietic or immune system cancers. Limited data from studies investigating immunotoxicity suggest that PCE exposure can alter white cell counts and immune system markers in humans and in mice (U.S. EPA 2012c). A more recent in vitro study showed that PCE exposure increased the mRNA expression of cytokines IL-6 and IL-10 in murine macrophages, albeit at cytotoxic concentrations (Kido et al. 2013). IL-6 is a pro-inflammatory cytokine but is involved in other reactions as well; IL-10 is an anti-inflammatory cytokine that may have been elevated as a response to the increase in IL-6. The role, if any, of these immune system perturbations in carcinogenicity induced by PCE is unknown. (U.S. EPA 2012c) noted that evidence for effects of PCE on hemolysis and bone marrow function in mice provides some support for a leukemogenic effect in rodents but concluded that data were inadequate to establish a mechanism for mononuclear cell leukemia in rats exposed to PCE.

Overall Conclusions

Overall, the reasonably available evidence for all three tumor sites likely supports a complex MOA, with multiple contributing mechanisms of varying significance. There is evidence of kidney and liver-specific genotoxicity from PCE metabolites and evidence of PCE genotoxicity in humans from epidemiological studies. Induction of other non-genotoxic mechanisms including cytotoxicity and PPARα activation are supported by various evidence, however there is insufficient causal link between these pathways and tumorigenesis. Induction of these pathways is often at doses higher than which have been shown to promote tumorigenesis, and the effects are not consistent across sex, dose, and time relative to the results of cancer bioassays. While α-2u-globulin-based kidney toxicity in male rats is not relevant to humans and the PPARα pathway is of reduced significant in humans, the reasonably available data does not support a clear indication that these are major contributors to the tumorigenesis observed in animal cancer bioassays. Therefore, animal carcinogenicity data is considered relevant to humans.

According to EPA's 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA 2005a), "a linear extrapolation approach is used when the mode of action information is supportive of linearity or mode of action is not understood". The evidence for at least a significant contribution of a genotoxic MOA supports use of the low-dose linear assumption, while other mechanisms are not well-enough supported to suggest a potential threshold approach. Therefore, EPA used the low-dose linear default non-threshold assumption for derivation of cancer slope factors (Section 3.2.5.3.3).

3.2.4 Weight of Scientific Evidence

3.2.4.1.1 Acute Toxicity

Acute exposures to PCE result in neurotoxicity effects that include central nervous system depression and visual processing, including loss of consciousness which can result in death. These acute

neurological effects are supported by both human and animal studies as described below in Section 3.2.4.1.2. There is only limited available information concerning acute irritation and hepatic effects and the available evidence is insufficiently quantitative for use in dose-response analysis. Therefore, acute toxicity other than neurological effects were not carried forward to dose-response analysis.

3.2.4.1.2 Neurotoxicity

The hazard database includes reported human evidence of visual deficits (Getz et al. 2012; Schreiber et al. 2002; Gobba et al. 1998; Cavalleri et al. 1994; Altmann et al. 1990), impaired cognition (Echeverria et al. 1995; Seeber 1989), increased risky behaviors with associated head injuries following prenatal or early childhood PCE exposure (Aschengrau et al. 2016a; Aschengrau et al. 2011), and decreased math test scores (Stingone et al. 2016). Ambiguous or conflicting evidence was found for increased risk of neurodegenerative diseases (Bove et al. 2014b; Goldman et al. 2012) and autism spectrum disorders (Talbott et al. 2015; von Ehrenstein et al. 2014; Roberts et al. 2013; Kalkbrenner et al. 2010). Clinical, biochemical, and neurophysiological signs of neurotoxicity were observed in adult rodents (Mattsson et al. 1998; Jonker et al. 1996; Tinston 1994; Kjellstrand et al. 1984) as well as indications of impaired neurobehavior and motor function in developing rats (Nelson et al. 1979). A single 4-week inhalation study in rats did not observe any clinical signs of neurotoxicity (Boverhof et al. 2013), however that study was primarily focused on immunological endpoints. Overall, based on numerous identified functional outcomes in human studies supported by both clinical and mechanistic findings in animals, neurotoxicity following PCE exposure is supported by the weight of evidence. Based on consistent supporting evidence and sufficient quantitative information, the endpoint of impaired visual function (including delayed neurological signaling, color confusion, and visual memory) was carried forward for dose-response analysis to represent the neurotoxicity hazard domain.

3.2.4.1.3 Kidney Toxicity

Mutti et al., (1992) and several other epidemiological studies from (U.S. EPA 2012e) suggest likely proximal tubular injury following long-term occupational exposure to PCE. Additionally, multiple animal studies on both rats and mice demonstrated renal effects in both sexes, including increased kidney weights, tubular histopathology, and other indications of kidney toxicity (Jonker et al. 1996; Tinston 1994; JISA 1993; NTP 1986b; NCI 1977). Since the publication of the IRIS Assessment, a single 4-week inhalation study in rats did not observe any effects on kidney weight or histology (Boverhof et al. 2013). Overall, based on effects seen in multiple studies in both animals and humans, kidney toxicity following PCE exposure is supported by the weight of evidence. Based on consistent supporting evidence and sufficient quantitative information, the endpoints of urinary biomarkers for nephrotoxicity and nuclear enlargement of proximal tubules were carried forward for dose-response analysis to represent the kidney hazard domain.

3.2.4.1.4 Liver Toxicity

The human literature database is limited, with some indication that PCE exposure affects human liver function as well as evidence of negative associations (Silver et al. 2014; U.S. EPA 2012c). The animal database shows very strong support for liver toxicity following PCE exposure, with reports of necrosis, vacuolization, inflammation, increased liver weight, biochemical markers, and other indicators of liver toxicity in both rats (Jonker et al. 1996; JISA 1993) and mice (Buben and O'Flaherty 1985). A four-week inhalation study in rats (Boverhof et al. 2013) that was published after the IRIS Assessment also reported hepatic effects (increased relative liver weights and hepatocellular hypertrophy) at the highest dose. Overall, based on strong and consistent evidence in animals, liver toxicity following PCE exposure is supported by the weight of evidence. Based on consistent supporting evidence and sufficient quantitative information, the endpoints of increased angiectasis, increased degeneration/necrosis, and

increased liver/body-weight ratio were carried forward for dose-response analysis to represent the liver hazard domain.

3.2.4.1.5 Reproductive/Developmental Toxicity

The EPA IRIS Assessment (U.S. EPA 2012c) reported strong epidemiological evidence of adverse pregnancy outcomes in women associated with PCE exposure. Human evidence was too limited to conclude anything about sperm quality or infertility (U.S. EPA 2012c; Eskenazi et al. 1991). Data from multiple human studies indicate an increased risk of spontaneous abortion (U.S. EPA 2012c). Animal evidence supports effects on both male and female reproductive systems (U.S. EPA 2012c; Tinston 1994; Beliles et al. 1980) as well as developmental outcomes (U.S. EPA 2012c; Carney et al. 2006). There were not any relevant studies published after the IRIS Assessment. Overall, evidence of both male and female reproductive effects in animals as and associations between exposure and female reproductive in humans along with indications of developmental effects in both study types, both reproductive and developmental toxicity following PCE exposure are supported by the weight of evidence. Based on consistent supporting evidence and sufficient quantitative information, the reproductive endpoint of reduced sperm quality and the developmental endpoints of decreased fetal/placental weight, developmental neurotoxicity, and skeletal effects were carried forward for dose-response analysis to represent the reproductive/developmental hazard domain.

3.2.4.1.6 Immune System and Hematological Effects

Immune System Effects

The EPA IRIS Assessment (U.S. EPA 2012c) summarized a large dataset of human studies, some of which examined PCE as part of a class of solvents, as well as a few short-term animal studies. While some indications of immune effects were observed, the available data was not robust or consistent enough to conclude that immune effects are likely to result from PCE exposure. Studies published after the IRIS Assessment provide conflicting evidence of immunotoxicity based on no effects observed on immune organs (Boverhof et al. 2013) and positive indications of allergic reaction (Seo et al. 2012) following PCE exposure. Overall, based on the absence of consistently observed effects in animals or humans, the data for immune effects is inconclusive is not supported by the weight of evidence. Therefore, this hazard domain was not carried forward for dose-response analysis.

Hematological Effects

Decreased red blood cells and hemoglobin levels with increased total white blood cell and lymphocyte counts were observed in a single occupational epidemiology study as described in the EPA IRIS Assessment (U.S. EPA 2012e). Evidence of anemia was observed in mice but not rat studies (U.S. EPA 2012e) and the more recent 4-week inhalation study published after the IRIS assessment (Boverhof et al. 2013) also did not observe any hematological effects. Overall, while there is some indication of hematological evidence in humans and mice, the human data is limited and conflicting results were observed in rats and mice. Therefore, hematological effects following PCE exposure is insufficiently supported by the weight of evidence and this hazard domain was not carried forward for dose-response analysis.

3.2.4.1.7 Cancer

Weight of Evidence Conclusion

In accordance with EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA 2005a), PCE is considered “likely to be carcinogenic in humans” by all routes of exposure based on conclusive evidence in animals and suggestive evidence in humans.

There is conclusive evidence of the carcinogenicity of PCE, administered by ingestion or inhalation, in rats and mice. The most notable findings were statistically significant increases in the incidence of liver tumors (hepatocellular adenomas and/or carcinomas) in male and female B6C3F1 and Crj:BDF1 mice exposed by inhalation (JISA 1993; NTP 1986a) and male and female B6C3F1 mice exposed by ingestion (NCI 1977). Significant increases were also observed in the incidences of mononuclear cell leukemia (MCL) in male and female rats (F344/N and/or F344/DuCrj) exposed to PCE by inhalation (JISA 1993; NTP 1986a). Additional findings potentially related to treatment included increases in testicular interstitial cell tumors and renal tubular adenomas and adenocarcinomas in male F344/N rats exposed by inhalation (NTP 1986a), brain gliomas in male and female F344/N rats exposed by inhalation (NTP 1986a), hemangiosarcomas/ hemangiomas in male Crj:BDF1 mice exposed by inhalation (JISA 1993), and adenomas of the Harderian gland in male Crj:BDF1 mice exposed by inhalation (JISA 1993).

There is a pattern of evidence associating PCE exposure with several types of cancer, specifically bladder cancer, NHL, and MM. Additional data were available showing weaker support for cancers at other sites, including esophageal, lung, and blood (lymphoma). Studies provide more limited support for associations with bladder and breast cancer, with little or no support for associations with kidney, esophagus, or liver cancer or MM, and no useful information for cervical cancer.

Available data indicate that multiple modes of action are likely to be involved in PCE-induced liver cancers in male and female mice and possibly renal cancers in male rats as well (Section 3.2.3.2.4). Metabolism is a key event in the modes of action for both liver and kidney carcinogenicity. Importantly, there appear to be marked sex- and strain-related differences, and possibly species differences, in the degrees of oxidative and glutathione conjugative metabolism of PCE, which could explain the species and sex specificity of liver and kidney tumors induced by this compound. Several PCE metabolites originating from the glutathione pathway are mutagenic, particularly the electrophilic sulfur species that result from β-lyase activation of TCVC in the kidney. There is less evidence for non-mutagenic modes of action for kidney carcinogenicity associated with PCE exposure; available data do not support significant roles for α-2u globulin accumulation, cytotoxicity unrelated to α-2u globulin accumulation or PPARα agonism in renal tumor formation. In contrast, there is evidence suggesting that several modes of action, in addition to mutagenicity, may be operant in the liver, including: epigenetic changes leading to oncogene activation; cytotoxicity, inflammation, and oxidative stress; activation of PPARα leading to perturbations in cell proliferation or apoptosis; and other changes in gene expression that may influence cellular energetics, growth, and/or cell cycle. The importance of any one of these modes of action likely depends on dose, species, sex, and strain, given the variability in and importance of PCE metabolism to the various modes of action.

3.2.5 Dose-Response Assessment

3.2.5.1 Selection of Studies for Dose-Response Assessment

Dose-response analysis started with the consideration of all acceptable toxicity studies identified in the prior sections and selection of the studies that reported both adverse effects and data amenable to dose-response assessment. Dose-response assessment was organized into 5 domains: (1) acute toxicity, (2) neurotoxicity, (3) kidney toxicity, (4) liver toxicity and (5) reproductive/developmental toxicity.

3.2.5.1.1 Non-Cancer Toxicity from Acute/Short-Term Exposure

Based on the weight of the scientific evidence evaluation neurotoxicity was selected for dose-response analysis for effects from acute/short-term exposure. Quantitative data amenable to dose-response

assessment from human studies (controlled experiments) are available for this endpoint. Studies available for evaluating acute exposures include controlled human exposures (Altmann et al. 1990). Data are also available from animal studies to support this health effect domain following acute exposure. The human studies are considered adequate and are preferable to animal studies.

In the study by Altmann et al. (1990), male volunteers were exposed to PCE at 10 or 50 ppm, 4 hours/day for 4 days. At 50 ppm, increased latencies in pattern reversal visual-evoked potential (p<0.05) were observed. No effects on brainstem auditory-evoked potential were noted at either concentration. Because faint odor was reported by 33% of the subjects at 10 ppm and 29% of the subjects at 50 ppm on the first day of testing, and by 15% of the subjects at 10 ppm and 36% of the subjects at 50 ppm on the last day of testing, the investigators concluded that only a few subjects could identify their exposure condition. PCE in the blood increased with exposure duration, and based on linear regression, PCE was associated with increased pattern reversal visual-evoked potential latencies (r=-0.45, p<0.03) (Altmann et al. 1990). EPA considered a no-observed-adverse-effect level (NOAEL) of 10 ppm for exposures of 4 hours/day. The study scored a medium in data quality.

Other studies assessed different endpoints in the spectrum of neurotoxicity effects. Hake and Stewart (1977) exposed 4 male subjects sequentially to 0, 20, 100, and 150 ppm (each concentration 1 week) PCE 7.5 hours/day for 5 days. Changes in flash-evoked potentials or equilibrium tests were not observed. Subjective evaluation of EEG (electroencephalogram) scores suggested cortical depression in subjects exposed at 100 ppm. Decreases in the Flanagan coordination test were observed at ≥100 ppm. Rowe et al. (1952) exposed 6 volunteers to 106 ppm PCE for 1 hr. Eye irritation and a slight fullness in the head was noted by one subject, but other neurotoxicity endpoints were not evaluated.

The National Research Council (NRC) (2010) review of the PCE IRIS assessment included a recommendation of five studies for consideration in deriving the reference concentration (RfC) (Boyes et al. 2009; Gobba et al. 1998; Echeverria et al. 1995; Cavalleri et al. 1994; Altmann et al. 1990). Of these studies recommended for consideration by NRC two are acute studies [the human chamber study of Altmann et al. (1990) and the rodent study of Boyes et al. (2009)]. These were judged by EPA in the IRIS assessment to be supportive, but were not considered further for deriving candidate RfCs because of the preference to use quality studies of chronic, human exposures over studies of acute exposures. For the dose-response assessment of effects from acute exposures the Altmann et al. (1990) study in humans is preferred rather than the Boyes et al. (2009) study in rodents.

Based on these considerations, EPA chose the effects observed in Altmann et al. (1990) for dose-response analysis of acute effects. These studies identified increased latencies for pattern reversal visual-evoked potentials at 50 ppm and a NOAEL of 10 ppm.

3.2.5.1.2 Non-Cancer Toxicity from Chronic Exposure

The studies presented below are the principal studies containing adequate quantitative dose-response information for various endpoints within each health domain. See Section 3.2.5.4 for selection of the most representative studies within each domain.

Neurotoxicity

Based on the review in the EPA IRIS Assessment for PCE (U.S. EPA 2012c) and NRC (2010), two studies, Cavalleri et al. (1994) and Echeverria et al. (1995), are considered the principal studies for the evaluation of chronic neurotoxicity. Endpoints selected were reaction time measures (Echeverria et al.

1995), cognitive changes (Echeverria et al. 1995), and visual function changes (Cavalleri et al. 1994). EPA's data quality evaluations of these studies were both medium. The 2012 Perchloroethylene IRIS Assessment (U.S. EPA 2012c) additionally calculated the midpoint of the range from these two studies, and this value was also brought forward to dose-response analysis.

Kidney

Two acceptable studies were identified that contained adequate dose-response information: (Mutti et al. 1992) and (JISA 1993). Mutti et al. (1992) was an epidemiological study that identified urinary markers of neprotoxicity. JISA (1993) observed nuclear enlargement of proximal tubules in both rats and mice. Mutti et al. (1992) scored a Medium in data quality and JISA (1993) scored a High.

Liver

Three studies were considered for dose-response analysis of liver effects. The same JISA (1993) study that examined kidney effects also observed increased liver angiectasis (extreme dilation of blood or lymph vessels) in mice. An NTP study (1986b) that also scored high in data quality identified increased liver degeneration and necrosis in mice, while the medium-quality study (Buben and O'Flaherty 1985) reported increased liver/body weight ratio in mice following PCE administration.

Reproductive/Developmental

A single reproductive study reported adequate dose-response information. Beliles et al. (1980) identified reduced sperm quality following 5 days of PCE exposure in mice. The study scored a high in data quality.

For developmental effects, three relevant studies were identified. Nelson et al. (1979) identified decreased weight gain and developmental neurotoxicity in the form of altered behavior and changes in brain acetylcholine. The study only scored a Low in data quality, however it was still considered for dose-response analysis because it is the only identified study with adequate dose-response information relating to functional and molecular indicators of developmental neurotoxicity, and the CNS is an important target of perchloroethylene. The other two studies both scored a High in data quality and were also utilized for dose-response analysis. Tinston et al. (1994) identified increased neonatal pup death and CNS depression in a two-generation study, and (Carney et al. 2006) observed decreased fetal/placental weight and skeletal effects in a short-term developmental toxicity study.

3.2.5.1.3 Cancer

As discussed in the Weight of Evidence Section 3.2.4.1.7, based on EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA 2005a). PCE is characterized as "likely to be carcinogenic in humans by all routes of exposure," based on conclusive evidence in mice and rats and suggestive evidence in humans. No available human studies of cancer were found to be suitable for dose-response assessment. Therefore, the following dose-response assessment is based on data from rodent bioassays. Multiple MOAs for PCE carcinogenicity were considered in the MOA Section 3.2.3.2.4 specific to each tumor type. Overall, the tumors reported in rodent bioassays are considered relevant to humans and human cancer risks are estimated from the rodent dose-response data.

As discussed in Section 3.2.3.2.3 three chronic exposure studies in rats and mice include an oral gavage study in mice and female rats by the National Cancer Institute (NCI 1977) and two inhalation studies in mice and rats (JISA 1993; NTP 1986b) established that the administration of PCE, either by ingestion or by inhalation to sexually mature rats and mice, results in increased incidence of tumors. Mouse liver tumors (hepatocellular adenomas and carcinomas) and rat mononuclear cell leukemia (MCL) were

reported in both sexes in two lifetime inhalation bioassays employing different rodent strains (JISA 1993; NTP 1986b), and mouse liver tumors were also reported in both sexes in an oral bioassay (NCI 1977). Tumors reported in a single inhalation bioassay include kidney and testicular interstitial cell tumors in male F344 rats (NTP 1986b), brain gliomas in male and female F344 rats (NTP 1986b), and hemangiomas or hemangiosarcomas in male Crj:BDF1 mice (JISA 1993). The NCI (1977) study was considered to be inconclusive because of the high incidence of respiratory disease, and high mortality with PCE exposure. See (U.S. EPA 2012e) for more discussion.

All three bioassays (JISA 1993; NTP 1986b; NCI 1977) showed increases in hepatocellular tumors in male and female mice. Hemangiomas also increased in male mice and MCL increased in both sexes of rats. The data is summarized in Table 3-4 below.

Despite the positive results, the NCI (1977) study was considered to be inconclusive because of the high incidence of respiratory disease, and high mortality with PCE exposure. Therefore considered the JISA (1993) and NTP (1986b) studies for dose-response analysis. Both studies scored a High for data quality, however (JISA 1993) examined an additional dose level and covers a broader dose range. Therefore, the JISA (1993) study was selected for use in dose-response analysis and POD derivation. It is **bolded** in Table 3-4 below.

Table 3-4. Tumor incidence in mice exposed to PCE

Bioassay	Doses/Exposures: Administered	Doses/Exposures: Continuous Equivalent	Sex	Body Weight[a] (kg)	Survival-adjusted tumor incidence[b] (%)	
Hepatocellular adenomas or carcinomas						
NCI (1977)[c] $B6C3F_1$ mice Gavage: 5 d/wk, 78 wk	Vehicle control 450 mg/kg-day 900	0[e] mg/kg-day 332 663	Male	0.030	2/20 32/48 27/45	(10) (67) (60)
	Vehicle control 300 mg/kg-day[d] 600	0[e] mg/kg-day 239 478	Female	0.025	0/20 19/48 19/45	(0) (40) (42)
NTP (1986b) $B6C3F_1$ mice Inhalation: 6 hr/d, 5 d/wk, 104 wk	0 ppm 100 200	0 ppm 18 36	Male	0.037	17/49 31/47 41/50	(35) (70) (82)
	0 ppm 100 200	0 ppm 18 36	Female	0.032	4/45 17/42 38/48	(9) (40) (79)
JISA(1993) **Crj:BDF1 mice** **Inhalation:** **6 hr/d,** **5 d/wk,** **104 wk**	0 ppm 10 50 250	0 ppm 1.8 9.0 45	Male	0.048	13/46 21/49 19/48 40/49	(28) (43) (40) (82)
	0 ppm 10 50 250	0 ppm 1.8 9.0 45	Female	0.035	3/50 3/47 7/48 33/49	(6) (6) (15) (67)
Hemangiosarcomas[e], liver or spleen						

Bioassay	Doses/Exposures		Sex	Body Weight[a] (kg)	Survival-adjusted tumor incidence[b] (%)	
	Administered	Continuous Equivalent				
JISA (1993) Same conditions as above	0 ppm 10 50 250	0 ppm 1.8 9.0 45	Male	0.048	4/46 2/49 7/48 11/49	(4) (2) (13) (18)
Mononuclear cell leukemia (MCL)						
NTP (1986b) F344/N rats Inhalation: 6 hr/d, 5 d/wk, 104 wk	0 ppm 200 400	0 ppm 36 71	Male	0.44	28/50 37/48 37/50	(56) (77) (74)
	0 ppm 200 400	0 ppm 36 71	Female	0.32	18/50 30/50 29/50	(36) (60) (58)
JISA (1993) F344/CuCrj rats Inhalation: 6 hr/d, 5 d/wk, 104 wk	0 ppm 50 200 600	0 ppm 9 36 110	Male	0.45	11/50 14/50 22/50 27/50	(22) (28) (44) (54)
	0 ppm 50 200 600	0 ppm 9 36 110	Female	0.3	10/50 17/50 16/50 19/50	(20) (34) (32) (38)

Note: Data sets carried through dose-response modeling shown in bold. Data is from Table 5-13 and 5-15 in (U.S. EPA 2012e).

[a]Average body weight reached during adulthood.

[b]Animals dying before the first appearance of the tumor of interest but no later than Week 52 were omitted from the totals because these animals were presumed not to have adequate time on study to develop tumors.

[c]No adenomas were reported in this study.

[d]Gavage doses listed were increased after 11 weeks by 100 mg/kg-day in each low-dose group or by 200 mg/kg-day in each high-dose group. Animals surviving the 78-week exposure period were observed until Week 90 study termination. Lifetime average daily (administered) doses (LADDs) were calculated as follows:

LADD (mg/kg-day) = Cumulative administered dose (mg/kg)/(total days on study)
= {[(initial dose rate × 11 weeks) + (later dose rate × 67 weeks)]/90 weeks}
× 5/7 (days)

[e]These tumors were reported as hemangioendotheliomas in the JISA (1993) report. The term has been updated to hemangiosarcoma. Note that these incidences do not match those tabulated in Tables 11 and 12 of the JISA report summary. The incidences reported here represent a tabulation of hemangioendotheliomas in liver or spleen from the individual animal data provided in the JISA report.

3.2.5.2 Potentially Exposed and Susceptible Subpopulations

TSCA requires the risk evaluation "determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost of other non-risk factors, including an unreasonable risk to a potentially exposures of susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." 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."

During problem formulation (U.S. EPA 2018d), EPA identified potentially exposed or susceptible subpopulations during the development and refinement of the life cycle, conceptual models, exposure scenarios, and analysis plan. In this section, EPA addresses the potentially exposed or susceptible subpopulations identified as relevant based on *greater susceptibility*. EPA addresses the subpopulations identified as relevant based on *greater exposure* in Section 2.4.3.

Factors affecting susceptibility examined in the available studies on PCE include lifestage, biological sex, genetic polymorphisms, race/ethnicity, preexisting health status, lifestyle factors, and nutrition status. PCE is lipophilic and accumulates in fatty fluids and tissues in the human body (Section 0). Additionally, the PCE half-life is substantially higher in adipose tissue compared to others (55-65 hours in adipose, <12-40 hours in others, see Section 3.2.2.1.3). Subpopulations that may have higher body fat composition, and therefore may be more highly exposed to sustained internal PCE concentrations/doses, include pubescent and adult women (including women of child-bearing age) as well as any individual with an elevated body-mass-index. Based on evidence of developmental toxicity from PCE exposure, pregnant women, the developing fetus and newborn infants are all considered highly susceptible subpopulations, and therefore women of childbearing age are susceptible by proxy. Effects on male fertility are more likely to present in older men, while kidney and liver effects are of most concern to subpopulations with pre-existing liver or kidney dysfunction. The partitioning of PCE to fatty tissue is of particular concern for those with fatty liver disease. Neurological endpoints are primarily related to visual function, pattern recognition, and memory. Therefore, subpopulations with poor vision or neurocognitive deficiencies may be especially susceptible to these hazards.

Variability in CYP metabolic capacity is generally believed to vary by approximately 10-fold among all humans, however individual variations in *in vitro* CYP2E1 activity as high as 20-50 fold have also been reported. Diagnoses of polymorphisms in carcinogen-activating and -inactivating enzymes and cancer susceptibility have been noted, and GST polymorphisms have been associated with increased risk of kidney cancer in the related chemical trichloroethylene. Co-exposure to other pollutants and drugs may also have either an activating or inhibitory effect on PCE-metabolizing enzymes (U.S. EPA 2012c).

3.2.5.3 Derivation of Points of Departure (PODs)

3.2.5.3.1 Non-Cancer PODs for Acute/Short-term Inhalation Exposure

Workers and consumers can be exposed to a single acute exposure to PCE 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. The duration of 4 hrs/day is based on the study conditions of Altmann et al. (1990). Longer durations of 8 hrs/day and 12 hrs/day are representative of typical work shifts and are used for occupational settings. For consumers, EPA also evaluated a 24-hr exposure to account for exposure scenarios when a user remains in the house after using a PCE-containing product, i.e., a consumer product used for a specific length of time, with subsequent exposure to dissipating concentrations of PCE in the indoor environment over the course of a day. Conversion of the acute PODs for different exposure durations are shown in Table 3-5.

Altmann et al. (1990) is a relatively well-conducted study of 10 volunteers each that identified increased latencies for pattern reversal visual-evoked potentials after 4 hrs/day for 4 days exposure to 50 ppm and no effects at 10 ppm. EPA's data quality evaluation rated this study medium quality. EPA used the NOAEC of 10 ppm. The ATSDR Toxicity Profile included this NOAEC among endpoints for derivation of the acute MRL (minimum risk level) (ATSDR 2019). The acute MRL is derived for exposures up to 14 days and additional information was considered for exposures longer than the 4 days of the Altmann

et al. (1990). This is consistent with how EPA is considering Altmann et al. (1990) for acute exposures to workers and consumers.

Table 3-5. Conversion of Acute PODs for Different Exposure Durations

Exposure Duration	POD	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
4 hrs/day duration of the study	10 ppm (68 mg/m^3)	Neurotoxicity increased latencies for pattern reversal visual-evoked potentials	UF_A=1; UF_H=10; UF_L=1 Total UF=10	Altmann et al. (1990)	Medium
8 hrs/day	5 ppm (34 mg/m^3)				
12 hrs/day	3.3 ppm (22 mg/m^3)				
24 hrs/day	1.7 ppm (11 mg/m^3)				

EPA applied a composite UF of 10 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 PCE. Some of the specific variabilities/uncertainties for PCE are accounted for with this UF_H include toxicokinetic differences.

3) **A LOAEC-to-NOAEC uncertainty factor (UF_L) of 1 -** The POD is based on a NOAEC so this factor is not needed.

3.2.5.3.2 Non-Cancer PODs for Chronic Inhalation Exposure

All chronic PODs were derived as 24hr Human Equivalent Concentration (HEC) values, with results from animal studies adjusted for continuous exposure based on the output from the PBPK model as presented in (U.S. EPA 2012e). All PODs are presented in Table 3-8.

Neurotoxicity

EPA identified LOAELs for color confusion from (Cavalleri et al. 1994) and impaired pattern recognition and reaction time in pattern memory from (Echeverria et al. 1995) as relevant endpoints for POD derivation. For the studies and endpoints selected, it was determined that PODs could not be derived using dose-response modeling (described in more detail in (U.S. EPA 2012e)). Therefore, the midpoint of the range of the two LOAELs from each study was also derived as a representative POD. This is consistent with the use of the midpoint for the reference concentration/dose in (U.S. EPA 2012e). For occupational human studies such as these, the HEC derivation also involved adjusting the breathing rate from 10 m^3/day over 8 hrs to 20m^3/day over 24 hrs, and multiplying the PODs by 5/7 to adjust from weekday working hours to continuous exposure (U.S. EPA 2012e).

EPA applied a composite UF of 100 for the inhalation benchmark MOE for neurotoxicity, 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) An 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, PCE.

3) A LOAEC-to-NOAEC uncertainty factor (UF_L) of 10
The POD is based on a LOAEC so this factor is needed.

4) Subchronic to chronic factor (UF_S) of 1
The data for these endpoints come from chronic studies covering greater than 10% of human lifetime, so an additional adjustment for shorter-duration studies is not required.

Alternative HEC for Occupational Scenarios

In addition to the HEC derived from the 2012 IRIS Assessment (U.S. EPA 2012e), EPA derived 8 hr HEC values for the above endpoints based on occupational exposure.

The 24 hr HEC as originally derived was applicable to the general population, who would be continuously exposed to PCE at a resting breathing rate. The data for these endpoints are from epidemiological studies of dry cleaning and laundry workers exposed to PCE. In order to account for increased breathing rate of workers (i.e. 10 m^3 over 8 hr as opposed to 20 m^3 over 24 hr, according to (U.S. EPA 2012e), EPA additionally derived 8 hr occupational HECs using the 8 hr LOAEC values from the original studies. 12 hr HECs were also derived based on adjustment from the 8 hr values for use with 12 hr Occupational Exposure Scenarios (OES). These additional derivations did not result in any change to the uncertainty factors.

Kidney

EPA identified a LOAEL from (Mutti et al. 1992) for urinary biomarkers along with NOAELs from (JISA 1993) for proximal tubule nuclear enlargement in both mice and rats. Cumulative UFs for the two NOAELs is 30, with a UF_H =10 for human uncertainty/variability and UF_A = 3 for interspecies toxicodynamic uncertainty/variability, because only toxicokinetic differences are captured by the PBPK model. The LOAEL from (Mutti et al. 1992) is a human study and therefore has a UF_A of 1, however it has an additional UF_L of 10 for being based on a LOAEL and therefore the cumulative UF is 100. All studies are of chronic duration, so UF_S = 1.

Liver

EPA identified three distinct liver endpoints in mice as suitable for dose-response analysis. The NOAEL from (JISA 1993) for increased angiectasis (abnormal dilation of blood vessels) has a cumulative UF of 30 based on UF_A and UF_H as described above. A LOAEL was obtained for increased liver

degeneration/necrosis from (NTP 1986b), resulting in a cumulative UF of 300 due to the added UF_L of 10. These two studies are of chronic duration, so $UF_S = 1$. A LOAEL for increased liver/body-weight ratio from subchronic data in (Buben and O'Flaherty 1985) has a cumulative UF of 3000 due to the added UF_L of 10 and $UF_S = 10$.

Reproductive/Developmental

A reproductive NOAEL for reduced sperm quality in mice was obtained from (Beliles et al. 1980). Despite being of only 5 days exposure, this duration this exposure duration covers the window of sperm production while the observation period up to 10 weeks covered the full period of spermatogenesis. Therefore, longer exposure would not be expected to result in additional sensitivity and $UF_S = 1$. The cumulative UF is 30 based on UF_A and UF_H as described above. PODs from three developmental toxicity studies in rats (Carney et al. 2006; Tinston 1994; Nelson et al. 1979) were derived. The durations were sufficient to cover the developmental window, so $UF_S = 1$ and cumulative UF= 30 based on NOAELs from animals as previously described.

3.2.5.3.3 Cancer Slope Factor Derivation

This section provides details of the dose-response modeling carried out for developing cancer risk values and is summarized from the EPA IRIS Assessment for PCE (U.S. EPA 2012c). This summary focuses on hepatocellular tumors, the tumor type that was observed in all three animal bioassays and was the basis of the cancer slope factors in the EPA IRIS Assessment for PCE (U.S. EPA 2012c). The steps include estimation of dose metrics using relevant PBPK modeling, suitable adjustment to continuous daily exposures from intermittent bioassay exposures, dose-response modeling in the range of observation, interspecies extrapolation, extrapolation to low exposures, and route-to-extrapolation. An overview of these steps is provided in Figure 3-2.

As stated previously, the available evidence likely supports a complex MOA for PCE tumorigenesis, with multiple contributing mechanisms of varying significance. Based on EPA's 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA 2005a), a low-dose linear default approach is supported because the "mode of action information is supportive of linearity or mode of action is not understood." Therefore, EPA derived cancer PODs as an inhalation unit risk (IUR) and oral slope factor (OSF) based on this linear modeling approach.

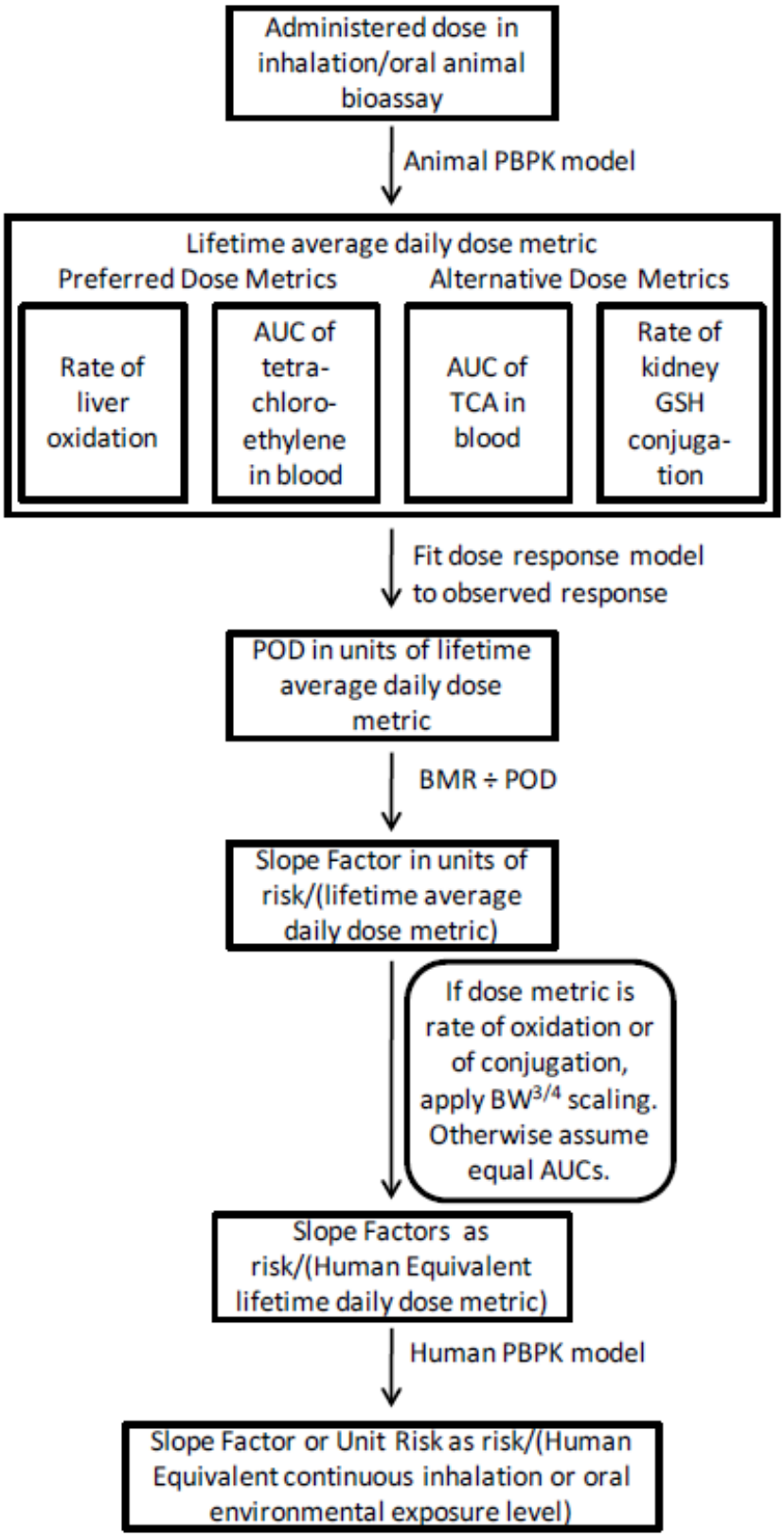
Administered dose in inhalation/oral animal bioassay
Animal PBPK model
Lifetime average daily dose metric
Preferred Dose Metrics
Alternative Dose Metrics
Rate of liver oxidation
AUC of tetra-chloro-ethylene in blood
AUC of TCA in blood
Rate of kidney GSH conjuga-tion
Fit dose response model to observed response
POD in units of lifetime average daily dose metric
BMR ÷ POD
Slope Factor in units of risk/(lifetime average daily dose metric)
If dose metric is rate of oxidation or of conjugation, apply BW3/4 scaling. Otherwise assume equal AUCs.
Slope Factors as risk/(Human Equivalent lifetime daily dose metric)
Human PBPK model
Slope Factor or Unit Risk as risk/(Human Equivalent continuous inhalation or oral environmental exposure level)

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Figure 3-2. Sequence of steps for extrapolating from PCE bioassays in animals to human-equivalent exposures expected to be associated with comparable cancer risk (combined interspecies and route-to-route extrapolation).

Several metabolites of PCE are genotoxic both *in vivo and in vitro* (Section 3.2.3.2.1), and it is thought that the hepatocarcinogenicity of the parent compound is mediated through the action of one or more of its metabolites (Section 3.2.3.2.4). Oxidative metabolism is thought to predominate in the liver, and TCA is the major resultant urinary excretion product. As discussed in Section 3.2.3.2.1, TCA appears to be formed from spontaneous decomposition of trichloroacetyl chloride, which is known to bind to macromolecules. Dichloroacetic acid (DCA) may be formed from dechlorination of TCA, but DCA produced from this pathway is likely to be rapidly metabolized in the liver and not detected in blood or urine. DCA that has been detected in urine is thought to be the result of kidney- specific β-lyase metabolism of the results of GSH conjugation of PCE, and DCA produced from this pathway is presumed to not play a role in liver toxicity or cancer. The potential role of GST conjugates of PCE in liver carcinogenicity, although unknown, is presumed to be less important than the role of oxidative metabolites.

As described in (U.S. EPA 2012c) EPA modeled the JISA bioassay data (JISA 1993) for male and female mice using the dose metrics of total liver oxidative metabolism, PCE AUC, and TCA AUC in blood. Total liver oxidative metabolism is considered the most relevant dose-metric for liver cancer and TCA AUC in liver was an alternative dose metric. Total liver oxidative metabolism was selected as the primary dose metric over TCA AUC because while TCA is the major resultant urinary excretion product of oxidative metabolism, TCA is not formed directly but instead from hydrolysis of trichloroacetyl chloride (Section 3.2.3.2.4). Tumor phenotype data also suggest that TCA may not be the sole tumorigenic metabolite of PCE, although the limited available data precludes any definitive conclusions. PCE AUC in blood was considered the best dose metric for hemangiomas/ hemangiosarcomas in female mice and MCL in both male and female rats. Modeling for both dose metrics generated fits for one-, two-, and three-stage models (details for hepatocellular cancer in Appendix E). All model fits had adequate goodness-of-fit p-values ($p > 0.05$), and overall adequate fit. A summary of the results for hepatocellular adenomas or carcinomas, hemangiomas/hemangiosarcomas, and MCL from JISA (1993) are shown in Table 3-6 based on the preferred dose metric. Extrapolation to humans using total oxidative metabolism led to a BMD_{10} of 2.9, and its lower bound benchmark dose ($BMDL_{10}$) was 1.4-fold lower at 2.1 $mg/kg^{3/4}$-day liver oxidative metabolism. Linear extrapolation from the POD to low internal dose, followed by conversion to human exposures, led to a human equivalent unit risk of 1.8×10^{-3} per ppm. Extrapolation to humans using TCA AUC in liver led to a human equivalent internal dose POD ($BMCL_{10}$) of 69 mg-hr/L-day TCA in blood. Linear extrapolation from the POD to low internal dose, followed by conversion to human exposures, led to a human equivalent unit risk of 1.5×10^{-3} per ppm, slightly lower than the estimate using total liver oxidative metabolism. Dose-response modeling of the male mouse liver tumor data using administered exposure fit the data points similarly to when using total oxidative metabolism or TCA AUC in liver (details in (U.S. EPA 2012c)).

Table 3-6. Human equivalent candidate unit risks, derived using PBPK-derived dose metrics and multistage model; tumor incidence data from JISA (1993) for hepatocellular adenomas or carcinomas

Study Group	Tumor type (multistage model with all dose groups unless otherwise specified)	Human Equivalents				
		POD[a] in internal dose units and dose metric used			Candidate SF /internal dose unit[b]	Candidate IUR /ppm (PBPK range)[c]
Primary dose metrics						
Male mice JISA (1993)	Hepatocellular adenomas or carcinomas	BMD_{10} $BMDL_{10}$	2.9 2.1	Total liver oxidative metabolism, mg/kg0.75-d	49E-3	**1.8E-3** (1.6–1.8)
	Hemangiomas, hemangiosarcomas	BMD_{10} $BMDL_{10}$	63 34	PCE AUC in blood, mg-hr/L-d	2.9E-3	**5.9E-3** (5.9–6.9)
Female mice JISA (1993)	Hepatocellular adenomas or carcinomas	BMD_{10} $BMDL_{10}$	8.4 4.0	Total liver oxidative metabolism, mg/kg0.75-d	25E-3	**0.90E-3** (0.84–0.93)
Male rats JISA (1993)	MCL	BMD_{10} $BMDL_{10}$	46 30	PCE AUC in blood, mg-hr/L-d	3.4	**8.8** (6.8-8.0)
	MCL (Michaelis-Menten)	BMD_{10} $BMDL_{10}$	20 5.0	PCE AUC in blood, mg-hr/L-d	20	**40** (40-47)
Female rats JISA (1993)	MCL	BMD_{10} $BMDL_{10}$	136 61	PCE AUC in blood, mg-hr/L-d	1.6	**3.3** (3.3-3.9)
	MCL (control and low dose groups only)	BMD_{10} $BMDL_{10}$	11 5.2	PCE AUC in blood, mg-hr/L-d	19	**39** (39-45)
Female and male rats combined JISA (1993)	MCL (Michaelis-Menten)	BMD_{10} $BMDL_{10}$	17 3.0	PCE AUC in blood, mg-hr/L-d	33	**68** (67-71)

Note: From Table 5-18 in the U.S. EPA (2012e) IRIS assessment of PCE; SF = Slope Factor; IUR = Inhalation Unit Risk; MCL= Mononuclear cell leukemias.

[a] PODs were estimated at the indicated BMRs in terms of extra risk; i.e., BMDL10 = lower bound for the level of the internal dose metric associated with 10% extra risk. Dose metric units are in the first column and include cross-species scaling to a human equivalent internal dose metric. Refer to Appendix D for dose-response modeling details.

[b] Slope Factor = BMR/BMDLBMR in units of risk per dose metric unit (as given in the first column).

[c] Inhalation unit risk (IUR) is given by the product of the slope factor in units of risk per dose metric unit and an inhalation dose metric conversion factor (DMCFppm): IUR = BMR/BMDLBMR × DMCFppm, where the DMCFppm is derived from the PBPK model.

Human inhalation cancer risk was assessed using several different sex-specific animal tumor data sets and the PBPK model in U.S. EPA (2012e). These results, and their uncertainties are discussed in detail there.

The majority of the National research Council (NRC) peer review panel for the IRIS assessment (U.S. EPA 2012e) recommended that the male mouse hepatocellular tumors be used for cancer risk estimation. Therefore, the primary inhalation unit risk is 2×10^{-3} per ppm or 3×10^{-7} per µg/m^3 (rounding to one significant digit), based on the male mouse hepatocellular tumor data from the JISA (1993) bioassay. Some members of the NRC peer review panel recommended that the MCL data be

used for cancer risk estimation. The inhalation unit risk would be 7×10^{-2} per ppm, or 1×10^{-5} per µg/m3 (rounding to one significant digit) if it were based on the combined male and female rat MCL data, which provided increased statistical power and improved model fit compared to either sex alone.

3.2.5.4 Points of Departure for Human Health Hazard Endpoints and Confidence Levels

Confidence Levels

For the acute endpoint, the value used in this risk evaluation is from Altmann et al. (1990), a medium quality short-term study demonstrating neurotoxicity based on impaired visual function associated with delayed neurological signaling. This endpoint is robustly supported by multiple human and animal studies. The data from Altmann et al. (1990) is based on 4 days of 4 hr/day exposure, so applying the dose-response analysis to a single day of exposure involves some uncertainty, however it is unlikely that outcomes would substantially differ between a single day and 4 days of exposure. Overall, there is medium-high confidence in this endpoint.

For chronic non-cancer endpoints, multiple endpoints are available representing the health domains of neurotoxicity, kidney toxicity, liver toxicity, immune toxicity, and reproductive/developmental toxicity. These endpoints are supported by data in both humans and animals and the range of PODs is within ~10-fold for most endpoints, although the full set of endpoints range by as much as 150-fold. Overall, there is medium-high confidence in the chronic endpoints.

For cancer, there is evidence of carcinogenicity in multiple tissues. The IUR (Inhalation Unit Risk) was developed from a High-quality animal study, however the limited available human data was ambiguous. Overall, there is medium confidence in the cancer endpoint.

Table 3-7. Summary of PODs for Evaluating Human Health Non-Cancer Hazards from Acute Exposure Scenarios

Target Organ System	Species - route	Human Equivalent Concentration (HEC)	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
CNS	Humans - Inhalation	4 hrs/day = 10 ppm (68 mg/m^3) 8 hrs/day = 5 ppm (34 mg/m^3) 12 hrs/day = 3.3 ppm (22 mg/m^3) 24 hrs/day = 1.7 ppm (11 mg/m^3)	Neurotoxicity increased latencies for pattern reversal visual-evoked potentials	UF_A=1; UF_H=10; UF_L=1 **Total UF=10**	Altmann et al. (1990)	Medium

Best Representative Chronic Studies For Each Health Domain

From among all chronic studies, EPA selected the most robust studies or PODs from within each health domain to serve as representative endpoints for risk estimation. These studies are highlighted in blue in Table 3-8 below. There is High confidence in these robust PODs. Justification for the selections for each health domain are provided below:

CNS (Neurotoxicity)

PODs were derived from two studies (Echeverria et al. 1995; Cavalleri et al. 1994) that both observed CNS effects presenting as visual deficits. Both studies scored a Medium in data quality and both studies are based on human data with equivalent cumulative UFs. Therefore, the midpoint of the range as

derived in (U.S. EPA 2012c) is the best representative POD for this endpoint and the neurotoxicity domain overall. EPA additionally derived occupational HECs for this POD, as described in Section 3.2.5.3.2. These HECs are provided in a separate row highlighted in green.

Kidney Effects

While there was a Medium-quality human study that reported urinary markers of nephrotoxicity (Mutti et al. 1992), this POD was derived from a LOAEL, which resulted in a cumulative UF of 100. The rodent study by JISA (1993) score a High in data quality and only had a combined UF of 30, indicating reduced uncertainty surrounding the POD. Therefore this study was used to represent the kidney domain. There was no discernible difference among the mice and rat data from that study, so the POD derived from mice was used in order to represent the most sensitive and robust endpoint.

Liver Effects

Three studies provided sufficient dose-response information for liver effects in mice (JISA 1993; NTP 1986b; Buben and O'Flaherty 1985). Only the data from (JISA 1993) did not require a LOAEL-to-NOAEL UF, and that study was additionally of High quality. Additionally, increased liver/body weight ratio is not considered adverse on its own and may be due to induction of PPARα, which is less active in humans. Therefore, the POD from (JISA 1993) for increased angiectasis was selected to represent the liver domain.

Reproductive/Developmental

Reproductive

There is only a single adequate study examining reproductive effects (Beliles et al. 1980), which observed reduced sperm quality in males following only 5 days exposure. This study scored High in data quality and was therefore used to represent reproductive effects. Of note, despite this study only examining 5 days of exposure, this exposure duration covers the window of sperm production while the observation period up to 10 weeks covered the full period of spermatogenesis. Since PCE is not bioaccumulative, continuous exposure is not expected to result in a more sensitive toxicological response.

Developmental

Three studies demonstrated adequate dose-response information for developmental endpoints, each reporting varying but overlapping effects. Nelson et al. (1979) observed decreased weight gain in offspring along with indications of developmental neurotoxicity. Tinston et al. (1994) reported neonatal mortality as well as CNS effects in a multigenerational study. Carney et al. (2006) observed decreased placental and fetal weight along with skeletal effects. Nelson et al. (1979) scored a low in data quality while the other two studies scored a high. Among the two high-quality studies, the POD from (Tinston 1994) was selected to represent the domain because the data comes from a 2-generation study which would be expected to capture all potential developmental outcomes, as opposed to the short-duration study used in (Carney et al. 2006).

8069 **Table 3-8. Summary of PODs for Evaluating Human Health Non-Cancer Hazards from Chronic**
8070 **Exposure Scenarios**

Target Organ System	Species - route	Human Equivalent Concentration (HEC)	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality Score
CNS	Humans - Inhalation	2.2 ppm (15 mg/m^3)	Neurotoxicity - Color confusion	UF_A=1; UF_H=10; UF_L=10 UF_S = 1 **Total UF=100**	Cavalleri et al. (1994)	Medium
	Humans - Inhalation (inferred)	8.3 ppm (56 mg/m^3)	Visual reproduction, pattern memory, pattern recognition and reaction time in pattern memory	UF_A=1; UF_H=10; UF_L=10 UF_S = 1 **Total UF=100**	Echeverria et al. (1995)	Medium
	Humans - Inhalation	5.2 ppm (36 mg/m^3)	Midpoint of the range of the two neurotoxicity studies	UF_A=1; UF_H=10; UF_L=10 UF_S = 1 **Total UF=100**	Based on U.S. EPA (2012c)	Medium
	Humans - Inhalation	14.5 ppm [8 hr] (99 mg/m^3) 9.7 ppm [12 hr] (66 mg/m^3)	Midpoint of the range of the two neurotoxicity studies (adjusted for 8 and 12 hr occupational TWAs)	UF_A=1; UF_H=10; UF_L=10 UF_S = 1 **Total UF=100**	Based on U.S. EPA (2012c)	Medium
Kidney	Humans - Inhalation (inferred)	5.0 ppm (34 mg/m^3)	Urinary markers of nephrotoxicity	UF_A=1; UF_H=10; UF_L=10 UF_S = 1 **Total UF=100**	Mutti et al. (1992)	Medium
	Rats - Inhalation	9.0 ppm (61 mg/m^3)	Nuclear enlargement in proximal tubules	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	JISA (1993)	High
	Mice - Inhalation	2.1 ppm (14 mg/m^3)	Nuclear enlargement in proximal tubules	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	JISA (1993)	High
Liver	Mice - Inhalation	31 ppm (210 mg/m^3)	Increased angiectasis in liver	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	JISA (1993)	High
	Mice - Inhalation	310 ppm (2100 mg/m^3)	Increased liver degeneration/necrosis	UF_A=3; UF_H=10; UF_L=10 UF_S = 1 **Total UF=300**	NTP (1986b)	High

Target Organ System	Species - route	Human Equivalent Concentration (HEC)	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality Score
	Mice - Oral (gavage)	40 ppm (270 mg/m^3)	Increases liver/body-weight ratio	UF_A=3; UF_H=10; UF_L=10 UF_S = 10 **Total UF=3000**	Buben (1985)	Medium
Reproductive/ Developmental	Reproductive					
	Mice - Inhalation	21 ppm (140 mg/m^3)	Reduced sperm quality following 5 days exposure	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	Beliles et al. (1980)	High
	Developmental					
	Rats	29 ppm (200 mg/m^3)	Decreased weight gain; altered behavior, brain acetylcholine	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	Nelson et al. (1979)	Low
	Rats - Inhalation	18 ppm (122 mg/m^3)	Increased F_{2A} pup deaths by Day 29, CNS depression in F_1 and F_2	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	Tinston et al. (1994)	High
	Rats - Inhalation	16 ppm (110 mg/m^3)	Decreased fetal and placental weight, skeletal effects	UF_A=3; UF_H=10; UF_L=1 UF_S = 1 **Total UF=30**	Carney et al. (2006)	High

Notes: Rows shaded in blue indicate PODs selected as most robust and representative for the associated health domain. Row shaded in green indicates occupational HECs for the chronic neurotoxicity domain.

As explained in Section 3.2.5.3.3, the primary IUR is derived from male mouse hepatocellular tumor data, while the alternative IUR is from combined male and female rat MCL data. Both values are shown in Table 3-9.

Table 3-9. Summary of PODs for Evaluating Cancer Hazards from Chronic Inhalation Scenarios

Exposure Duration for Risk Analysis	Hazard Value	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality Score
CHRONIC EXPOSURE	IUR 2×10^{-3} per ppm (3×10^{-4} per mg/m^3)	male mouse hepatocellular tumors	Not applicable	JISA (1993)	High
	Alternate IUR: 7×10^{-2} per ppm (1×10^{-2} per mg/m^3)	Male and female rat mononuclear cell leukemia (MCL)	Not applicable	JISA (1993)	High

Notes:
The inhalation unit risk should not be used with exposures exceeding 60 ppm, or 400 mg/m^3 (the equivalent ambient exposures corresponding to the POD for male mouse hepatocellular tumors), because above this exposure level, the dose-response relationship is not linear, and the unit risk would tend to overestimate risk.
Cancer risks following acute exposures were not estimated. The relationship between a single short-term exposure to PCE and the induction of cancer in humans is not known.

3.2.5.4.1 Route to Route Extrapolation for Dermal PODs

Workers and consumers can be exposed to PCE under various exposure scenarios via dermal routes. EPA did not identify toxicity studies by the dermal route that were adequate for dose-response assessment. Dermal candidate values derived by two methods were compared and the results are shown in Table 3-10. Dermal candidate values were calculated based on route-to-route extrapolation from two different routes either inhalation or oral PODs. For all endpoints previously derived from animal or human studies in the EPA IRIS Assessment (U.S. EPA 2012c), both oral and inhalation PODs (as HECs or HEDs) were derived from the original study data using the best available approaches for incorporating PCE specific toxicokinetic data (i.e. the PBPK model) when possible. Extrapolation to oral HEDs was not available for all endpoints.

Extrapolating from inhalation PODs to the dermal route account for human inhalation and body weight and assume average exposure factors from the Exposure Factors Handbook (U.S. EPA 2011a) shown in the equations below. Extrapolating from oral PODs to the dermal route considered differences in oral and dermal absorption. EPA assumed 100% oral and inhalation absorption, supported by studies in animals (ATSDR 2019; U.S. EPA 2012c). EPA accounted for dermal absorption in the dermal exposure estimate (see Section 2.4.1.29). Therefore, the oral HEDs were used directly for dermal exposures.

Inhalation to dermal extrapolation for non-cancer effects:
dermal POD = inhalation POD [mg/m^3] × inhaled volume (m^3) ÷ body weight (kg)

Inhalation to dermal extrapolation for cancer effects:
dermal slope factor = IUR [per mg/m^3] ÷ inhaled volume (m^3) × body weight (kg) ,

where the inhaled volume was the ventilation rate 1.25 m^3/hr (for light activity) times the appropriate exposure duration (4 hours from Altmann et al. (1990)) for acute endpoints, or 20 m^3 per day for 24 hrs duration and the chronic endpoints and a body weight of 80 kg. These exposure factors are based on EPA RfC Guidance (U.S. EPA 1994c) for inhalation rates and the 2011 Exposure Factors Handbook (U.S. EPA 2011a) for body weight. EPA assumes that activities involving PCE exposure involve some movement, and thus, assumed a ventilation rate for light activity.

PODs were derived from Altmann et al. (1990) for a range of inhalation exposure durations, the route to route extrapolation for dermal used the duration of the experimental study (4 hrs) and the air concentration in the study (a NOAEC of 10 ppm or 68 mg/m^3) for extrapolation to the dermal route.

There is uncertainty regarding the likelihood that dermal exposure will result in cancer, but because humans may experience different cancers than rodents, EPA has assumed that the slope factor can be considered generally representative of the potential for cancers of other types and that this is relevant to model via the dermal route. When both an HEC and HED value was available for a given endpoint, EPA derived dermal PODs via extrapolation from both values. For all endpoints the difference in the derived dermal POD between routes is no more than approximately 2-fold. In considering the relative

uncertainties involved in extrapolation via either route, the most robust and sensitive POD was selected for use in risk estimation. The dermal POD value to be used for risk estimates is bold in the table below, and the selected representative studies are highlighted in blue, as was done for HEC values.

Differences in absorption across routes are accounted for in the occupational (Section 2.4.1.29) and consumer (Section 2.4.2.2.2) dermal exposure assessments, respectively. While EPA assumes 100% absorption via oral and inhalation routes (Section 3.2.2.1.1), the volatility of PCE significantly decreases the expected dermal absorption under non-occluded conditions. The occupational exposure estimates incorporated modeled absorption under non-occluded conditions through the *Dermal Exposure to Volatile Liquids Model* while consumer dermal exposure utilizes the permeability module from the Consumer Exposure Model (CEM) was used to estimate dermal exposure only for COUs under which impeded evaporation is expected.

Table 3-10. Derivation of Dermal PODs by Route-to-Route Extrapolation

Target Organ System and Effect	Inhalation POD and Duration	Inhalation to Dermal Adjustments	Inhalation to Dermal HED (mg/kg-day)	Oral to Dermal[a] HED (mg/kg-day)	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
Acute Exposures							
CNS Neurotoxicity increased latencies for pattern reversal visual-evoked potentials	10 ppm (68 mg/m^3) 4 hrs/day	1.25 m^3/hr 4 hrs/day 80 kg BW	**4.25**[b]	N/A[c]	UF_A=1; UF_H=10; UF_L=1 **Total UF=10**	Altmann et al. (1990)	Medium
Chronic Exposures							
CNS Neurotoxicity Color confusion	2.2 ppm (15 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	3.75	**2.6**	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Cavalleri et al. (1994)	Medium
CNS Neurotoxicity Visual reproduction, pattern memory, pattern recognition and reaction time in pattern memory	8.3 ppm (56 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	14	**9.7**	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Echeverria et al. (1995)	Medium
Midpoint of the range of the two neuorotoxicity endpoints	5.2 ppm (36 mg/m^3)	20 m^3/day 80 kg BW	9.0	**6.2**	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Based on U.S. EPA (2012c)	Medium

Target Organ System and Effect	Inhalation POD and Duration	Inhalation to Dermal Adjustments	Inhalation to Dermal HED (mg/kg-day)	Oral to Dermal[a] HED (mg/kg-day)	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
Kidney Urinary Markers of nephrotoxicity	5.0 ppm (34 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	8.5	**5.4**	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Mutti et al. (1992)	Medium
Kidney Nuclear enlargement in proximal tubules	9.0 ppm (61 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	15	**9.5**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (, 1993, 630653)	High
Kidney Nuclear enlargement in proximal tubules	2.1 ppm (14 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	3.5	**2.2**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (, 1993, 630653)	High
Liver Increased angiectasis in liver	31 ppm (210 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	52.5	**24.5**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (1993)	High
Liver Increased liver degeneration/ necrosis	310 ppm (2100 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	525	**252**	UF_A=3; UF_H=10; UF_L=10 **Total UF=300**	NTP (1986b)	High
Liver Increases liver/body-weight ratio	40 ppm (270 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	67.5	**32**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Buben (1985)	Medium
Developmental Decreased weight gain; altered behavior, brain acetylcholine	29 ppm (200 mg/m^3)	20 m^3/day 80 kg BW	**50**	N/A	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Nelson et al. (1979)	Low
Developmental Reduced sperm quality following 5 days exposure	21 ppm (140 mg/m^3)	20 m^3/day 80 kg BW	35	**22**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Beliles et al. (1980)	High
Developmental Increased F_{2A} pup deaths by Day 29, CNS depression in F_1 and F_2	18 ppm (122 mg/m^3)	20 m^3/day 80 kg BW	**31**	N/A	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Tinston et al. (1994)	High

Target Organ System and Effect	Inhalation POD and Duration	Inhalation to Dermal Adjustments	Inhalation to Dermal HED (mg/kg-day)	Oral to Dermal[a] HED (mg/kg-day)	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
Developmental Decreased fetal and placental weight, skeletal effects	16 ppm (110 mg/m^3)	20 m^3/day 80 kg BW	**28**	N/A	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Carney et al. (2006)	High
Cancer							
male mouse hepatocellular tumors	3 × 10^{-4} per mg/m^3	20 m^3/day 80 kg BW	1 × 10^{-3} per mg/kg/day	**2 × 10^{-3} per mg/kg/day**	Not applicable	JISA (1993)	High
Male and female rat MCL	1 × 10^{-2} per mg/m^3	20 m^3/day 80 kg BW	4 × 10^{-2} per mg/kg/day	6 × 10^{-2} per mg/kg/day	Not applicable	JISA (1993)	High

Notes:

[a] The oral to dermal slope factors should not be used with exposures exceeding 50 mg/kg/day (the equivalent ambient exposures corresponding to the POD for male mouse hepatocellular tumors), because above this exposure level, the dose-response relationship is not linear, and the unit risk would tend to overestimate risk.

[b] The PODs highlighted in bold are used in calculating risks

[c] N/A an acute oral to dermal POD was not calculated since an acute oral POD was not identified and the inhalation to dermal POD was used for assessing risk from dermal exposures

Note: Cancer risks following acute exposures were not estimated. The relationship between a single short-term exposure to PCE and the induction of cancer in humans is not known.

3.2.6 Key Assumptions and Uncertainties for Human Health Hazard

3.2.6.1 Hazard ID and Weight of Scientific Evidence

There is medium-high confidence in the database and WOE determinations for human health hazard. All but one of the studies considered for dose-response analysis scored either Medium or High in data quality evaluation and were determined to be highly relevant to the pertinent health outcome. EPA selected the best representative chronic study for each identified endpoint to use for risk estimation, taking into account factors such as data quality evaluation score, species, cumulative uncertainty factor, and relevance. The only study considered for dose-response analysis that scored a Low in data evaluation was (Nelson et al. 1979), however the health outcomes observed in this study were covered by the other two high-quality developmental toxicity studies, (Tinston 1994) and (Carney et al. 2006).

For most health domains, the weight of scientific evidence was very clear, with consistent results observed across multiple species and representing multiple endpoints within the health domain. The data was a bit more ambiguous for immune and hematological effects however. While there was some indication of specific endpoints related to immunotoxicity or blood effects, EPA determined that the database was not fully consistent and there was an absence of adequate quantitative information available to conclude that the domains supported dose-response analysis (Section 0). There is uncertainty whether the PODs for other endpoints carried forward are sufficiently protective of any potential immune or hematological effects that were not accounted for in this risk evaluation. Additionally, there is some uncertainty as to the weight of the evidence for liver effects relating to human relevance. Consistent effects were only observed in rodents and the potential influence of certain MOA that are more highly active in rodents (i.e. PPARα, Section 3.2.3.2.4) suggests that observed liver

toxicity may have reduced significance to the majority of human populations. However, susceptible subpopulations such as those with liver disease (Section 3.2.5.2) may still be of high risk of liver toxicity from sustained PCE exposure.

3.2.6.2 Derivation of PODs, UFs, and PBPK Results

Conceptually, the POD should represent the maximum exposure level at which there is no appreciable risk for an adverse effect in the study population under study conditions (i.e., the threshold in the dose-response relationship). In fact, it is not possible to know that exact exposure level even for a laboratory study because of experimental limitations (e.g. the ability to detect an effect, the doses used and dose spacing, measurement errors, etc.), and POD approximations like the doses used (i.e., a NOAEL) an exposure level which is modeled from the reasonably available doses used (i.e., BMDL) are used. The application of UFs is intended to account for this uncertainty/variability to allow for estimating risk for sensitive human subgroups exposed continuously for a lifetime. While the selection of UFs is informed by reasonably available data, the true necessary extent of adjustment most appropriate for capturing all relevant uncertainty and variability is unknown.

For this draft risk evaluation, non-cancer PODs were all based on NOAELs and LOAELs because the data for the selected endpoints was unable to be BMD modeled. This results in reduced precision in POD estimates because the POD is dependent on the dose selection of the study as opposed to the response rate/level for the effect of interest.

For each of these types of PODs, there are additional uncertainties pertaining to adjustments to the administered exposures (doses). Typically, administered exposures (doses) are converted to equivalent continuous exposures (daily doses) over the study exposure period under the assumption that the effects are related to concentration × time, independent of the daily (or weekly) exposure regimen (i.e., a daily exposure of 6 hours to 4 ppm is considered equivalent to 24 hours of exposure to 1 ppm). However, the validity of this assumption is generally unknown, and, if there are dose-rate effects, the assumption of $C \times t$ equivalence would tend to bias the POD downwards.

For the PBPK analyses in this assessment (Section 3.2.2.2), the actual administered exposures are taken into account in the PBPK modeling, and equivalent daily values (averaged over the study exposure period) for the dose-metrics are obtained. EPA determined that the peer-reviewed PBPK model sufficiently accounted for any variability and uncertainties in route-to-route extrapolation, and therefore inhalation and oral data were considered equivalently relevant. Nonetheless, this PBPK model, like any model, does not incorporate all possible sources of biological uncertainty or variability.

Use of the PBPK model resulted in data derived HEC and HED values replacing default assumptions and uncertainty factors that would have otherwise been used such as allometric scaling and a UF_{TK} of 3 in accounting for interspecies toxicokinetic variability. Data-derived values are always preferred to default uncertainty adjustments and improve confidence in the adjusted PODs. There is additional uncertainty for dermal PODs which required route-to-route extrapolation based on assumed exposure factors without the availability of a dermal compartment in the PBPK model.

3.2.6.3 Cancer Dose-Response

There is uncertainty concerning the selected POD for cancer dose-response. EPA derived an IUR and dermal SF based on the low dose linear assumption. The MOA (Section 3.2.3.2.4) concludes that genotoxicity is likely to be at least a partial contributor to the MOA and any non-mutagenic mechanisms for carcinogenesis that would be associated with a threshold are likely only relevant at higher doses

above those associated with tumorigenesis. Nonetheless, the linear assumption always has some inherent uncertainty.

Additionally, EPA selected the male mouse data for hepatocellular adenoma/carcinoma to use as the representative cancer POD based on the majority recommendation from the NRC peer review panel of the IRIS Assessment (U.S. EPA 2012e) (Section 3.2.5.3.3). This is further supported based on a stronger weight of evidence for liver effects compared to immune outcomes. However, the NRC panel was not unanimous and some members believed that the MCL data was better representative. The MCL IUR for the combined male and female dataset is 35x higher than the hepatocellular cancer IUR selected for use as the representative cancer POD. An adjustment was not made to account for the additional risk from MCL or hemangiomas and therefore the selected cancer POD may underestimate total cancer risk from PCE.

3.2.6.4 Confidence Ratings for Endpoints and Selected Representative PODs

There is medium-high confidence in the acute non-cancer endpoint and POD based on neurotoxicity, medium-high confidence in the chronic non-cancer endpoints and PODs, and medium confidence in the cancer endpoint. There is high confidence in the robust chronic non-cancer PODs selected to represent each health domain for risk estimation. Confidence ratings are a half-step lower (e.g. medium instead of medium-high) for all dermal PODs because derivation required extrapolation across routes without the availability of a PBPK model dermal compartment. See Section 3.2.5.4 for more details on the confidence descriptions for each category.

4 RISK CHARACTERIZATION

4.1 Environmental Risk

EPA took fate, exposure, and environmental hazard into consideration to characterize environmental risk of PCE. As stated in Section 2.1, PCE has low potential to bioconcentrate in biota and moderate potential to accumulate in wastewater biosolids, soil, or sediment. Releases of PCE 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 PCE is low. EPA modeled environmental exposure with surface water concentrations of PCE ranging from 9.7E-09 ppb to 2,034 ppb from facilities releasing the chemical to surface water. Measured surface water concentrations in ambient water range from below the detection limit to 1.7 ppb. The modeled data represents estimated concentrations near facilities that are actively releasing PCE to surface water, while the reported measured concentrations represent sampled ambient water concentrations of PCE. Differences in magnitude between modeled and measured concentrations may be due to measured concentrations not being geographically or temporally close to known releasers of PCE.

As stated in Section Summary of Environmental Hazard 3.1.5, EPA concludes that PCE poses a hazard to environmental aquatic receptors to include: aquatic invertebrates, fish, and aquatic plants. The most sensitive species for acute toxicity were two daphnid species, *Ceriodaphnia dubia* and *Daphnia magna*. The acute toxicity value was as low as 2.5 mg/L based on immobilization of daphnia. PCE presents an acute hazard to fish based on mortality of rainbow trout as the most sensitive species with acute toxicity values as low as 4.8 mg/L for mortality LC_{50}. For chronic exposures, PCE is a hazard to aquatic invertebrates, with a chronic toxicity value of 0.5 mg/L; and a chronic toxicity value of 0.8 mg/L for fish. PCE is also a hazard for green microalgae with toxicity values as low as 2.0E-02 mg/L.

EPA assigned an overall quality level of high, medium or low to 30 acceptable studies. These studies contained relevant aquatic toxicity data for fish, aquatic invertebrates, and aquatic plants. As shown in Table 3-1, EPA identified 10 aquatic toxicity studies as the most relevant for quantitative assessment. Four of the 10 studies were carried forward for characterizing the potential environmental risks from PCE. The rationale for selecting these studies is provided in Section 3.1.3 Weight of Scientific Evidence.

A total of 10 acceptable aquatic environmental hazard studies were identified for PCE. EPA assigned nine high, and one medium for overall quality levels during data evaluation (See Table 3-1 in Section 3.1.2 and the *Draft Risk Evaluation for Perchloroethylene: Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies* (U.S. EPA 2020i). The *Draft Risk Evaluation for Perchloroethylene: Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies* (U.S. EPA 2020i) presents details of the data evaluations for each study, including scores for each metric and the overall study score.

Given PCE's conditions of use under TSCA outlined in problem formulation (U.S. EPA 2018d), EPA determined that environmental exposures are expected for aquatic species, and risk estimation is discussed in Section 4.1.2.

4.1.1 Risk Estimation Approach

To assess environmental risk, EPA evaluates environmental hazard and exposure data. EPA used modeled exposure data from E-FAST (U.S. EPA 2014b), as well as monitored data from the WQP (Nwqmc 2017), to characterize the exposure of PCE to aquatic species. Environmental risks are estimated by calculating a risk quotients (RQ). As stated previously, modeled data were used to represent surface water concentrations near facilities actively releasing PCE to surface water. The modeled concentrations were used to represent ambient water concentrations of PCE. 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:

RQ = Predicted Environmental Concentration / 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 invertebrates and algae shown in Table 3-2, and the environmental concentrations described in Table 4-1, were used to calculate RQs (U.S. 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 above the hazard benchmark in an aquatic environment.

Frequency and duration of exposure also affect the potential for adverse effects in aquatic organisms. Therefore, the number of days that a COC was exceeded was also calculated using E-FAST (U.S. EPA 2014b), as described in Section 2.3.1.2. The days of exceedance modeled in E-FAST are not necessarily consecutive and could occur sporadically throughout the year. 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).

Calculation of Days of COC Exceedance

The Probabilistic Dilution Model (PDM) portion of E-FAST 2014 (U.S. EPA 2014b) was also run for free-flowing water bodies, which predicts the number of days per year a chemical's concentration of concern (COC) in an ambient water body will be exceeded. 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 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 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 per year (see required inputs below).

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 4-1and Figure 4-2 (east and west U.S.) for the maximum days of release scenario, and in Figure 4-3and Figure 4-4 (east and west U.S.) for the 20-days of release scenario. Overall, there are 33 U.S. states/territories with either a measured concentration or a predicted concentration; at the watershed level, there are 109 HUC-8 areas and 149 HUC-12 areas with either measured or predicted concentrations. 5.3.68Appendix D provides a list of states/territories with facility releases (as mapped) and/or monitoring sites.

EPA also used surface water monitoring data from the Water Quality Portal (Nwqmc 2017) and from the published literature to characterize the risk of PCE to aquatic organisms. These monitored surface water concentrations reflect concentrations of PCE in ambient water. EPA's Storage and Retrieval (STORET) data and USGS's National Water Information System (NWIS) data were extracted on Oct 3rd, 2018 from the WQP. These data show an average concentration for PCE of 0.2 ± 0.6 µg/L or ppb in surface water from 1,597 measurements taken throughout the U.S. between 2013 and 2017. The highest value recorded during these years was 1.7 µg/L or ppb, which was measured in 2014. Table 4-1 shows that algae RQ were greater 1 at the maximum observed concentration. All other RQs were close to zero.

Table 4-1. RQs Calculated using Monitored Environmental Concentrations from Water Quality Portal

Monitored Surface Water Concentrations (ppb) from 2013-2017	RQ using Acute COC of 1,342 ppb	RQ using Chronic COC of 50 ppb	RQ using algae COC of 1.4 ppb
Mean (SD): 0.23 (0.55) ppb	0.0	0.0	0.2
Maximum: 1.69 ppb	0.0	0.0	1.2

Surface Water Concentrations

The surface water concentrations associated with the monitoring stations and facility releases are denoted on the maps using COCs (Section 3.1.4) to determine the concentration thresholds:

Red ≥1,342 µg/L (exceeds all COC for algae, aquatic invertebrate, and fish

orange 50-1,341 µg/L (exceeds the COC for algae and aquatic invertebrate, but not for fish)

green 1.4 to 49 µg/L (exceeds the COC for algae, but not for aquatic invertebrate or fish)

blue Detected, but less than 1.4 µg/L (less than all COC)

purple Not Detected (applies only to measured concentrations; detection limits vary)

For the predicted concentrations, the concentrations represent conditions under low flow conditions (i.e., 7Q10 flows). The harmonic mean concentrations were not mapped but are presented in the detailed summary tables.

Symbols and Layering

Due to the scale of the maps found in Section 4, some symbols may overlap each other if the monitoring stations and facilities are near each other or there are multiple releases modeled for the same facility (i.e., one facility is both a direct discharger and a receiving facility). As such, the maps are layered to make sure that the most important information is always be visible. The following rules were applied:

- Monitoring stations (small circles) are always on top of indirect discharge releases (medium triangles), which are always on top of direct discharge releases (large squares), and

- Within each symbol type (monitoring station, direct release, and indirect release), a higher concentration level is always on top of a lower concentration level (i.e., from top to bottom: ≥1,342 µg/L (red), 50-1,341 µg/L (orange), 1.4-49 µg/L (green), <1.4µg/L (blue), and not detected (purple).

Figure 4-1 Concentrations of PCE from PCE-Releasing Facilities (Maximum Days of Release Scenario) and WQX Monitoring Stations: Year 2016, East US. All indirect releases are mapped at the receiving facility unless the receiving.

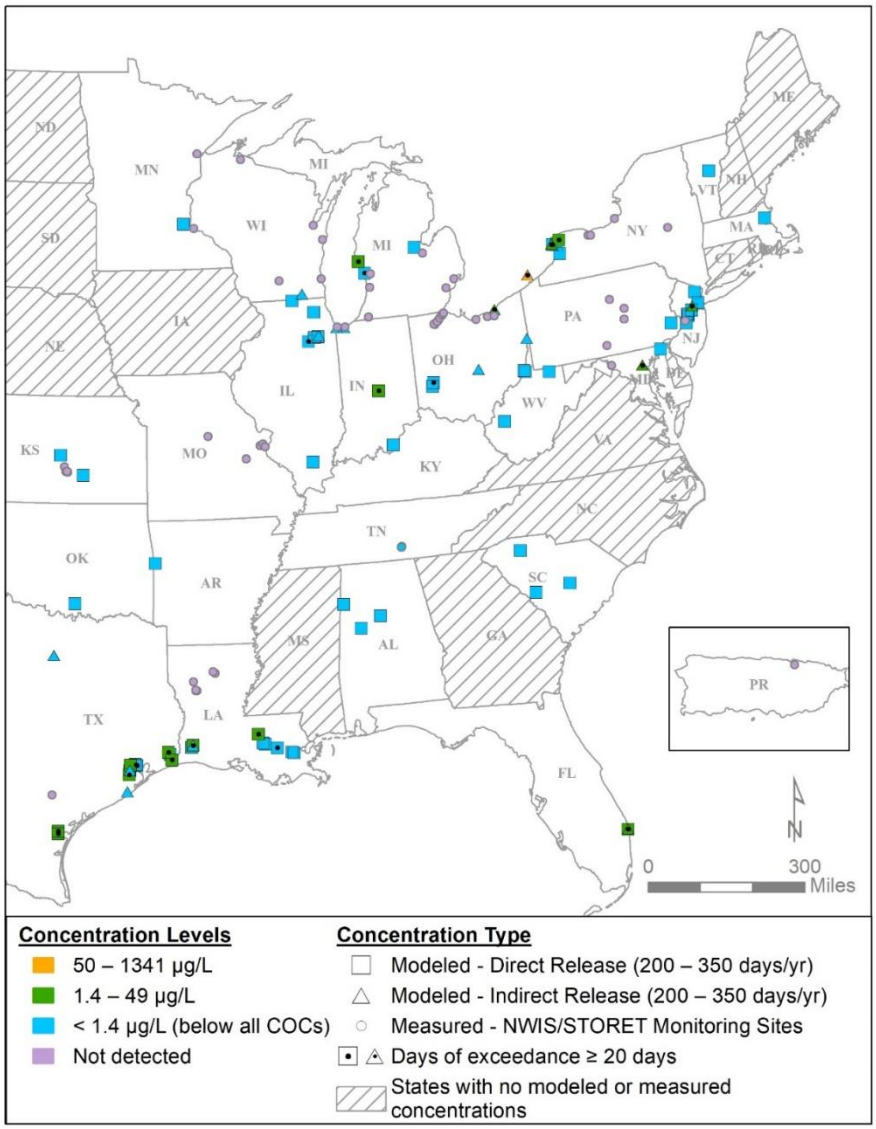
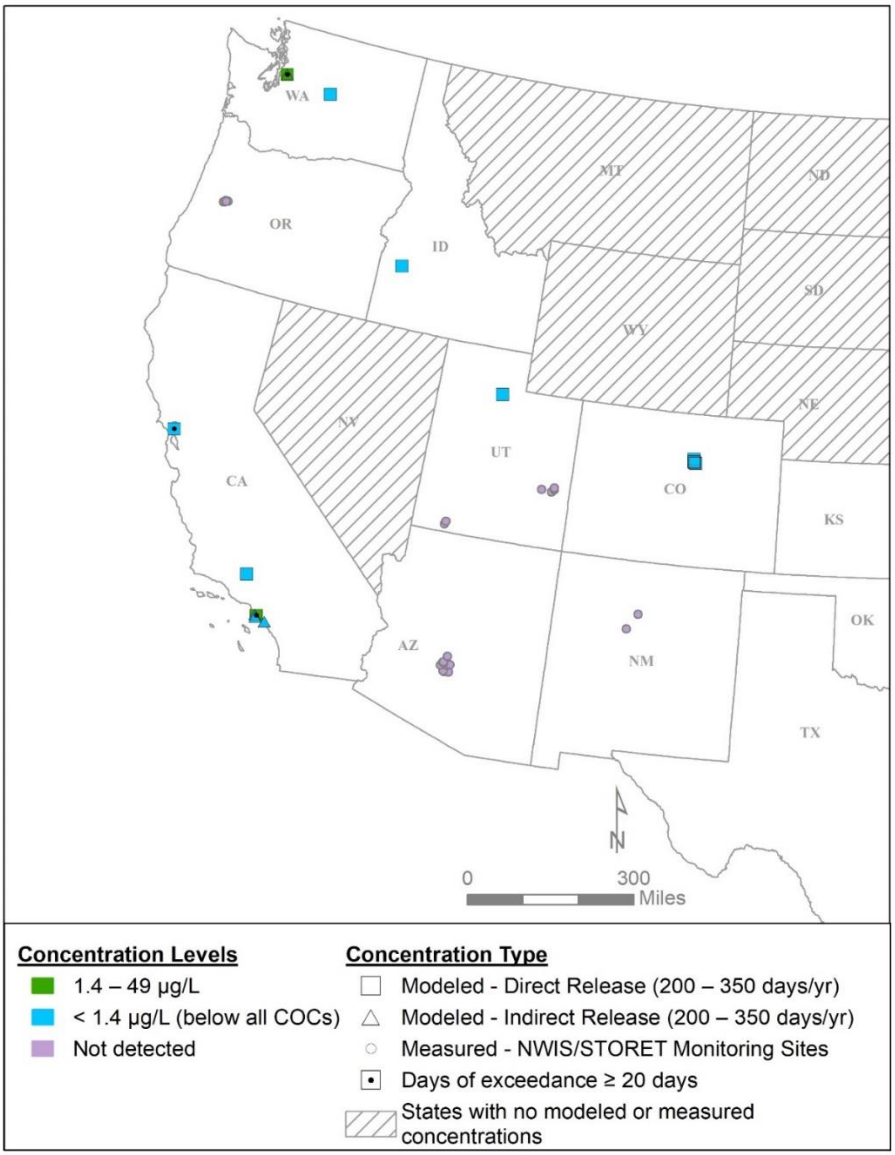


Figure 4-2 Concentrations of PCE from PCE-Releasing Facilities (Maximum Days of Release Scenario) and WQX Monitoring Stations: Year 2016, West US. All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.


WA
OR
CA
ID
NV
MT
ND
SD
WY
NE
UT
CO
KS
AZ
NM
OK
TX
N
0
300
Miles
Concentration Levels
1.4 – 49 µg/L
< 1.4 µg/L (below all COCs)
Not detected
Concentration Type
Modeled - Direct Release (200 – 350 days/yr)
Modeled - Indirect Release (200 – 350 days/yr)
Measured - NWIS/STORET Monitoring Sites
Days of exceedance ≥ 20 days
States with no modeled or measured concentrations

8381
8382

Figure 4-3. Concentrations of PCE from PCE-Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, East US. All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.

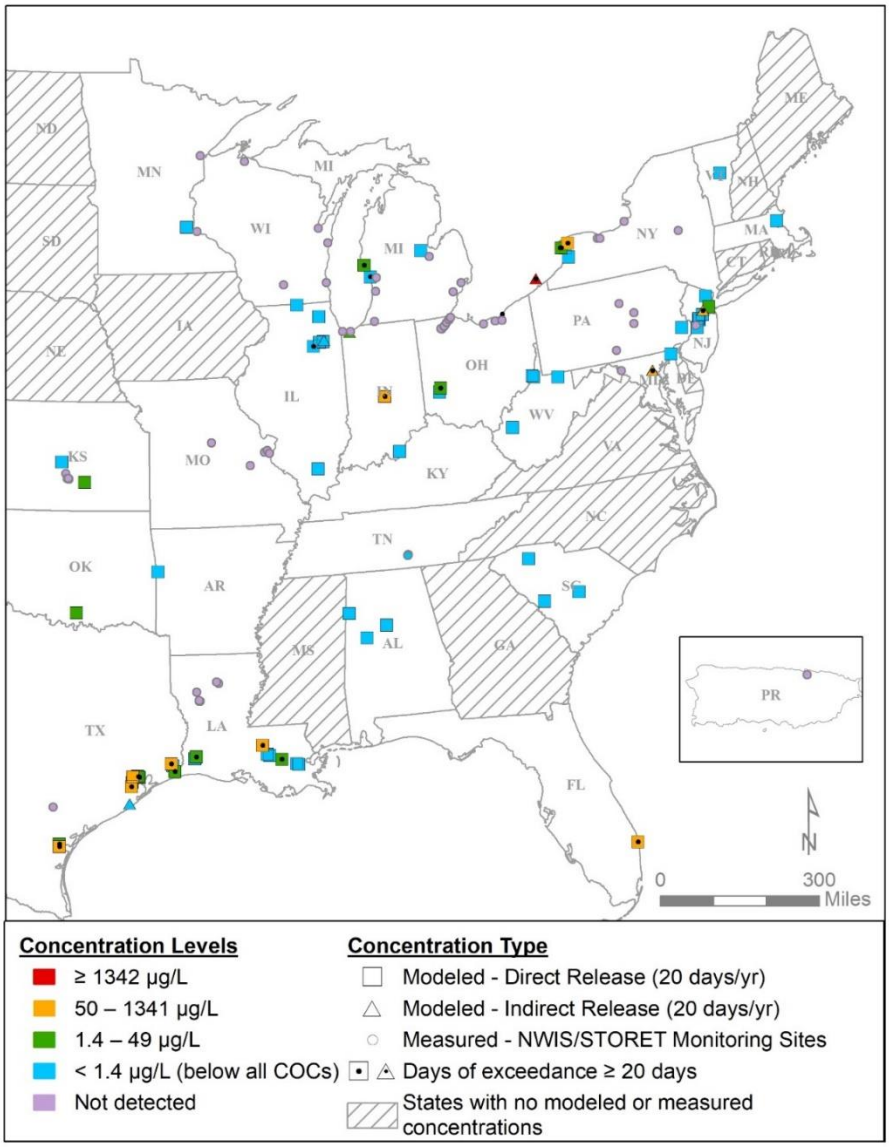
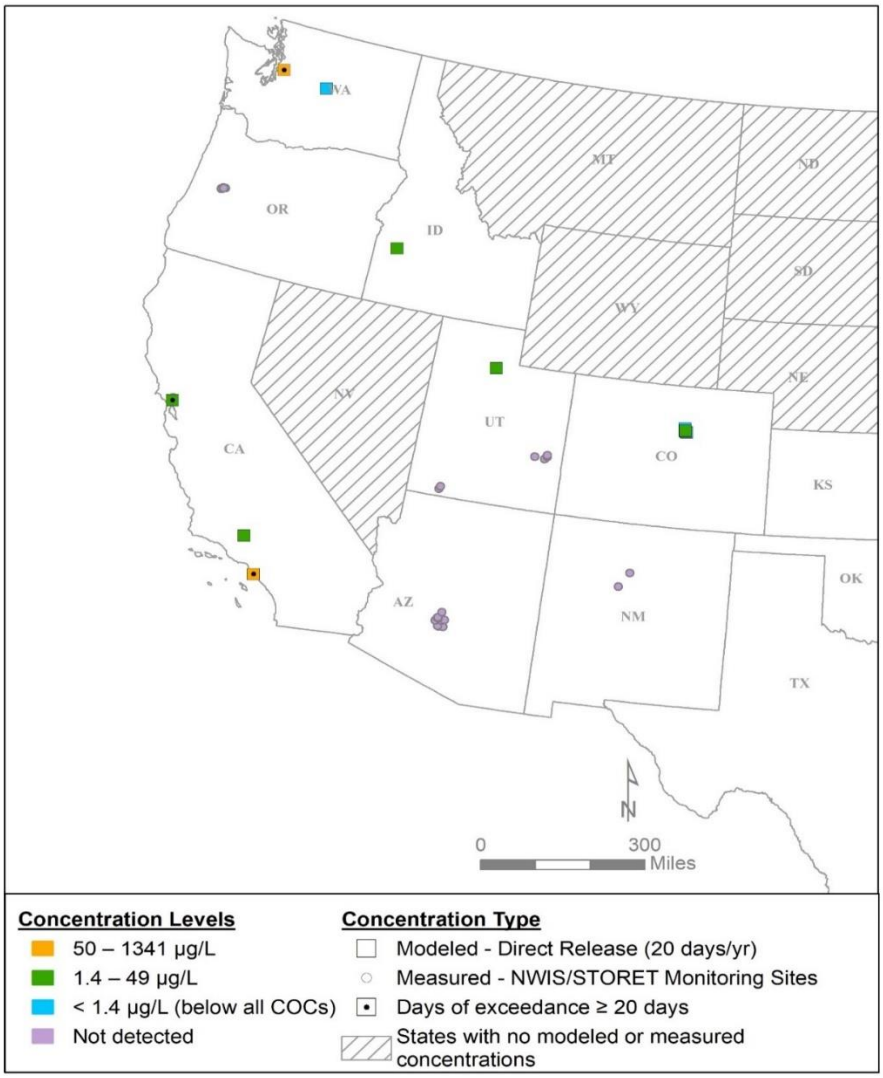


Figure 4-4. Concentrations of PCE from PCE-Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, West US. All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.



4.1.2 Risk Estimation for Aquatic Environment

To characterize potential risk due to PCE exposure, RQs were calculated based on modeled data from E-FAST (U.S. EPA 2014b) for sites that had surface water discharges of PCE according to TRI and DMR data (Table 4-1**)**. Surface water concentrations of PCE were modeled for 97 releases: six manufacturing releases, four import/repackaging, 18 processing as a reactant releases, four incorporation into formulation, 17 open top vapor degreasing releases, two industrial dry cleaning releases, One commercial dry cleaning release (based on data from 12,822 facilities), five maskants for chemical milling releases, 12 industrial processing aid releases, eight other industrial use releases, seven other commercial uses releases, and 13 waste handling, disposal, treatment, and recycling releases. Direct releases facilities (releasees from an active facility directly to surface water) were modeled with two scenarios based on 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) were only modeled with a high-end days of releases scenario. As stated in Section 2.3.1.1, the maximum releases frequency (200 to 365 days) is based on release estimates specific to the facility's condition of use and the low-end releases frequency (20 days) is an estimate of releases that could lead to chronic risk for aquatic organisms.

As stated previously, the frequency and duration of exposure affects potential for adverse effects in aquatic organisms. Therefore, the number of days a COC was exceeded was also calculated using E-FAST. Facilities with RQs and days of exceedance that indicate risk for aquatic organisms (facilities with an acute RQ ≥ 1, or a chronic or algae RQ ≥ 1 and 20 days or more of exceedance for the chronic or algae COC) are presented in Table 4-110.

Confidence in Risk Estimation for Aquatic Environment

Confidence ratings for aquatic exposure scenarios are informed by uncertainties surrounding inputs and approaches used in modeling surface water concentrations. Other considerations that impact confidence in the aquatic exposure scenarios include the model used (E-FAST 2014, (U.S. EPA 2014b)) and its associated default and user-selected values and related uncertainties. As described in Section 2.3.4.4, there are uncertainties related to the ability of E-FAST 2014 (U.S. EPA 2014b) to incorporate downstream fate and transport; the likely number of release days from given discharging facilities; and in some cases (i.e., when the NPDES for the discharging facility cannot be found within the E-FAST database), the applied stream flow distribution. Based on the data quality, uncertainties, and weight of scientific evidence, confidence in the surface water concentration estimate is medium.

Based on the data quality, weight of scientific evidence, and uncertainties, confidence in acute and chronic COCs for fish and invertebrates are high. The COC for algae is based on a single study that EPA assigned an overall quality level of medium. Additionally, algae species tend to vary widely in their sensitivity to chemical pollutants, and data were only available for three algal species and may not represent the most sensitive species at a given site. Therefore, confidence in algae COC is medium. The overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium based on the surface water PCE concentration and COC confidence levels.

Manufacturing

Six facilities were manufacturing PCE. Two of these facilities had RQs ≥ 1 and 20 days or more of exceedance for algae. Exceedances occurred using direct and indirect scenarios.

- Greenchem, West Palm Beach, FL: Using the scenario of 350 days of maximum direct release to surface water resulted in a surface water concentration of 18 ppb, algae had an RQ = 13 and 189 days of exceedance, with average direct release concentration resulted in a surface water concentration of 5.6 ppb, algae had an RQ = 4.0 and 100 days of exceedance. Using the

maximum indirect release (80% removal) release scenario to surface water resulted in a surface water concentration of 3.7 ppb, algae had an RQ = 2.7 and 77 days of exceedance.

- Univar USA Inc, Redmond, WA: Using the scenario of 350 days of maximum direct release to surface water resulted in a surface water concentration of 18 ppb, algae had an RQ = 13 and 189 days of exceedance. With average direct release concentration from 350 days of direct release resulted in a surface water concentration of 5.6 ppb, algae had an RQ = 4.0 and 100 days of exceedance. Using the maximum indirect release (80% removal) scenario to surface water resulted in a surface water concentration of 3.7 ppb, algae had an RQ = 2.6 and 100 days of exceedance.

Four of the six facilities in the Manufacturing COU did not have NPDES permits. Lack of a NPDES permit increases the uncertainty in the surface water release estimate for those facilities. EPA identified risk to algae from direct and indirect release of PCE to surface water from two of the facilities without NPDES permits. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Import/Repackaging

Of the four facilities importing/repackaging PCE, a single facility, Hubbard-Hall Inc, Waterbury, CT, had RQs ≥ 1 and 20 days or more of exceedance for algae. Using the scenario of 250 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 29 ppb, algae had an RQ = 21 and 230 days of exceedance. Using the scenario of 20 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 360 ppb, algae had an RQ = 257 and 20 days of exceedance.

EPA identified risk to algae with 80% PCE removal from waste water treatment at one of the four facilities in the Import/Repackaging COU. Indicating that with the Import/Repackaging COU, risk to algae can exist even with waste water treatment if the rate of PCE release to surface water is high. This was also the only facility lacking a NPDES permit which increases the uncertainty associated with the surface water release estimate. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Processing as a Reactant

Of the 18 facilities processing PCE as a reactant, six facilities had RQs ≥ 1 and 20 days or more of exceedance for aquatic organisms. All exceedances occurred using the direct release to surface water scenario.

- Dupont-Chemours Montague Site, Montague, MI: Using the scenario of 350 days of direct release to still surface water resulted in a surface water concentration of 2.4 ppb, algae had an RQ = 1.7 and 350 days of exceedance. Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 35 ppb, algae had an RQ = 25 and 20 days of exceedance.
- Eagle U.S. 2 LLC - Lake Charles Complex, Lake Charles, LA: Using the scenario of 350 days of direct release to surface water resulted in a surface water concentration of 1.5 ppb, algae had an RQ = 1.1 and 29 days of exceedance.
- Flint Hills Resources Corpus Christi LLC - West Plant, Corpus Christi, TX: Using the scenario of 350 days of direct release to still surface water resulted in a surface water concentration of 3.0 ppb, algae had an RQ = 2.2 and 350 days of exceedance. Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 52 ppb, algae had an RQ

= 37 and 20 days of exceedance, and aquatic invertebrates had a chronic RQ = 1.0 and 20 days of exceedance.

- Honeywell International Inc-Baton Rouge Plant, Baton Rouge, LA: Using the scenario of 350 days of direct release to surface water resulted in a surface water concentration of 4.9 ppb, algae had an RQ = 3.5 and 193 days of exceedance. Using the scenario of 20 days of direct release to surface water resulted in a surface water concentration of 85 ppb, algae had an RQ = 61 and 20 days of exceedance.
- Keeshan And Bost Chemical Co., Inc., Manvel, TX: Using the scenario of 350 days of direct release to still surface water resulted in a surface water concentration of 5.0 ppb, algae had an RQ = 3.6 and 350 days of exceedance. Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 100 ppb, algae had an RQ = 71 and 20 days of exceedance, and aquatic invertebrates had a chronic RQ = 2.0 and 20 days of exceedance.
- Premcor Refining Group Inc Port Arthur, Port Arthur, TX: Using the scenario of 350 days of direct release to surface water resulted in a surface water concentration of 2.0 ppb, algae had an RQ = 1.4 and 67 days of exceedance.

EPA identified risk to algae and a chronic risk to aquatic organisms from direct release of PCE to surface water from the Processing as a Reactant COU at six facilities. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Incorporation into Formulation

Of the four facilities using PCE for incorporation into formulations, a single facility, Lord Corp, Saegertown, PA, had RQs ≥ 1 for acute risks, and RQs ≥ 1 and 20 days or more of exceedance for chronic and algae risks. Using the scenario of 300 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 136 ppb, algae had an RQ = 97 and 299 days of exceedance, and aquatic invertebrates had a chronic RQ = 2.7 and 127 days of exceedance. Using the scenario of 20 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 2034 ppb, algae had an RQ = 1,453 and 20 days of exceedance, aquatic invertebrates had an acute RQ = 1.5 and a chronic RQ = 41 with 20 days of exceedance.

EPA identified elevated acute and chronic risk to aquatic organisms from direct release of PCE to surface water from the Incorporation into Formulation COU at a single facility. The facility showing risk has a NPDES permit. However, one of the facilities that was not identified with risk lacked a NPDES permit. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Open Top Vapor Degreasing

Of the 17 open-top vapor degreasing facilities, two facilities had RQs ≥ 1 and 20 days or more of exceedance for algae.

- Equistar Chemicals LP, La Porte, TX: Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 3.2 ppb, algae had an RQ = 2.3 and 20 days of exceedance.
- GM Components Holdings LLC, Lockport, NY: Using the scenario of 260 days of direct release to surface water resulted in a surface water concentration of 5.9 ppb, algae had an RQ = 4.2 and

131 days of exceedance. Using the scenario of 20 days of direct release to surface water resulted in a surface water concentration of 78 ppb, algae had an RQ = 56 and 20 days of exceedance.

EPA identified risk to algae from direct release of PCE to surface water from the Open Top Vapor Degreasing COU at two facilities. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Dry Cleaning (Industrial and Commercial)

Two industrial and One commercial dry cleaning releases (based on data from 12,822 facilities) were modeled for the risk estimate. The model used both high-end and central tendency release data for direct and indirect releases. None of the facility releases show a surface water concentration that resulted in an RQs ≥ 1 for acute risk or RQs ≥ 1 and 20 days of exceedance for chronic or algal risk.

No risks were identified for aquatic organisms with this COU. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Maskants for Chemical Milling

Releases from five maskants for chemical milling facilities were modeled for the risk estimate. The model used direct and indirect releases to surface water including still water bodies. None of the facility releases show a surface water concentration that resulted in an RQs ≥ 1 or any days of exceedance.

No risks were identified for aquatic organisms with this COU. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Industrial Processing Aid

Of the 12 industrial processing aid facilities, six facilities had RQs ≥ 1 and 20 days or more of exceedance for algae.

- Chevron Products Co Richmond Refinery, Richmond, CA: Using the scenario of 20 days of direct release to surface water resulted in a surface water concentration of 2.7 ppb, algae had an RQ = 1.9 and 20 days of exceedance.
- ExxonMobil Oil Beaumont Refinery Beaumont, TX: Using the scenario of 300 days of direct release to surface water resulted in a surface water concentration of 5.5 ppb, algae had an RQ = 4.0 and 55 days of exceedance. Using the scenario of 20 days of direct release to surface water resulted in a surface water concentration of 97 ppb, algae had an RQ = 69 and 20 days of exceedance.
- Marathon Petroleum Co LP, Garyville, LA: Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 6.6 ppb, algae had an RQ = 4.7 and 20 days of exceedance.
- Occidental Chemical Corp Niagara Plant, Niagara Falls, NY: Using the scenario of 300 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 6.3 ppb, algae had an RQ = 4.5 and 92 days of exceedance. Using the scenario of 20 days of direct release to still surface water resulted in a surface water concentration of 20 ppb, algae had an RQ = 14 and 20 days of exceedance.
- Tesoro Los Angeles Refinery-Carson Operations, Carson, CA: Using the scenario of 300 days of direct release to surface water resulted in a surface water concentration of 12 ppb, algae had an RQ = 8.5 and 169 days of exceedance.

- Valero Refining Co -Oklahoma Valero Ardmore Refinery, Ardmore, OK: Using a surrogate organic chemicals manufacturer, with 300 days of direct release to surface water resulted in a surface water concentration of 1.9 ppb, algae had an RQ = 1.3 and 42 days of exceedance.

EPA identified risk to algae from direct and indirect releases of PCE to surface water from the Industrial Processing Aid COU at six facilities. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Other Industrial Uses

Releases from seven with other industrial use facilities were modeled for the risk estimate. The model used direct releases to surface water. None of the facility releases show a surface water concentration that resulted in an RQs ≥ 1 or RQs ≥ 1 and 20 days of exceedance for chronic or algal risk.

No risks were identified for aquatic organisms with this COU. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Other Commercial Uses

Releases from seven other commercial use facilities were modeled for the risk estimate. The model used direct releases to surface water. None of the facility releases show a surface water concentration that resulted in an RQs ≥ 1 or RQs ≥ 1 and 20 days of exceedance for chronic or algal risk.

No risks were identified for aquatic organisms with this COU. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Waste Handling, Disposal, Treatment, and Recycling

Of the 13 facilities engaged in waste handling, disposal, treatment, and recycling of PCE, three facilities had RQs ≥ 1 and 20 days of exceedance for algae.

- Clean Harbors Deer Park LLC, La Porte, TX: Using the scenario of 250 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 9.0 ppb, algae had an RQ = 6.4 and 172 days of exceedance. Using the scenario of 20 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 113 ppb, algae had an RQ = 80 and 20 days of exceedance.
- Safety-Kleen Systems Inc, Smithfield, KY: Using the scenario of 250 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 35 ppb, algae had an RQ = 25 and 235 days of exceedance. Using the scenario of 20 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 436 ppb, algae had an RQ = 311 and 20 days of exceedance.
- Tier Environmental LLC, Bedford, OH: Using the scenario of 250 days of indirect release (80% removal) to surface water resulted in a surface water concentration of 3.1 ppb, algae had an RQ = 2.2 and 90 days of exceedance.

EPA identified risk to algae with 80% PCE removal from waste water treatment at three facilities. Indicating that with the Waste Handling, Disposal, Treatment, and Recycling COU, risk to algae can exist even with waste water treatment if the rate of PCE release to surface water is high. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

4.1.3 Risk Estimation for Sediment Pathways

EPA did not quantitatively analyze exposure to sediment organisms. PCE is expected to be moderately retained in sediment due to its water solubility (206 mg/L) and moderate partitioning to organic matter (log KOC = 2.95). Because PCE has moderate partitioning to organic matter, in sediments PCE is expected to be both adsorbed to the sediment organic matter and present in the pore water. However, depending on the microbial consortia present and their previous exposure and adaptation to PCE, PCE may undergo rapid biodegradation in sediment. Thus, PCE concentrations in sediment may be lower or somewhat greater than concentrations in overlying water. While no ecotoxicity studies were available for sediment-dwelling organisms (e.g., *Lumbriculus variegatus, Hyalella azteca, Chironomus riparius*), the toxicity of PCE to sediment invertebrates is expected to be similar to the toxicity to aquatic invertebrates because of the similarities in PCE concentrations. EPA calculated an acute aquatic invertebrate COC of 1,342 ppb, and a chronic aquatic invertebrate COC of 50 ppb to assess hazards to sediment organisms.

4.1.4 Risk Estimation for Land-Applied Biosolids Pathway

EPA did not analyze PCE for other releases to land during risk evaluation, including biosolids application to soil as indicated in the Problem Formulation.

EPA did not assess exposure to terrestrial organisms through soil, land-applied biosolids, or ambient air. PCE has moderate potential to partition to or accumulate in soil, but is primarily expected to volatilize to air or migrate through soil into groundwater based on its physical-chemical properties (log K_{OC} = 3, Henry's Law constant = 0.018 atm-m^3/mole, vapor pressure = 19 mmHg at 20°C). Therefore, physical-chemical properties do not support an exposure pathway through water and soil pathways to terrestrial organisms.

4.2 Human Health Risk

PCE 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. increased latencies for pattern reversal visual-evoked potentials) from acute exposure, developmental toxicity from repeated exposures (i.e. longer than acute, single day exposures and shorter than chronic, many year exposures) and multiple effects including CNS, kidney, liver and immune system toxicity from chronic exposures. The evaluation of cancer includes estimates of risk of lung and liver tumors.

4.2.1 Risk Estimation Approach

Equation 4-1 was used to calculate non-cancer risks using margins of exposure for acute or chronic exposure durations.

Equation 4-1 Equation to Calculate Non-Cancer Risks Following Acute or Chronic Exposures Using Margin of Exposures

$$MOE_{acute\ or\ chronic} = \frac{Non-cancer\ Hazard\ value\ (POD)}{Human\ Exposure}$$

Where:

MOE = Margin of exposure (unitless)
Hazard value (POD) = HEC (ppm)
Human Exposure = Exposure estimate (in ppm) from occupational or consumer exposure

assessment. ADCs were used for non-cancer chronic risks and acute concentrations were used for acute risks (see Section 3.2.5)

EPA/OPPT used margin of exposures (MOEs)[18] to estimate acute or chronic risks for non-cancer based on the following:

1. the lowest HECs within each health effects domain reported in the literature;
2. the endpoint/study-specific UFs applied to the HECs per the EPA Guidance (U.S. EPA, 2002); and
3. the exposure estimates calculated for PCE uses examined in this risk assessment (see Section 2 Exposures).

MOEs allow for the presentation of a range of risk estimates. The occupational exposure scenarios considered both acute and chronic exposures. All consumer uses considered only acute exposure scenarios. Different adverse endpoints were used based on the expected exposure durations. For non-cancer effects, risks for neurotoxicity (i.e. increased latencies for pattern reversal visual-evoked potentials) from acute exposure were evaluated.

For occupational exposure calculations, the 8 hr or 12 hr TWA was used to calculate inhalation MOEs for risk estimates for acute exposures and the chronic average daily concentration (ADC) was used for chronic exposures. For dermal estimates, acute and chronic retained doses were used. The total UF for each non-cancer POD 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. Risk estimates were calculated for all of the studies per health effects domain that EPA/OPPT considered suitable for the risk evaluation of acute and chronic exposure scenarios in the work plan risk assessment for PCE.

The PBPK model (Section 3.2.2.2) allowed it to be used to calculate internal dose metrics for inhaled and oral exposure to PCE for mice, rats, and humans and therefore was used for route-to-route extrapolation between oral and inhalation routes. Dermal candidate values were calculated based on route-to-route extrapolation from two different routes either inhalation or oral PODs. The PODs were extrapolated from POD values based on either human data or human equivalent values (e.g. $BMDL_{HEC}$) which have already been adjusted to account for animal to human extrapolation using the best available approaches for incorporating PCE specific toxicokinetic data (i.e. the PBPK model) when possible. When dermal HEDs were derived by both methods, the most sensitive resulting HED was selected for use in risk estimation in order to be health-protective.

Added cancer risks for repeated exposures to PCE were estimated using Equation 4-2. Estimates of added 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 added individual lifetime cancer risk).

[18] 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 3-5.

Equation 4-2 Equation to Calculate Added Cancer Risks

$$Risk = Human\ Exposure \times IUR$$

Where:

Risk = Added cancer risk (unitless)
Human exposure = Exposure estimate (LADC in mg/m^3) from occupational exposure assessment
IUR = Inhalation unit risk (2×10^{-3} per mg/m^3)

4.2.2 Risk Estimation for Inhalation Exposures to Workers

4.2.2.1 PODs used for Occupational Inhalation Risk Estimates

The risk assessment used the inhalation exposure estimates in Section 2.4.1 and the hazard PODs summarized in Table 3-7, Table 3-8, and Table 3-9. For acute exposure scenarios, PODs for 8 and 12hr exposure durations were used because those durations are most applicable to occupational exposure scenarios. From among all chronic studies, EPA selected the most robust studies and non-cancer PODs from within each health domain to serve as representative endpoints for risk estimation (Section 3.2.5.4). These representative PODs are presented below in Table 4-2 along with the acute POD. Non-cancer risk estimates were calculated with equation 4-1 and cancer risks were calculated with equation 4-2. Risk is indicated for each OES or COU by bold text and a shaded cell in the table.

Table 4-2. Selected Non-cancer PODs for Use in Risk Estimation of Inhalation Exposures

Target Organ System	Species	Human Equivalent Concentration (HEC)	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality Score
ACUTE EXPOSURE						
CNS	Humans	8 hrs/day = 5 ppm (34 mg/m^3) 12 hrs/day = 3.3 ppm (22 mg/m^3)	Neurotoxicity increased latencies for pattern reversal visual-evoked potentials	UF_A=1; UF_H=10; UF_L=1 **Total UF=10**	Altmann et al. (1990)	Medium
CHRONIC EXPOSURE						
CNS	Humans	5.2 ppm (36 mg/m^3)	Midpoint of the range of the two neurotoxicity studies	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Based on U.S. EPA (2012c)	Medium
Kidney	Mice	2.1 ppm (14 mg/m^3)	Nuclear enlargement in proximal tubules	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (1993)	High
Liver	Mice	31 ppm (210 mg/m^3)	Increased angiectasis in liver	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (1993)	High
Reproductive/ Developmental	*Reproductive*					
	Mice	21 ppm (140 mg/m^3)	Reduced sperm quality following 5 days exposure	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Beliles et al. (1980)	High

Target Organ System	Species	Human Equivalent Concentration (HEC)	Effect	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality Score
	Developmental					
	Rats	18 ppm (122 mg/m^3)	Increased F_{2A} pup deaths by Day 29, CNS depression in F_1 and F_2	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Tinston et al. (1994)	High
CANCER						
Liver	Mouse	IUR 2×10^{-3} per ppm (3×10^{-4} per mg/m^3)	Hepatocellular tumors (males)	N/A	JISA (1993)	High

EPA also provided chronic inhalation risk estimates as a sensitivity analysis based on 8 hr and 12 hr occupational neurotoxicity HECs (14.5 ppm and 9.7 ppm, respectively, see Table 3-8) compared to 8 hr or 12 hr TWA exposures. These risk estimates are approximately 36% lower than the risk estimates using the chronic HECs based on continuous 24 hr exposure. See Appendix G for risk estimates for all OES.

4.2.2.2 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 respirator use up to APF = 50 was plausible for those OES based on expert judgement and reasonably available information. Table 4-3 presents this information below, which is considered in the risk characterization for each OES in the following sections.

Table 4-3. 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	152 (75 8-hr TWA and 77 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
Repackaging	Monitoring data	10	N/A – monitoring data only	Not assessed	May use respirators	Industrial
Processing as a Reactant	Surrogate monitoring data from manufacturing	152 (75 8-hr TWA and 77 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
Incorporation into Formulation – Aerosol Packing	Monitoring data	5	N/A – monitoring data only	Not assessed	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
Incorporation into Formulation – Non-Aerosol Formulations	Modeling	N/A – model only	EPA/OAQPS AP-42 Loading Model & EPA/OPPT Mass Balance Model	Not assessed	May use respirators	Industrial
Open-Top Vapor Degreasing	Monitoring data	75 (63 worker and 12 ONUs)	N/A – monitoring data only	ONU monitoring data available	May use respirators	Industrial/ Commercial
Closed-Loop Vapor Degreasing	Monitoring data	15 (13 worker and 2 ONU)	N/A – monitoring data only	ONU monitoring data available	May use respirators	Industrial/ Commercial
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/ Commercial
Web Degreasing	Model	N/A – model only	Web Degreasing Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	May use respirators	Industrial/ Commercial
Cold Cleaning	Monitoring data supplemented by model	29	Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	May use respirators	Industrial/ Commercial
Aerosol Degreasing and Aerosol Lubricants	Monitoring data supplemented by model	130	Brake Servicing Near-Field/Far-Field Inhalation Exposure Model	Far-field model results	No respirator use – commercial use	Commercial
Dry Cleaning	Monitoring data supplemented by model	140 (135 workers and 5 ONUs)	Dry Cleaning Multi-Zone Inhalation Exposure Model	ONU monitoring data available supplemented by far-field model results	No respirator use – commercial use	Commercial
Paint and Coatings	Monitoring data	15	N/A – monitoring data only	Not assessed	May use respirators	Industrial/ Commercial
Adhesives	Monitoring data	13	N/A – monitoring data only	Not assessed	May use respirators	Industrial/ Commercial
Chemical Maskant	Monitoring data	24	N/A – monitoring data only	Not assessed	May use respirators	Industrial
Industrial Processing Aid	Monitoring data	89	N/A – monitoring data only	Not assessed	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
Other Industrial Uses	Model	N/A – model only	Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model	Not assessed	May use respirators	Industrial
Metalworking Fluid	Emission scenario document	N/A – emission scenario document	Estimates from Use of Metalworking Fluids ESD	Not assessed	No respirator use – ESD indicates respirators are not generally used	Industrial/ Commercial
Wipe Cleaning	Monitoring data	10 (4 workers and 6 ONUs)	N/A – monitoring data only	ONU monitoring data available	No respirator use – commercial use	Commercial
Other Spot Cleaning/Spot Removers (including Carpet Cleaning)	Monitoring data	3 (2 workers and 1 ONU)	N/A – monitoring data only	ONU monitoring data available	No respirator use – commercial use	Commercial
Other Commercial Uses	Monitoring data	92	N/A – monitoring data only	Not assessed	No respirator use – commercial use	Commercial
Other DoD Uses	Monitoring data	2	N/A – monitoring data only	Not assessed	May use respirators	Industrial/ Commercial
Disposal/Recycling	Model	N/A – model only	Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model	Not assessed	May use respirators	Industrial

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4.2.2.3 Manufacturing

For manufacturing, exposure estimates for TWAs of 15 mins, 30 mins, 8 hrs, and 12 hrs are available based on personal monitoring data samples, including 351 data points from one source. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Data were not available to estimate ONU exposures; EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is high for workers and low for ONUs. Section 2.4.1.6 describes the justification for this occupational scenario confidence rating.

Table 4-4. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Manufacturing

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**1.9**	154	19	48	96	10
		Central Tendency	154		1,538	3,846	7,692	
12-hr	3.3	High-End	16	161	156	389	778	10
		Central Tendency	161		1,610	4,024	8,049	

[1] Data from Altmann et al. (1990)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-5. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Manufacturing

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure data for 8 hr TWA								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**8.7**	701	**87**	218	436	100
		Central Tendency	701		7,008	17,520	35,040	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**3.5**	283	35	88	176	30
		Central Tendency	283		2,830	7,075	14,151	
Liver - Vessel dilation (JISA 1993)	31	High-End	52	4,178	520	1,300	2,599	30
		Central Tendency	4,178		41,778	104,446	208,892	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	35	2,830	352	880	1,761	30
		Central Tendency	2,830		28,302	70,754	141,508	

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	30	2,426	302	755	1,509	30
		Central Tendency	2,426		24,258	60,646	121,292	
Based on exposure data for 12 hr TWA								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**72**	741	716	1,791	3,581	100
		Central Tendency	741		7,407	18,517	37,034	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**29**	299	289	723	1,446	30
		Central Tendency	299		2,991	7,478	14,956	
Liver - Vessel dilation (JISA 1993)	31	High-End	427	4,416	4,270	10,675	21,349	30
		Central Tendency	4,416		44,156	110,390	220,780	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	289	2,991	2,892	7,231	14,462	30
		Central Tendency	2,991		29,912	74,780	149,561	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	30	2,426	302	755	1,509	30
		Central Tendency	2,426		24,258	60,646	121,292	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-6. Risk Estimation for Chronic, Cancer Inhalation Exposures for Manufacturing

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure data for 8 hr TWA								
Cancer Risk liver tumors	2.0E-3	High-End	**6.1E-4**	5.9E-6	6.1E-5	2.4E-5	1.2E-5	10^{-4}
		Central Tendency	5.9E-6		5.9E-7	2.4E-7	1.2E-7	
Based on exposure data for 12 hr TWA								
Cancer Risk liver tumors	2.0E-3	High-End	7.5E-5	5.6E-6	7.5E-6	3.0E-6	1.5E-6	10^{-4}
		Central Tendency	5.6E-6		5.6E-7	2.2E-7	1.1E-7	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.4 Repackaging

For repackaging, exposure estimates for TWAs of 15 mins, 30 mins, and 8 hrs are available based on personal monitoring data samples, including 17 data points from 1 source. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively, for the 8-hr TWAs. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, for the 15- and 30-min TWAs. EPA has not identified reasonably available data on potential ONU inhalation exposures from PCE 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 2.4.1.7. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.7 describes the justification for this occupational scenario confidence rating.

Table 4-7. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Import/Repackaging

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**6.1**	11	61	153	305	10
		Central Tendency	11		115	287	574	

[1] Data from Altmann et al. (1990)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-8. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Import/Repackaging

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**28**	**52**	278	695	1,390	100
		Central Tendency	**52**		523	1,308	2,617	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**11**	**21**	112	281	561	30
		Central Tendency	**21**		211	528	1,057	
Liver - Vessel dilation (JISA 1993)	31	High-End	166	312	1,657	4,413	8,287	30
		Central Tendency	312		3,120	7,799	15,599	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	112	211	1,123	2,807	5,614	30
		Central Tendency	211		2,113	5,283	10,567	
Developmental -	18	High-	96	181	962	2,406	4,812	30

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Mortality/ CNS effects (Tinston 1994)		End						
		Central Tendency	181		1,811	4,529	9,057	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-9. Risk Estimation for Chronic, Cancer Inhalation Exposures for Import/Repackaging

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk liver tumors	2.0E-3	High-End	**1.9E-4**	7.9E-5	1.9E-5	7.7E-6	3.8E-6	10^{-4}
		Central Tendency	7.9E-5		7.9E-6	3.2E-6	1.6E-6	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.5 Processing as Reactant

For processing as a reactant, exposure estimates for TWAs of 15 mins, 30 mins, and 8 hrs are available based on surrogate personal monitoring data samples, including 351 data points from one source. EPA uses surrogate data for PCE manufacturing to approximate exposures during processing as a reactant as monitoring data specific to this condition of use were not available and manufacturing sites and sites processing PCE as a reactant are expected to have similar operations. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Data were not available to estimate ONU exposures; EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. 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 for workers and low for ONUs. Section 2.4.1.8 describes the justification for this occupational scenario confidence rating.

Table 4-10. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Processing as Reactant

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**1.9**	154	19	48	96	10
		Central Tendency	154		1,538	3,846	7,692	
12-hr	3.3	High-End	16	161	156	389	778	10

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
		Central Tendency	161		1,610	4,024	8,049	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-11. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Processing as Reactant

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure data for 8 hr TWA								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**8.7**	701	**87**	218	436	100
		Central Tendency	701		7,008	17,520	35,040	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**3.5**	283	35	88	176	30
		Central Tendency	283		2,830	7,075	14,151	
Liver - Vessel dilation (JISA 1993)	31	High-End	52	4,178	520	1,300	2,599	30
		Central Tendency	4,178		41,778	104,446	208,892	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	35	2,830	352	880	1761	30
		Central Tendency	2,830		28,302	70,754	141,508	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	30	2,426	302	755	1,509	30
		Central Tendency	2,426		24,258	60,646	121,292	
Based on exposure data for 12 hr TWA								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**72**	741	716	1,791	3,581	100
		Central Tendency	741		7,407	18,517	37,034	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**29**	299	289	723	1,446	30
		Central Tendency	299		2,991	7,478	14,956	
Liver - Vessel dilation (JISA 1993)	31	High-End	427	4,416	4,270	10,675	21,349	30
		Central Tendency	4,416		44,156	110,390	220,780	
Reproductive - Sperm effects	21	High-End	289	2,991	2,892	7,231	14,462	30

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
(Beliles et al. 1980)		Central Tendency	2,991		29,912	74,780	149,561	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	248	2,564	2,479	6,198	12,396	30
		Central Tendency	2,564		25,639	64,098	128,195	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-12. Risk Estimation for Chronic, Cancer Inhalation Exposures for Processing as Reactant

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure data for 8 hr TWA								
Cancer Risk liver tumors	2.0E-3	High-End	**6.1E-4**	5.9E-6	6.1E-5	2.4E-5	1.2E-5	10^{-4}
		Central Tendency	5.9E-6		5.9E-7	2.4E-7	1.2E-7	
Based on exposure data for 12 hr TWA								
Cancer Risk liver tumors	2.0E-3	High-End	7.5E-5	5.6E-6	7.5E-6	3.0E-6	1.5E-6	10^{-4}
		Central Tendency	5.6E-6		5.6E-7	2.2E-7	1.1E-7	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.6 Incorporation into Formulation, Mixture, or Reactant Product

For incorporation into formulation, mixture, or reaction product, exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples for aerosol packing, including 5 data points from one source, and modeling for degreasing solvent, dry cleaning solvent, and miscellaneous product formulations. For aerosol packing, EPA calculated the median and maximum to characterize the central tendency and high-end exposure estimates, respectively. For the other formulation types, EPA calculated 50^{th} and 95^{th} percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified reasonably available data to estimate potential ONU inhalation exposures from PCE incorporation into formulation, mixture, or reaction product using monitoring data or modeling. 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 2.4.1.9. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the aerosol packing inhalation estimates in this scenario is high for workers and low for ONUs and EPA's overall confidence in the modeled exposures for other formulation types is medium for workers and low for ONUs. Section 2.4.1.9 describes the justification for this occupational scenario confidence rating.

Table 4-13. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Incorporation into Formulation, Mixture, or Reactant Product

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Aerosol Packing								
8-hr	5.0	High-End	**0.4**	**0.6**	**3.8**	**9.5**	19	10
		Central Tendency	**0.6**		**6.0**	15	30	
Degreasing Solvent								
8-hr	5.0	High-End	**1.9**	**6.9**	19	48	96	10
		Central Tendency	**6.9**		69	171	343	
Dry Cleaning Solvent								
8-hr	5.0	High-End	**0.4**	**1.3**	**3.5**	**8.9**	18	10
		Central Tendency	**1.3**		13	32	63	
Miscellaneous								
8-hr	5.0	High-End	**3.5**	13	35	89	177	10
		Central Tendency	13		126	315	629	

[1] Data from Altmann et al. (1990)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-14. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Incorporation into Formulation, Mixture, or Reactant Product

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Aerosol Packing								
CNS - Visual Effects	5.2	High-End	**1.7**	**2.7**	**17**	**43**	**87**	100
		Central Tendency	**2.7**		**27**	**69**	137	
Kidney - Histopathology	2.1	High-End	**0.7**	**1.1**	**7.0**	**18**	35	30
		Central Tendency	**1.1**		**11**	**28**	55	
Liver - Vessel dilation	31	High-End	**10**	**16**	103	258	517	30
		Central Tendency	**16**		164	410	819	
Reproductive - Sperm Effects	21	High-End	**7.0**	**11**	70	175	350	30
		Central Tendency	**11**		111	277	555	
Developmental - Mortality/CNS	18	High-End	**6.0**	**9.5**	60	150	300	30
		Central Tendency	**9.5**		95	237	475	

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Degreasing Solvent								
CNS - Visual Effects	5.2	High-End	**92**	328	918	2,296	4,591	100
		Central Tendency	328		3,277	8,194	16,387	
Kidney - Histopathology	2.1	High-End	37	132	371	927	1,854	30
		Central Tendency	132		1,324	3,309	6,618	
Liver - Vessel dilation	31	High-End	547	1,954	5,474	13,685	27,371	30
		Central Tendency	1,954		19,539	48,846	97,693	
Reproductive - Sperm Effects	21	High-End	371	1,324	3,708	9,271	18,542	30
		Central Tendency	1,324		13,236	33,089	66,179	
Developmental - Mortality/CNS	18	High-End	318	1,134	3,179	7,946	15,893	30
		Central Tendency	1,134		11,345	28,362	56,725	
Dry Cleaning Solvent								
CNS - Visual Effects	5.2	High-End	**17**	**60**	169	423	847	100
		Central Tendency	**60**		604	1,509	3,018	
Kidney - Histopathology	2.1	High-End	**6.8**	**24**	68	171	342	30
		Central Tendency	**24**		244	609	1,219	
Liver - Vessel dilation	31	High-End	101	360	1,009	2,523	5,047	30
		Central Tendency	360		3,599	8,996	17,993	
Reproductive - Sperm Effects	21	High-End	68	244	684	1,709	3,419	30
		Central Tendency	244		2,438	6,094	12,189	
Developmental - Mortality/CNS	18	High-End	59	209	586	1,465	2,930	30
		Central Tendency	209		2,089	5,224	10,447	
Miscellaneous								
CNS - Visual Effects	5.2	High-End	169	602	1,693	4,231	8,463	100
		Central Tendency	602		6,016	15,041	30,082	
Kidney - Histopathology	2.1	High-End	68	243	684	1,709	3,418	30
		Central Tendency	243		2,430	6,074	12,149	
Liver - Vessel dilation	31	High-End	1,009	3,587	10,090	25,226	50,451	30
		Central Tendency	3,587		35,868	89,669	179,338	
Reproductive - Sperm Effects	21	High-End	684	2,430	6,835	17,088	34,177	30
		Central Tendency	2,430		24,297	60,744	121,487	
	18	High-End	586	2,083	5,859	14,647	29,294	30

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Developmental - Mortality/CNS		Central Tendency	2,083		20,826	52,066	104,132	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-15. Risk Estimation for Chronic, Cancer Inhalation Exposures for Incorporation into Formulation, Mixture, or Reactant Product

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Aerosol Packing								
Cancer Risk liver tumors	2.0E-3	High-End	**3.1E-3**	**1.5E-3**	**3.1E-4**	**1.2E-4**	6.2E-5	10^{-4}
		Central Tendency	**1.5E-3**		**1.5E-4**	6.0E-5	3.0E-5	
Degreasing Solvent								
Cancer Risk liver tumors	2.0E-3	High-End	1.7E-5	4.7E-6	1.7E-6	6.7E-7	3.3E-7	10^{-4}
		Central Tendency	4.7E-6		4.7E-7	1.9E-7	9.4E-8	
Dry Cleaning Solvent								
Cancer Risk liver tumors	2.0E-3	High-End	9.1E-5	2.5E-5	9.1E-6	3.6E-6	1.8E-6	10^{-4}
		Central Tendency	2.5E-5		2.5E-6	1.0E-6	5.1E-7	
Miscellaneous								
Cancer Risk liver tumors	2.0E-3	High-End	9.1E-6	2.6E-6	9.1E-7	3.6E-7	1.8E-7	10^{-4}
		Central Tendency	2.6E-6		2.6E-7	1.0E-7	5.1E-8	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.7 Batch Open-Top Vapor Degreasing

For OTVDs, exposure estimates for TWAs of 15 mins, 4 hrs, and 8 hrs are available based on personal monitoring data samples, including 79 data points from multiple sources. For 8-hr TWAs, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, for the 4-hr TWA. For the 15-min TWA, exposures are based on the single data point that was available. EPA identified 12 of the 79 data points to be for ONU exposures at sites operating OTVDs as described in more detail above

in Section 2.4.1.10. 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 2.4.1.10 describes the justification for this occupational scenario confidence rating.

Table 4-16. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**0.2**	**1.0**	**1.6**	**3.9**	**7.8**	10
		Central Tendency	**2.4**	**8.2**	24	60	119	

[1] Data from Altmann et al. (1990)

Table 4-17. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**0.7**	**4.4**	**7.1**	**18**	**35**	100
		Central Tendency	**11**	**38**	108	271	542	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**0.3**	**1.8**	**2.9**	**7.2**	**14**	30
		Central Tendency	**4.4**	**15**	44	110	219	
Liver - Vessel dilation (JISA 1993)	31	High-End	**4.2**	**26**	42	106	212	30
		Central Tendency	65	224	647	1,616	3,233	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	**2.9**	**18**	29	72	143	30
		Central Tendency	44	152	438	1,095	2,190	
Developmental - Mortality/ CNS effects (Tinston 1994)	21	High-End	**2.5**	**15**	**25**	61	123	30
		Central Tendency	38	130	375	939	1,877	

Table 4-18. Risk Estimation for Chronic, Cancer Inhalation Exposures for Batch Open-Top Vapor Degreasing

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk liver tumors	2.0E-3	High-End	**7.5E-3**	**1.2E-3**	**7.5E-4**	**3.0E-4**	**1.5E-4**	10^{-4}
		Central Tendency	**3.8E-4**	**1.1E-4**	3.8E-5	1.5E-5	7.6E-6	

[1] Data from JISA (1993)

4.2.2.8 Batch Closed-Loop Vapor Degreasing

For batch closed-loop vapor degreasing, exposure estimates for TWAs of 4 hrs and 8 hrs are available based on personal monitoring data samples, including 18 data points from two sources. For worker 8-hr TWAs, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates. Due to the limited number of data points, for 4-hr TWAs and ONU 8-hr TWAs, EPA calculated the median and maximum to characterize the central tendency and high-end exposure estimates. EPA identified 2 of the 18 data points to be for ONU exposures at sites operating batch closed-loop vapor degreasers as described in more detail above in Section 2.4.1.11. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is high. Section 2.4.1.11 describes the justification for this occupational scenario confidence rating.

Table 4-19. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Batch Closed-Loop Vapor Degreasing

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	20	52	198	494	988	10
		Central Tendency	69	76	693	1,732	3,463	

[1] Data from Altmann et al. (1990)

Table 4-20. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Batch Closed-Loop Vapor Degreasing

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**90**	238	900	2,250	4,501	100
		Central Tendency	316	348	3,155	7,888	15,776	
Kidney - Histopathology (JISA 1993)	2.1	High-End	36	96	364	909	1,818	30
		Central Tendency	127	141	1,274	3,185	6,371	
Liver -	31	High-	537	1,418	5,366	13,416	26,832	30

Vessel dilation (JISA 1993)		End						
		Central Tendency	1,881	2,075	18,809	47,023	94,047	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	364	961	3,635	9,088	18,176	30
		Central Tendency	1,274	1,406	12,742	31,855	63,709	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	312	823	3,116	7,790	15,580	30
		Central Tendency	1,092	1,205	10,922	27,304	54,608	

Table 4-21. Risk Estimation for Chronic, Cancer Inhalation Exposures for Batch Closed-Loop Vapor Degreasing

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk	2.0E-3	High-End	5.9E-5	2.2E-5	5.9E-6	2.4E-6	1.2E-6	10^{-4}
		Central Tendency	1.3E-5	1.2E-5	1.3E-6	5.2E-7	2.6E-7	

[1] Data from JISA (1993)

4.2.2.9 Conveyorized Vapor Degreasing

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 PCE conveyorized vapor degreasing as described in more detail above in Section 2.4.1.12. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.12 describes the justification for this occupational scenario confidence rating.

Table 4-22. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Conveyorized Vapor Degreasing

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**2.7E-2**	**4.0E-2**	**0.3**	**0.7**	**1.3**	10
		Central Tendency	**6.4E-2**	**0.1**	**0.6**	**1.6**	**3.2**	

[1] Data from Altmann et al. (1990)

Table 4-23. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Conveyorized Vapor Degreasing

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**0.1**	**0.2**	**1.2**	**3.1**	**6.1**	100
		Central Tendency	**0.3**	**0.6**	**2.9**	**7.3**	**15**	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**4.9E-2**	**7.3E-2**	**0.5**	**1.2**	**2.5**	30
		Central Tendency	**0.1**	**0.2**	**1.2**	**2.9**	**5.9**	
Liver - Vessel dilation (JISA 1993)	31	High-End	**0.7**	**1.1**	**7.3**	**18**	37	30
		Central Tendency	**1.7**	**3.3**	**17**	43	87	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**0.5**	**0.7**	**4.9**	**12**	25	30
		Central Tendency	**1.2**	**2.3**	**12**	**29**	59	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**0.4**	**0.6**	**4.2**	**11**	**21**	30
		Central Tendency	**1.0**	**1.9**	**10**	**25**	50	

Table 4-24. Risk Estimation for Chronic, Cancer Inhalation Exposures for Conveyorized Vapor Degreasing

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk liver tumors	2.0E-3	High-End	**3.5E-2**	**2.3E-2**	**3.5E-3**	**1.4E-3**	**7.0E-4**	10^{-4}
		Central Tendency	**1.3E-2**	**7.0E-3**	**1.3E-3**	**5.4E-4**	**2.7E-4**	

[1] Data from JISA (1993)

4.2.2.10 Web Degreasing

For web 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 PCE web degreasing as described in more detail above in Section 2.4.1.13. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.13 describes the justification for this occupational scenario confidence rating.

Table 4-25. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Web Degreasing

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**2.8**	**4.3**	28	69	139	10
		Central Tendency	**8.2**	16	82	205	409	

[1] Data from Altmann et al. (1990)

Table 4-26. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Web Degreasing

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**13**	**19**	126	316	632	100
		Central Tendency	**37**	**71**	373	932	1,864	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**5.1**	**7.9**	51	128	255	30
		Central Tendency	**15**	**29**	151	376	753	
Liver - Vessel dilation (JISA 1993)	31	High-End	75	116	754	1,884	3,768	30
		Central Tendency	222	425	2,223	5,557	11,113	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	51	79	510	1,276	2,552	30
		Central Tendency	151	288	1,506	3,764	7,528	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	44	67	438	1,094	2,188	30
		Central Tendency	129	247	1,291	3,226	6,453	

Table 4-27. Risk Estimation for Chronic, Cancer Inhalation Exposures for Web Degreasing

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk liver tumors	2.0E-3	High-End	**3.3E-4**	**2.1E-4**	3.3E-05	1.3E-5	6.6E-6	10^{-4}
		Central Tendency	**1.1E-4**	5.5E-5	1.1E-05	4.2E-6	2.1E-6	

[1] Data from JISA (1993)

4.2.2.11 Cold Cleaning

For cold cleaning, exposure estimates for TWAs of 4 hrs and 8 hrs are available based on personal monitoring data samples, including 34 data points from two sources. EPA supplemented the identified 8-hr TWA exposure monitoring data using modeling with a near-field and far-field approach. For 8-hr TWAs from both monitoring data and modeling, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Due to the limited number of data points for 4-hr TWAs, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively. EPA did not identify monitoring data for ONUs; therefore, EPA used the modeled near-field air concentrations for worker exposures and the modeled far-field air concentrations for potential ONU inhalation exposures from PCE cold cleaning as described in more detail above in Section 2.4.1.14. 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 2.4.1.14 describes the justification for this occupational scenario confidence rating.

Table 4-28. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Cold Cleaning

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure monitoring data								
8-hr	5.0	High-End	**1.2**	EPA did not identify monitoring data for ONUs	12	30	61	10
		Central Tendency	**3.6**		36	89	179	
Based on exposure modeling								
8-hr	5.0	High-End	**3.3**	**6.4**	33	81	163	10
		Central Tendency	2,086	4,029	20,857	52,142	104,284	

[1] Data from Altmann et al. (1990)

8952 **Table 4-29. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Cold Cleaning**

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure monitoring data								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**5.5**	EPA did not identify monitoring data for ONUs	**55**	138	276	100
		Central Tendency	**16**		163	407	813	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**2.2**		**22**	56	111	30
		Central Tendency	**6.6**		66	164	329	
Liver - Vessel dilation (JISA 1993)	31	High-End	33		329	822	1,644	30
		Central Tendency	97		970	2,425	4,849	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	**22**		223	557	1,114	30
		Central Tendency	66		657	1,643	3,285	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**19**		191	477	955	30
		Central Tendency	56		563	1,408	2,816	
Based on exposure modeling								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**15**	**29**	148	371	741	100
		Central Tendency	9,501	18,354	95,007	237,516	475,033	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**6.0**	**12**	60	150	299	30
		Central Tendency	3,837	7,412	38,368	95,920	191,840	
Liver - Vessel dilation (JISA 1993)	31	High-End	88	174	884	2,210	4,420	30
		Central Tendency	56,639	109,419	566,385	1,415,963	2,831,927	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	60	118	599	1497	2,994	30
		Central Tendency	38,368	74,123	383,680	959,201	1,918,402	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	51	101	513	1,283	2,567	30
		Central Tendency	32,887	63,534	328,869	822,172	1,644,345	

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Table 4-30. Risk Estimation for Chronic, Cancer Inhalation Exposures for Cold Cleaning

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10	Worker APF 25	Worker APF 50	
Based on exposure monitoring data								
Cancer Risk liver tumors	2.0E-3	High-End	**9.7E-4**	EPA did not identify monitoring data for ONUs	9.7E-5	3.9E-5	1.9E-5	10^{-4}
		Central Tendency	**2.5E-4**		2.4E-05	1.0E-5	5.1E-6	
Based on exposure modeling								
Cancer Risk liver tumors	2.0E-3	High-End	**2.6E-4**	**1.3E-4**	2.6E-5	1.0E-5	5.2E-6	10^{-4}
		Central Tendency	4.1E-7	2.1E-7	4.1E-8	1.6E-8	8.1E-9	

[1] Data from JISA (1993)

4.2.2.12 Aerosol Degreasing and Aerosol Lubricants

For aerosol degreasing and aerosol lubricants, exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including 197 data points from multiple sources. EPA supplemented the identified exposure monitoring data using modeling with a near-field and far-field approach to estimate 1- and 8-hr TWAs. For both monitoring data and modeling, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA did not identify monitoring data for ONUs; therefore, EPA used the modeled near-field air concentrations for worker exposures and the modeled far-field air concentrations for potential ONU inhalation exposures from PCE aerosol degreasing and aerosol lubricants as described in more detail above in Section 2.4.1.15. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is high. Section 2.4.1.15 describes the justification for this occupational scenario confidence rating

Table 4-31. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Aerosol Degreasing and Aerosol Lubricants

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Based on exposure monitoring data								
8-hr	5.0	High-End	**0.6**	EPA did not identify monitoring data for ONUs	**6.4**	16	32	10
		Central Tendency	**3.5**		35	87	174	
Based on exposure modeling								
8-hr	5.0	High-End	**0.3**	**6.8**	**2.9**	**7.3**	15	10
		Central Tendency	**0.9**	50	**9.1**	23	46	

[1] Data from Altmann et al. (1990)

[2] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-32. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Aerosol Degreasing and Aerosol Lubricants

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[1]	Worker APF 25[1]	Worker APF 50[1]	
Based on exposure monitoring data								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**2.9**	EPA did not identify monitoring data for ONUs	**29**	**73**	146	100
		Central Tendency	**16**		158	396	792	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**1.2**		**12**	**30**	59	30
		Central Tendency	**6.4**		64	160	320	
Liver - Vessel dilation (JISA 1993)	31	High-End	**17**		175	436	873	30
		Central Tendency	94		944	2,360	4,720	
Reproductive - Sperm effects (Beliles et al. 1980**)**	29	High-End	**12**		118	296	591	30
		Central Tendency	64		639	1,599	3,197	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**10**		101	253	507	30
		Central Tendency	55		548	1,370	2,740	
Based on exposure modeling								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**1.3**	**31**	**13**	**33**	**66**	100
		Central Tendency	**4.2**	260	**42**	104	208	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**0.5**	**12**	**5.4**	**13**	**27**	30
		Central Tendency	**1.7**	105	**17**	42	84	
Liver - Vessel dilation (JISA 1993)	31	High-End	**7.9**	182	79	198	395	30
		Central Tendency	**25**	1,550	248	620	1,240	
Reproductive - Sperm effects (Beliles et al. 1980**)**	29	High-End	**5.4**	124	54	134	268	30
		Central Tendency	**17**	1,050	168	420	840	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**4.6**	106	46	115	230	30
		Central Tendency	**14**	900	144	360	720	

[1] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-33. of Risk Estimation for Chronic, Cancer Inhalation Exposures for Aerosol Degreasing and Aerosol Lubricants

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Based on exposure monitoring data								
Cancer Risk liver tumors	2.0E-3	High-End	**1.8E-3**	EPA did not identify monitoring data for ONUs	**1.8E-4**	7.3E-5	3.6E-5	10^{-4}
		Central Tendency	**2.6E-4**		2.6E-5	1.0E-5	5.2E-6	
Based on exposure modeling								
Cancer Risk liver tumors	2.0E-3	High-End	**3.1E-3**	**1.4E-4**	**3.14E-4**	**1.3E-4**	6.3E-5	10^{-4}
		Central Tendency	**9.4E-4**	2.0E-5	9.40E-5	3.8E-5	1.9E-5	

[1] Data from JISA (1993)

[2] EPA does not expect routine use of PPE with this exposure scenario.

4.2.2.13 Dry Cleaning and Spot Cleaning

For dry cleaning, exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including 31 data points from two sources for post-2006 NESHAP data and 124 data points from multiple sources for fourth and fifth generation machine data. EPA supplemented the identified 8-hr TWA exposure monitoring data using modeling with a near-field and far-field approach. For both monitoring data and modeling, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. The lone exception to this is for ONU monitoring data where, due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, for fourth and fifth generation machine data and a single data point for the post-2006 NESHAP data. EPA used both monitoring data and the modeled far-field air concentrations for potential ONU inhalation exposures from PCE dry cleaning as described in more detail above in Section 2.4.1.16. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is high. Section 2.4.1.16 describes the justification for this occupational scenario confidence rating.

Table 4-34. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Dry Cleaning and Spot Cleaning

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
8-hr	5.0	High-End	**0.3**	14[3]	**2.6**	**6.4**	13	10
		Central Tendency	**1.4**		14	34	69	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure modeling								
12-hr	3.3	High-End	**0.1**	**2.1**	**1.1**	**2.8**	**5.6**	10
		Central Tendency	**2.4**	30	24	59	118	
4th/5th Gen Only Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
8-hr	5.0	High-End	**0.9**	41	**8.9**	22	45	10
		Central Tendency	**5.1**	358	51	128	256	

[1] Data from Altmann et al. (1990)
[2] EPA does not expect routine use of PPE with this exposure scenario.
[3] ONU exposure data for Post-2006 Dry Cleaning did not distinguish between central tendency and high-end.

Table 4-35. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Dry Cleaning and Spot Cleaning

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[1]	Worker APF 25[1]	Worker APF 50[1]	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**1.0**	**56**	**10**	**25**	**50**	100
		Central Tendency	**6.1**	**64**	**61**	152	303	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**0.4**	**23**	**4.0**	**10**	**20**	30
		Central Tendency	**2.4**	**26**	**24**	61	122	
Liver - Vessel dilation (JISA 1993)	31	High-End	**5.9**	334	59	148	297	30
		Central Tendency	36	379	361	903	1,806	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**4.0**	226	40	101	201	30
		Central Tendency	24	257	245	612	1,224	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**3.4**	194	86	172	34	30
		Central Tendency	**21**	220	524	1,049	210	

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[1]	Worker APF 25[1]	Worker APF 50[1]	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure modeling								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**0.5**	**9.5**	**5.0**	**12**	**25**	100
		Central Tendency	**11**	136	105	263	527	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**0.2**	**3.8**	**2.0**	**5.0**	**10**	30
		Central Tendency	**4.3**	55	43	106	213	
Liver - Vessel dilation (JISA 1993)	31	High-End	**3.0**	56	30	74	148	30
		Central Tendency	63	809	628	1,569	3,139	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**2.0**	38	**20**	50	100	30
		Central Tendency	43	548	425	1,063	2,126	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**1.7**	33	**17**	43	86	30
		Central Tendency	36	470	365	911	1,823	
4th/5th Gen Only Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**3.5**	158	**35**	**87**	174	100
		Central Tendency	**23**	1,582	226	564	1,129	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**1.4**	64	**14**	35	70	30
		Central Tendency	**9.1**	639	91	228	456	
Liver - Vessel dilation (JISA 1993)	31	High-End	**21**	944	207	518	1,036	30
		Central Tendency	135	9,432	1,346	3,364	6,728	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**14**	639	140	351	702	30
		Central Tendency	91	6,389	912	2,279	4,558	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**12**	548	120	301	602	30
		Central Tendency	78	5,476	781	1,953	3,907	

[1] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-36. of Risk Estimation for Chronic, Cancer Inhalation Exposures for Dry Cleaning and Spot Cleaning

Endpoint, Tumor Types[1]	IUR (risk per mg/m^3)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
Cancer Risk liver tumors	2.0E-3	High-End	**5.4E-3**	9.5E-5	**5.4E-4**	**2.1E-4**	**1.1E-4**	10^{-4}
		Central Tendency	**6.8E-4**	6.5E-5	6.8E-5	2.7E-5	1.4E-5	
Post-2006 Dry Cleaning (including spot cleaning) - Based on exposure modeling								
Cancer Risk liver tumors	2.0E-3	High-End	**8.1E-3**	**4.3E-4**	**8.1E-4**	**3.3E-4**	**1.6E-4**	10^{-4}
		Central Tendency	**3.8E-4**	2.9E-5	3.8E-5	1.5E-5	7.6E-6	
4th/5th Gen Only Dry Cleaning (including spot cleaning) - Based on exposure monitoring data								
Cancer Risk liver tumors	2.0E-3	High-End	**1.5E-3**	3.4E-5	**1.5E-4**	6.1E-5	3.1E-5	10^{-4}
		Central Tendency	**1.8E-4**	2.6E-6	1.8E-5	7.3E-6	3.7E-6	

[1] Data from JISA (1993)
[2] EPA does not expect routine use of PPE with this exposure scenario.

4.2.2.14 Adhesives, Sealants, Paints, and Coatings

For adhesives, sealants, paints, and coatings, exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including 13 data points from one source for adhesives/sealants and 20 data points from multiple sources. For adhesives/sealants, discrete data points were not available; therefore, EPA used the mean and maximum reported in the study to characterize the central tendency and high-end, respectively. For 8-hr TWAs for paints/coatings, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Due to the limited number of data points for 15-min TWAs, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified reasonably available data on potential ONU inhalation exposures from PCE adhesives, sealants, 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 2.4.1.17. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.17 describes the justification for this occupational scenario confidence rating.

Table 4-37. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesives, Sealants, Paints, and Coatings

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
		Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50		
Paints/Coatings								
8-hr	5.0	High-End	**1.1**	21	11	27	55	10
		Central Tendency	21		214	536	1,071	
Adhesives								
8-hr	5.0	High-End	**6.2**	57	62	154	308	10
		Central Tendency	57		565	1,413	2,825	

[1] Data from Altmann et al. (1990)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-38. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Adhesives, Sealants, Paints, and Coatings

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Paints/Coatings								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**5.0**	**98**	**50**	125	250	100
		Central Tendency	**98**		976	2,440	4,881	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**2.0**	39	**20**	50	101	30
		Central Tendency	39		394	986	1,971	
Liver - Vessel dilation (JISA 1993)	31	High-End	30	582	298	744	1,489	30
		Central Tendency	582		5,819	14,548	29,096	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**20**	394	202	504	1,009	30
		Central Tendency	394		3,942	9,855	19,710	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**17**	338	173	432	864	30
		Central Tendency	338		3,379	8,447	16,894	
Adhesives								
CNS -	5.2	High-	**28**	257	281	702	1,404	100

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Visual effects (U.S. EPA 2012c)		End						
		Central Tendency	257		2,574	6,434	12,868	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**11**	104	113	283	567	30
		Central Tendency	104		1,039	2,598	5,197	
Liver - Vessel dilation (JISA 1993)	31	High-End	167	1,534	1,674	4,184	8,369	30
		Central Tendency	1,534		15,343	38,358	76,716	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	113	1,039	1,134	2,835	5,669	30
		Central Tendency	1,039		10,394	25,984	51,969	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	97	891	972	2,430	4,859	30
		Central Tendency	891		8,909	22,272	44,545	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-39. of Risk Estimation for Chronic, Cancer Inhalation Exposures for Adhesives, Sealants, Paints, and Coatings

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
	Paints/Coatings							
Cancer Risk	2.0E-3	High-End	**1.1E-3**	4.2E-5	**1.1E-4**	4.3E-5	2.1E-5	10^{-4}
		Central Tendency	4.2E-5		4.2E-6	1.7E-6	8.5E-7	
	Adhesives							
Cancer Risk	2.0E-3	High-End	**1.9E-4**	1.6E-5	1.9E-5	7.6E-6	3.8E-6	10^{-4}
		Central Tendency	1.6E-5		1.6E-6	6.4E-7	3.2E-7	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.15 Maskant for Chemical Milling

For maskant for chemical milling, exposure estimates for TWAs of 15 mins, 4 hrs, and 8 hrs are available based on personal monitoring data samples, including 53 data points from two sources. EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified reasonably available data on potential ONU inhalation exposures from PCE maskants for chemical milling. 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 2.4.1.18. In lieu of data, EPA uses worker central tendency

values as a surrogate to estimate risks for ONUs. 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 for workers and low for ONUs. Section 2.4.1.18 describes the justification for this occupational scenario confidence rating.

Table 4-40. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Maskant for Chemical Milling

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**2.4**	**4.1**	24	59	119	10
		Central Tendency	**4.1**		41	103	206	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-41. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Maskant for Chemical Milling

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**11**	**19**	108	271	541	100
		Central Tendency	**19**		188	470	939	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**4.4**	**7.6**	44	109	219	30
		Central Tendency	**7.6**		76	190	379	
Liver - Vessel dilation (JISA 1993)	31	High-End	65	112	645	1,614	3,227	30
		Central Tendency	112		1,120	2,800	5,601	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	44	76	437	1,093	2,186	30
		Central Tendency	76		759	1,897	3,794	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	37	65	375	937	1,874	30
		Central Tendency	65		650	1,626	3,252	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-42. Risk Estimation for Chronic, Cancer Inhalation Exposures for Maskant for Chemical Milling

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk	2.0E-3	High-End	**4.9E-4**	**2.2E-4**	4.9E-5	2.0E-5	9.9E-6	10^{-4}
		Central Tendency	**2.2E-4**		2.2E-5	8.8E-6	4.4E-6	

[1] Data from JISA (1993)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.16 Industrial Processing Aid

For industrial processing aid, exposure estimates TWAs of 30 mins and 8 hrs are available based on personal monitoring data samples, including 91 data points from multiple sources. For 8-hr TWAs, EPA calculated 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates for the 30-min TWA. EPA has not identified reasonably available data on potential ONU inhalation exposures from PCE industrial processing aids. 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 2.4.1.19. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.19 describes the justification for this occupational scenario confidence rating.

Table 4-43. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Industrial Processing Aid

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	**4.2**	83	42	106	212	10
		Central Tendency	83		833	2,083	4,167	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-44. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Industrial Processing Aid

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
CNS -	5.2	High-	**19**	380	193	483	965	100

Visual effects (U.S. EPA 2012c)		End						
		Central Tendency	380		3,796	9,490	18,980	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**7.8**	153	78	195	390	30
		Central Tendency	153		1,533	3,833	7,665	
Liver - Vessel dilation (JISA 1993)	31	High-End	115	2,263	1,151	2,877	5,753	30
		Central Tendency	2,263		22,630	56,575	113,150	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	78	1,533	779	1,949	3,897	30
		Central Tendency	1,533		15,330	38,325	76,650	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	67	1,314	668	1,670	3,341	30
		Central Tendency	1,314		13,140	32,850	65,700	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-45. Risk Estimation for Chronic, Cancer Inhalation Exposures for Industrial Processing Aid

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk	2.0E-3	High-End	**2.8E-4**	1.1E-5	2.8E-5	1.1E-5	5.5E-6	10^{-4}
		Central Tendency	1.1E-5		1.1E-6	4.4E-7	2.2E-7	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.17 Metalworking Fluids

For metalworking fluids, exposure estimates for TWAs of 8 hrs are available based on estimates from the Emission Scenario Document (ESD) on the Use of Metalworking Fluids (OECD 2011). EPA uses the geometric mean and 90th percentile as presented in the ESD to characterize the central tendency and high-end exposure estimates, respectively. EPA has not identified reasonably available data on potential ONU inhalation exposures from PCE metalworking fluids. 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 2.4.1.20. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.20 describes the justification for this occupational scenario confidence rating.

Table 4-46. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Metalworking Fluids

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
8-hr	5.0	High-End	239	869	2,387	5,968	11,937	10
		Central Tendency	869		8,692	21,731	43,462	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[3] EPA does not assume routine use of PPE with this exposure scenario.

Table 4-47. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Metalworking Fluids

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	1,087	3,960	10,875	27,187	54,374	100
		Central Tendency	3,960		39,595	98,988	197,976	
Kidney - Histopathology (JISA 1993)	2.1	High-End	439	1,599	4,392	10,979	21,959	30
		Central Tendency	1,599		15,990	39,976	79,952	
Liver - Vessel dilation (JISA 1993)	31	High-End	6,483	23,605	64,830	162,075	324,151	30
		Central Tendency	23,605		236,048	590,121	1,180,242	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	4,392	15,990	43,917	109,793	219,586	30
		Central Tendency	15,990		159,904	399,759	799,518	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	3,764	13,706	37,643	94,108	188,217	30
		Central Tendency	13,706		137,060	342,651	685,302	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[2] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-48 Risk Estimation for Chronic, Cancer Inhalation Exposures for Metalworking Fluids

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
Cancer Risk liver tumors	2.0E-3	High-End	4.9E-6	1.0E-6	4.9E-7	2.0E-7	9.8E-8	10^{-4}
		Central Tendency	1.0E-6		1.0E-7	4.2E-8	2.1E-8	

[1] Data from JISA (1993)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[3] EPA does not expect routine use of PPE with this exposure scenario.

4.2.2.18 Wipe Cleaning and Metal/Stone Polishes

For wipe cleaning and metal/stone polishes, exposure estimates for TWAs of 15 mins, 4 hrs, and 8 hrs are available based on personal monitoring data samples, including 20 data points from two sources. For 8-hr TWAs for ONUs and 15-min TWAs for workers, EPA uses the 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, for worker 8-hr TWAs. The 4-hr TWA estimates are based on a single data point. EPA identified 6 of the 20 data points to be for ONU exposures for wipe cleaning as described in more detail above in Section 2.4.1.21. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.21 describes the justification for this occupational scenario confidence rating.

Table 4-49. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Wipe Cleaning and Metal/Stone Polishes

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
8-hr	5.0	High-End	**2.2E-2**	**0.2**	**0.2**	**0.5**	**1.1**	10
		Central Tendency	**3.8E-2**	229	**0.4**	**0.9**	**1.9**	

[1] Data from Altmann et al. (1990)
[2] EPA does not expect routine use of PPE with this exposure scenario

Table 4-50. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Wipe Cleaning and Metal/Stone Polishes

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator	Worker APF 10[1]	Worker APF 25[1]	Worker APF 50[1]	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**0.1**	**1.0**	**1.0**	**2.5**	**5.0**	100
		Central Tendency	**0.2**	1,043	**1.7**	**4.3**	**8.6**	
Kidney - Histopathology	2.1	High-End	**4.0E-2**	**0.4**	**0.4**	**1.0**	**2.0**	30

(JISA 1993)		Central Tendency	**7.0E-2**	421	**0.7**	**1.7**	**3.5**	
Liver - Vessel dilation (JISA 1993)	31	High-End	**0.6**	**5.9**	**6.0**	**15**	30	30
		Central Tendency	**1.0**	6,220	**10**	**26**	51	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	**0.4**	**4.0**	**4.0**	**10**	**20**	30
		Central Tendency	**0.7**	4,213	**7.0**	**17**	35	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**0.3**	**3.4**	**3.5**	**8.6**	**17**	30
		Central Tendency	**0.6**	3,611	**6.0**	**15**	30	

[1] EPA does not expect routine use of PPE with this exposure scenario

Table 4-51. of Risk Estimation for Chronic, Cancer Inhalation Exposures for Wipe Cleaning and Metal/Stone Polishes

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Cancer Risk	2.0E-3	High-End	**5.3E-2**	**5.4E-3**	**5.3E-3**	**2.1E-3**	**1.1E-3**	10^{-4}
		Central Tendency	**2.4E-2**	4.0E-6	**2.4E-3**	**9.6E-4**	**4.8E-4**	

[1] Data from JISA (1993)
[2] EPA does not expect routine use of PPE with this exposure scenario

4.2.2.19 Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

For other spot cleaning/spot removers (including carpet cleaning), exposure estimates for TWAs of 8 hrs are available based on personal monitoring data samples, including 3 data points from one source. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, for worker 8-hr TWAs. The 8-hr TWA estimates for ONUs are based on a single data point. EPA identified 1 of the 3 data points to be for ONU exposures for other spot cleaning/spot removers (including carpet cleaning) as described in more detail above in Section 2.4.1.22. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium. Section 2.4.1.22 describes the justification for this occupational scenario confidence rating.

Table 4-52. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
8-hr	5.0	High-End	22	167	217	542	1,084	10
		Central Tendency	29		291	727	1,455	

[1] Data from Altmann et al. (1990)
[2] ONU exposure data did not distinguish central tendency and high-end.

[3] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-53. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU[1] No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**99**	759	987	2,468	4,936	100
		Central Tendency	133		1,325	3,313	6,627	
Kidney - Histopathology (JISA 1993)	2.1	High-End	40	307	399	997	1,993	30
		Central Tendency	54		535	1,338	2,676	
Liver - Vessel dilation (JISA 1993)	31	High-End	588	4,526	5,885	14,712	29,424	30
		Central Tendency	790		7,901	19,752	39,504	
Reproductive - Sperm effects (Beliles et al. 1980**)**	21	High-End	399	3,066	3,986	9,966	19,932	30
		Central Tendency	535		5,352	13,381	26,761	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	342	2,628	3,417	8,542	17,085	30
		Central Tendency	459		4,588	11,469	22,938	

[1] ONU exposure data did not distinguish central tendency and high-end
[2] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-54. of Risk Estimation for Chronic, Cancer Inhalation Exposures for Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Cancer Risk	2.0E-3	High-End	5.4E-5	7.0E-6	5.4E-6	2.2E-6	1.1E-6	10^{-4}
		Central Tendency	3.1E-5	5.4E-6	3.1E-6	1.2E-6	6.2E-7	

[1] Data from JISA (1993)
[2] EPA does not expect routine use of PPE with this exposure scenario.

4.2.2.20 Other Industrial Uses

For other industrial uses, exposure estimates for TWAs of 30 mins, 1 hrs, and 8 hrs are available based on modeling. EPA characterized the central tendency exposure estimates assuming unloading/loading of a tank truck and the high-end assuming unloading/loading of a railcar. EPA has not identified reasonably available data on potential ONU inhalation exposures from other industrial uses. 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 2.4.1.23. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation

estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.23 describes the justification for this occupational scenario confidence rating.

Table 4-55. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Other Industrial Uses

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	139	628	1,390	3,475	6,949	10
		Central Tendency	628		6,284	15,710	31,419	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-56. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Other Industrial Uses

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	633	2,862	6,331	15,828	31,656	100
		Central Tendency	2,862		28,624	71,560	143,120	
Kidney - Histopathology (JISA 1993)	2.1	High-End	256	1,156	2,557	6,392	12,784	30
		Central Tendency	1,156		11,560	28,899	57,798	
Liver - Vessel dilation (JISA 1993)	31	High-End	3,774	17,064	37,743	94,358	188,716	30
		Central Tendency	17,064		170,643	426,608	853,216	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	2,557	11,560	25,568	63,920	127,840	30
		Central Tendency	11,560		115,597	288,992	577,985	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	2,192	9,908	21,915	54,788	109,577	30
		Central Tendency	9,908		99,083	247,708	495,416	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-57. Risk Estimation for Chronic, Cancer Inhalation Exposures for Other Industrial Uses

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk	2.0E-3	High-End	8.4E-6	1.4E-6	8.4E-7	3.4E-7	1.7E-7	10^{-4}
		Central Tendency	1.4E-6		1.4E-7	5.8E-8	2.9E-8	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.21 Other Commercial Uses

For other commercial uses, exposure estimates for TWAs of 15 mins and 8 hrs are available based on personal monitoring data samples, including 24 data points for printing applications, 3 data points for photocopying, and 102 data points for photographic film applications. Exposure estimates for mold release products are based on area monitoring data samples, including 4 data points from one source. EPA calculated the 50th and 95th percentiles to characterize the central tendency and high-end exposure estimates, respectively, for 8-hr TWAs for printing applications and 15-min and 8-hr TWAs for photographic film applications. Due to the limited number of data points, EPA used the median and maximum to characterize the central tendency and high-end exposure estimates, respectively, photocopying. The 15-min TWA exposure estimates for printing applications is based on a single data point. For mold release products, discrete data points were not available; therefore, EPA used the mean and maximum reported in the study to characterize the central tendency and high-end, respectively. EPA has not identified reasonably available data on potential ONU inhalation exposures from other commercial uses. 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 2.4.1.24. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. 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 for printing, photographic film, and photocopying workers, medium for mold release workers, and low for ONUs. Section 2.4.1.24 describes the justification for this occupational scenario confidence rating.

Table 4-58. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Other Commercial Uses

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
Printing								
8-hr	5.0	High-End	**0.8**	**2.6**	**8.4**	21	42	10
		Central Tendency	**2.6**		26	65	130	
Photocopying								
8-hr	5.0	High-End	10,000	26,667	100,000	250,000	500,000	10
		Central Tendency	26,667		266,667	666,667	1,333,333	

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
Photographic Film								
8-hr	5.0	High-End	**8.9E-2**	**0.8**	**0.9**	**2.2**	**4.4**	10
		Central Tendency	**0.8**		**7.9**	20	40	
Mold Release								
8-hr	5.0	High-End	25	50	250	625	1,250	10
		Central Tendency	50		500	1,250	2,500	

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[3] EPA does not expect routine use of PPE with this exposure scenario (including all sub-scenarios).

Table 4-59. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Other Commercial Uses

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Printing								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**3.8**	**12**	**38**	**96**	192	100
		Central Tendency	**12**		119	297	594	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**1.5**	**4.8**	**15**	39	77	30
		Central Tendency	**4.8**		48	120	240	
Liver - Vessel dilation (JISA 1993)	31	High-End	**23**	71	228	571	1,142	30
		Central Tendency	71		708	1,770	3,541	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**15**	48	155	387	774	30
		Central Tendency	48		480	1,199	2,399	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**13**	41	133	332	663	30
		Central Tendency	41		411	1,028	2,056	
Photocopying								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	45,552	121,472	455,520	1,138,800	2,277,600	100
		Central Tendency	121,472		1,214,720	3,036,800	6,073,600	
Kidney - Histopathology	2.1	High-End	18,396	49,056	183,960	459,900	919,800	30

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
(JISA 1993)		Central Tendency	49,056		490,560	1,226,400	2,452,800	
Liver - Vessel dilation (JISA 1993)	31	High-End	271,560	724,160	2,715,600	6,789,000	13,578,000	30
		Central Tendency	724,160		7,241,600	18,104,000	36,208,000	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	183,960	490,560	1,839,600	4,599,000	9,198,000	30
		Central Tendency	490,560		4,905,600	12,264,000	24,528,000	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	157,680	420,480	1,576,800	3,942,000	7,884,000	30
		Central Tendency	420,480		4,204,800	10,512,000	21,024,000	
Photographic Film								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**0.4**	**3.6**	**4.0**	**10**	**20**	100
		Central Tendency	**3.6**		**36**	**90**	181	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**0.2**	**1.5**	**1.6**	**4.1**	**8.2**	30
		Central Tendency	**1.5**		**15**	37	73	
Liver - Vessel dilation (JISA 1993)	31	High-End	**2.4**	**22**	**24**	60	120	30
		Central Tendency	**22**		216	539	1,079	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	**1.6**	**15**	**16**	41	82	30
		Central Tendency	**15**		146	365	731	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	**1.4**	**13**	**14**	35	70	30
		Central Tendency	**13**		125	313	626	
Mold Release								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	114	228	1,139	2,847	5,694	100
		Central Tendency	228		2,278	5,694	11,388	
Kidney - Histopathology (JISA 1993)	2.1	High-End	46	92	460	1,150	2,300	30
		Central Tendency	92		920	2,300	4,599	
Liver - Vessel dilation (JISA 1993)	31	High-End	679	1,358	6,789	16,973	33,945	30
		Central Tendency	1,358		13,578	33,945	67,890	

Endpoint	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10[2]	Worker APF 25[2]	Worker APF 50[2]	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	460	920	4,599	11,498	22,995	30
		Central Tendency	920		9,198	22,995	45,990	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	394	788	3,942	9,855	19,710	30
		Central Tendency	788		7,884	19,710	39,420	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[2] EPA does not expect routine use of PPE with this exposure scenario (including all sub-scenarios).

Table 4-60. Risk Estimation for Chronic, Cancer Inhalation Exposures for Other Commercial Uses

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10[3]	Worker APF 25[3]	Worker APF 50[3]	
Printing								
Cancer Risk	2.0E-3	High-End	**1.4E-3**	**3.5E-4**	**1.4E-4**	5.6E-5	2.8E-5	10^{-4}
		Central Tendency	**3.5E-4**		3.5E-5	1.4E-5	7.0E-6	
Photocopying								
Cancer Risk	02.0E-3	High-End	1.2E-7	3.4E-8	1.2E-8	4.7E-9	2.3E-9	10^{-4}
		Central Tendency	3.4E-8		3.4E-9	1.4E-9	6.8E-10	
Photographic Film								
Cancer Risk	2.0E-3	High-End	**1.3E-2**	**1.1E-3**	**1.3E-3**	**5.3E-4**	**2.6E-4**	10^{-4}
		Central Tendency	**1.1E-3**		**1.1E-4**	4.6E-5	2.3E-5	
Mold Release								
Cancer Risk	2.0E-3	High-End	4.7E-5	1.8E-5	4.7E-6	1.9E-6	9.4E-7	10^{-4}
		Central Tendency	1.8E-5		1.8E-6	7.3E-7	3.6E-7	

[1] Data from JISA (1993)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[3] EPA does not expect routine use of PPE with this exposure scenario (including all sub-scenarios).

4.2.2.22 Laboratory Chemicals

EPA does not have data to assess worker exposures to PCE during laboratory use. However, due to the expected safety practices when using chemicals in a laboratory setting, PCE is expected to be applied in small amounts under a fume hood, thus reducing the potential for inhalation exposures.

4.2.2.23 Waste Handling, Disposal, Treatment, and Recycling

For waste handling, disposal, treatment, and recycling, exposure estimates for TWAs of 30 mins, 1 hrs, and 8 hrs are available based on modeling. EPA characterized the central tendency exposure estimates assuming unloading/loading of a tank truck and the high-end assuming unloading/loading of a railcar. EPA has not identified reasonably available data on potential ONU inhalation exposures from 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 2.4.1.26. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is medium for workers and low for ONUs. Section 2.4.1.26 describes the justification for this occupational scenario confidence rating.

Table 4-61. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
8-hr	5.0	High-End	139	628	1,390	3,475	6,949	10
		Central Tendency	628		6,284	15,710	31,419	

[1] Data from Altmann et al. (1990)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-62. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

Endpoint[1]	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	633	2,862	6,331	15,828	31,656	100
		Central Tendency	2,862		28,624	71,560	143,120	
Kidney - Histopathology (JISA 1993)	2.1	High-End	256	1,156	2,557	6,392	12,784	30
		Central Tendency	1,156		11,560	28,899	57,798	
Liver - Vessel dilation (JISA 1993)	31	High-End	3,774	17,064	37,743	94,358	188,716	30
		Central Tendency	17,064		170,643	426,608	853,216	
	21	High-	3,531	15,963	35,308	88,270	176,540	30

Endpoint[1]	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Reproductive - Sperm effects (Beliles et al. 1980)		End						
		Central Tendency	15,963		159,634	399,085	798,170	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	2,557	11,560	25,568	63,920	127,840	30
		Central Tendency	11,560		115,597	288,992	577,985	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-63. Risk Estimation for Chronic, Cancer Inhalation Exposures for Waste Handling, Disposal, Treatment, and Recycling

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Cancer Risk	2.0E-3	High-End	8.4E-6	1.4E-6	8.4E-7	3.4E-7	1.7E-7	10^{-4}
		Central Tendency	1.4E-6		1.4E-7	5.8E-8	2.9E-8	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.2.24 Other Department of Defense Uses

For other department of defense uses, exposure estimates TWAs of 15 mins, 1 hr, and 8 hrs are available based on personal monitoring data samples, including 4 data points from multiple sources. For the oil analysis results exposure results are based on a single data point (one for each TWA duration). For the water pipe repair, only one data point was available that measured below the LOD; therefore, EPA characterized the central tendency and high-end exposures as half the LOD and the LOD, respectively. EPA has not identified reasonably available data on potential ONU inhalation exposures from other department of defense uses. 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 2.4.1.27. In lieu of data, EPA uses worker central tendency values as a surrogate to estimate risks for ONUs. Considering the overall strengths and limitations of the data, EPA's overall confidence in the occupational inhalation estimates in this scenario is high for workers and low for ONUs. Section 2.4.1.27 describes the justification for this occupational scenario confidence rating.

Table 4-64. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Other Department of Defense Uses

HEC Time Period Endpoint = CNS Effects[1]	Acute HEC (ppm)	Exposure Level	MOEs for Acute Exposures					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
Water Pipe Repair								
8-hr	5.0	High-End	**2.2**	**4.3**	22	54	108	10
		Central Tendency	**4.3**		43	108	216	
Oil Analysis[3]								
8-hr	5.0	High-End	**5.7**	**5.7**	57	142	284	10
		Central Tendency						

[1] Data from Altmann et al. (1990)
[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.
[3] Oil analysis exposure data did not distinguish between central tendency and high-end.

Table 4-65. Risk Estimation for Chronic, Non-Cancer Inhalation Exposures for Other Department of Defense Uses

Endpoint[1]	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Water Pipe Repair								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**68**	164	684	1,710	3,420	100
		Central Tendency	164		1,642	4,104	8,208	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**28**	66	276	691	1,381	30
		Central Tendency	66		663	1,657	3,315	
Liver - Vessel dilation (JISA 1993)	31	High-End	408	979	4,077	10,194	20,387	30
		Central Tendency	979		9,786	24,465	48,930	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	276	633	2,762	6,905	13,811	30
		Central Tendency	663		6,629	16,573	33,146	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	237	568	2,368	5,919	11,838	30
		Central Tendency	568		5,682	14,205	28,411	
Oil Analysis								
CNS - Visual effects (U.S. EPA 2012c)	5.2	High-End	**43**	**52**	431	1,077	2,154	100
		Central Tendency	**52**		517	1,293	2,585	

Endpoint[1]	Chronic HEC (ppm)	Exposure Level	MOEs for Chronic Exposure					Benchmark MOE (= Total UF)
			Worker No respirator	ONU No respirator[1]	Worker APF 10	Worker APF 25	Worker APF 50	
Kidney - Histopathology (JISA 1993)	2.1	High-End	**17**	**21**	174	435	870	30
		Central Tendency	**21**		209	522	1,044	
Liver - Vessel dilation (JISA 1993)	31	High-End	257	308	2,569	6,422	12,843	30
		Central Tendency	308		3,082	7,706	15,412	
Reproductive - Sperm effects (Beliles et al. 1980)	21	High-End	240	288	2,403	6,007	12,014	30
		Central Tendency	288		2,883	7,209	14,417	
Developmental - Mortality/ CNS effects (Tinston 1994)	18	High-End	174	209	1740	4350	8700	30
		Central Tendency	209		2088	5220	10440	

[1] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

Table 4-66. Risk Estimation for Chronic, Cancer Inhalation Exposures for Other Department of Defense Uses

Endpoint, Tumor Types[1]	IUR (risk per ppm)	Exposure Level	Cancer Risk Estimates					Benchmark
			Worker No respirator	ONU No respirator[2]	Worker APF 10	Worker APF 25	Worker APF 50	
		Water Pipe Repair						
Cancer Risk	2.0E-3	High-End	7.8E-05	2.5E-05	7.8E-06	3.1E-6	1.6E-6	10^{-4}
		Central Tendency	2.5E-05		2.5E-06	1.0E-6	5.0E-7	
		Oil Analysis						
Cancer Risk	2.0E-3	High-End	**1.2E-04**	8.0E-05	1.2E-05	5.0E-6	2.5E-6	10^{-4}
		Central Tendency	8.0E-05		8.0E-06	3.2E-6	1.6E-6	

[1] Data from JISA (1993)

[2] EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown.

4.2.3 Risk Estimation for Dermal Exposures to Workers

To assess dermal exposure, EPA used the *Dermal Exposure to Volatile Liquids Model* (see Section 2.4.1.5) to calculate the dermal retained dose. EPA "binned" exposure scenarios based on likely level of exposure. Overall, EPA has a medium level of confidence in the assessed baseline exposure. The hazard HEDs are summarized in Table 3-7,

Table 3-8 and Table 3-9. From among all chronic studies, EPA selected the most robust studies and non-cancer PODs from within each health domain to serve as representative endpoints for risk estimation (Section 3.2.5.4). These representative PODs are presented below in Table 4-2 along with the single acute POD. Dermal PODs were calculated as extrapolated from both inhalation and oral POD values,

when possible (Section 3.2.5.4.1 and Table 3-10). When extrapolation was available via both routes, the more sensitive POD was selected in order to be health-protective given the relative similarity in magnitude of uncertainties via either route. Of note, in all cases the difference in the derived dermal POD between routes is no more than approximately 2-fold. The dermal POD value to be used for risk estimates is bold in the table below. Non-cancer risk estimates were calculated with equation 4-1 and cancer risks were calculated with equation 4-2.

Table 4-67. Selected Non-cancer PODs for Use in Risk Estimation of Dermal Exposures

Target Organ System and Effect	Inhalation POD and Duration	Inhalation to Dermal Adjustments	Inhalation to Dermal HED (mg/kg-day)	Oral to Dermal HED (mg/kg-day)	Total Uncertainty Factor (UF) for Benchmark MOE	Reference	Data Quality
ACUTE EXPOSURE							
CNS Neurotoxicity increased latencies for pattern reversal visual-evoked potentials	10 ppm (68 mg/m^3) 4 hrs/day	1.25 m^3/hr 4 hrs/day 80 kg BW	**4.25**	N/A	UF_A=1; UF_H=10; UF_L=1 **Total UF=10**	Altmann et al. (1990)	Medium
CHRONIC EXPOSURE							
Midpoint of the range of the two neuorotoxicity endpoints	5.2 ppm (36 mg/m^3)	20 m^3/day 80 kg BW	9.0	**6.2**	UF_A=1; UF_H=10; UF_L=10 **Total UF=100**	Based on U.S. EPA (2012c)	Medium
Kidney Nuclear enlargement in proximal tubules	2.1 ppm (14 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	3.5	**2.2**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (, 1993, 630653)	High
Liver Increased angiectasis in liver	31 ppm (210 mg/m^3) 24 hrs/day	20 m^3/day 80 kg BW	52.5	**24.5**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	JISA (1993)	High
Developmental Reduced sperm quality following 5 days exposure	21 ppm (140 mg/m^3)	20 m^3/day 80 kg BW	35	**22**	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Beliles et al. (1980)	High
Developmental Increased F_{2A} pup deaths by Day 29, CNS depression in F_1 and F_2	18 ppm (122 mg/m^3)	20 m^3/day 80 kg BW	**31**	N/A	UF_A=3; UF_H=10; UF_L=1 **Total UF=30**	Tinston et al. (1994)	High
CANCER							
male mouse hepatocellular tumors	3×10^{-4} per mg/m^3	20 m^3/day 80 kg BW	1×10^{-3} per mg/kg/day	**2×10^{-3} per mg/kg/day**	Not applicable	JISA (1993)	High

4.2.3.1 Industrial Uses That Generally Occur in Closed Systems

For these uses, dermal exposure is likely limited to chemical loading/unloading activities (e.g. connecting hoses) and taking quality control samples. The exposure scenarios include:

- Manufacture
- Import/Repackaging
- Processing as a Reactant
- Incorporation into Formulation, Mixture, or Reaction Product
- Industrial Processing Aid
- Other Industrial Uses
- Waste Handling, Disposal, Treatment, and Recycling

Table 4-68. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Industrial Uses That Generally Occur in Closed Systems

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**1.2**	**6.0**	12	24	10
		Central Tendency	**3.6**	18	36	72	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

Table 4-69. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Industrial Uses That Generally Occur in Closed Systems

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**2.6**	**13**	**26**	**51**	100
		Central Tendency	**7.7**	**38**	**77**	154	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**0.9**	**4.6**	**9.1**	**18**	30
		Central Tendency	**2.7**	**14**	**27**	55	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**10**	51	101	203	30
		Central Tendency	30	152	304	608	
Reproductive - Sperm effects (Beliles et al. 1980**)**	22	High-End	**9.1**	45	91	182	30
		Central Tendency	**27**	136	273	546	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**13**	64	128	256	30
		Central Tendency	**38**	192	384	769	

Table 4-70. Risk Estimation for Chronic, Cancer Dermal Exposures for Industrial Uses That Generally Occur in Closed Systems

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Cancer Risk liver tumors	2.0E-3	High-End	**2.5E-3**	**5.0E-4**	**2.5E-4**	**1.2E-4**	10^{-4}
		Central Tendency	**6.4E-4**	**1.3E-4**	6.4E-5	3.2E-5	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)

4.2.3.2 Industrial Degreasing and Chemical Maskant Uses Which Are Not Closed Systems

For these uses, there is greater opportunity for dermal exposure during activities such as charging and draining degreasing/milling equipment, drumming waste solvent, handling recycled/re-captured maskants, and removing waste sludge. The exposure scenarios include:

- Batch Open-Top Vapor Degreasing
- Batch Closed-Loop Vapor Degreasing
- Conveyorized Vapor Degreasing
- Web Degreasing
- Cold Cleaning
- Maskant for Chemical Milling

Table 4-71. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Industrial Degreasing and Chemical Maskant Uses Which Are Not Closed Systems

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**1.2**	**6.0**	12	24	10
		Central Tendency	**3.6**	18	36	72	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

Table 4-72. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Industrial Degreasing and Chemical Maskant Uses Which Are Not Closed Systems

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**2.6**	**13**	**26**	**51**	100
		Central Tendency	**7.7**	**38**	**77**	154	
Kidney - Histopathology	2.2	High-End	**0.9**	**4.5**	**9.1**	**18**	30

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
(JISA 1993)		Central Tendency	**2.7**	**14**	**27**	55	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**10**	51	101	203	30
		Central Tendency	30	152	304	608	
Reproductive - Sperm effects (Beliles et al. 1980)	22	High-End	**9.1**	45	91	182	30
		Central Tendency	**27**	136	273	546	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**13**	64	128	256	30
		Central Tendency	**38**	192	384	769	

Table 4-73. Risk Estimation for Chronic, Cancer Dermal Exposures for Industrial Degreasing and Chemical Maskant Uses Which Are Not Closed Systems

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Cancer Risk liver tumors	2.0E-3	High-End	**2.5E-3**	**5.0E-4**	**2.5E-4**	**1.2E-4**	10^{-4}
		Central Tendency	**6.4E-4**	**1.3E-4**	6.4E-5	3.2E-5	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)

4.2.3.3 Aerosol Uses

For these uses, workers are likely to have direct dermal contact with film applied to substrate and incidental deposition of aerosol to skin. The exposure scenario is specific to aerosol degreasing and aerosol lubricants. EPA does not expect routine use of dermal PPE with this exposure scenario for commercial use.

Table 4-74. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Aerosol Uses

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5[3]	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**0.8**	**4.0**	**8.0**	16	10
		Central Tendency	**2.4**	12	24	48	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

Table 4-75. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Aerosol Uses

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**1.7**	**8.6**	**17**	**34**	100
		Central Tendency	**5.1**	**26**	**51**	103	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**0.6**	**3.0**	**6.1**	**12**	30
		Central Tendency	**1.8**	**9.1**	**18**	36	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**6.8**	34	68	135	30
		Central Tendency	**20**	101	203	406	
Reproductive - Sperm effects (Beliles et al. 1980**)**	22	High-End	**6.1**	30	61	121	30
		Central Tendency	**18**	91	182	364	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**8.6**	43	86	171	30
		Central Tendency	**26**	128	257	513	

Table 4-76. Risk Estimation for Chronic, Cancer Dermal Exposures for Aerosol Uses

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Cancer Risk liver tumors	2.0E-3	High-End	**3.7E-3**	**7.4E-4**	**3.7E-4**	**1.9E-4**	10^{-4}
		Central Tendency	**9.6E-4**	**1.9E-4**	9.6E-5	4.8E-5	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)

4.2.3.4 Commercial Activities of Similar Maximum Concentration

Most of these uses are uses with concentrations up to 100% PCE and occur at dry cleaners, and/or uses expected to have direct dermal contact with bulk liquids. At dry cleaning shops, workers may be exposed to bulk liquids while charging and draining solvent to/from machines, removing and disposing sludge, and maintaining equipment. Workers can also be exposed to PCE used in spot cleaning products at the same shop. The exposure scenarios include:

- Dry Cleaning and Spot Cleaning
- Wipe Cleaning and Metal/Stone Polishes
- Other Spot Cleaning/Spot Remover
- Other Commercial Uses

EPA does not expect routine use of dermal PPE with these exposure scenarios for commercial use.

Table 4-77. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Commercial Activities of Similar Maximum Concentration

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5[2]	Worker PF 10[2]	Worker PF 20[2]	
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**0.8**	**3.9**	**7.9**	16	10
		Central Tendency	**2.4**	12	24	47	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

[2] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-78. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Commercial Activities of Similar Maximum Concentration

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5[1]	Worker PF 10[1]	Worker PF 20[1]	
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**1.7**	**8.4**	**17**	**34**	100
		Central Tendency	**5.0**	**25**	**50**	101	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**0.6**	**3.0**	**6.0**	**12**	30
		Central Tendency	**1.8**	**8.9**	**18**	36	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**6.6**	33	66	133	300
		Central Tendency	**20**	99	199	398	
Reproductive - Sperm effects (Beliles et al. 1980)	22	High-End	**6.0**	30	60	119	30
		Central Tendency	**18**	89	179	357	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**8.4**	42	84	168	30
		Central Tendency	**25**	126	252	503	

[1] EPA does not expect routine use of PPE with this exposure scenario.

Table 4-79. Risk Estimation for Chronic, Cancer Dermal Exposures for Commercial Activities of Similar Maximum Concentration

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5[2]	Worker PF 10[2]	Worker PF 20[2]	
Cancer Risk liver tumors	2.0E-3	High-End	**3.8E-3**	**7.6E-4**	**3.8E-4**	**1.9E-4**	10^{-4}
		Central Tendency	**9.8E-4**	**2.0E-4**	9.8E-5	4.9E-5	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)
[2] EPA does not expect routine use of PPE with this exposure scenario.

4.2.3.5 Metalworking Fluids

These product formulations are expected to be used in industrial settings and workers may be exposed when unloading the metalworking fluid from containers; transferring fluids to the trough; and performing metal shaping operations. The exposure scenario is specific to metalworking fluids.

Table 4-80. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Metalworking Fluids

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	12	60	120	241	10
		Central Tendency	36	181	361	722	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

Table 4-81. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Metalworking Fluids

Endpoint	Chronic HED (mg/kg/ day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**26**	128	256	513	100
		Central Tendency	**77**	384	769	1,538	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**9.1**	45	91	182	30
		Central Tendency	**27**	136	273	546	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	101	506	1,013	2,026	30
		Central Tendency	304	1,519	3,039	6,077	
Reproductive - Sperm effects (Beliles et al. 1980**)**	22	High-End	91	455	910	1819	30
		Central Tendency	273	1364	2729	5457	
Developmental - Mortality/	31	High-End	128	641	1282	2563	30

Endpoint	Chronic HED (mg/kg/ day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
CNS effects (Tinston 1994)		Central Tendency	384	1922	3845	7690	

Table 4-82. Risk Estimation for Chronic, Cancer Dermal Exposures for Metalworking Fluids

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Cancer Risk liver tumors	2.0E-3	High-End	**2.5E-4**	5.0E-5	2.5E-5	1.2E-5	10^{-4}
		Central Tendency	6.4E-5	1.3E-5	6.4E-6	3.2E-6	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)

4.2.3.6 Adhesives, Sealants, Paints, and Coatings

These product formulations may have both industrial and commercial uses and workers may be exposed when mixing coating/adhesive, charging products to application equipment (e.g., spray guns, roll applicators, etc.), and cleaning application equipment. Other workers may also have incidental contact with applied products during subsequent fabrication steps. The exposure scenario is specific to adhesives, sealants, paints, and coatings.

Table 4-83. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Adhesives, Sealants, Paints, and Coatings

Endpoint [1]	Acute HED (mg/kg/day)	Exposure Level	MOEs for Acute Exposures				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Commercial Uses							
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**1.0**	**4.9**	**9.8**	20	10
		Central Tendency	**3.0**	15	30	59	
Industrial Uses							
CNS - Visual effects (U.S. EPA 2012c)	4.3	High-End	**1.5**	**7.5**	15	30	10
		Central Tendency	**4.5**	23	45	90	

[1] Based on route to route extrapolation from inhalation exposure data from Altmann et al. (1990) see Table 3-7

Table 4-84. Risk Estimation for Chronic, Non-Cancer Dermal Exposures for Adhesives, Sealants, Paints, and Coatings

Endpoint	Chronic HED (mg/kg/day)	Exposure Level	MOEs for Chronic Exposure				Benchmark MOE (= Total UF)
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Commercial Uses							
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**2.1**	**10**	**21**	**42**	100
		Central Tendency	**6.3**	**31**	**63**	126	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**0.7**	**3.7**	**7.4**	**15**	30
		Central Tendency	**2.2**	**11**	**22**	45	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**8.3**	41	83	166	30
		Central Tendency	**25**	124	248	497	
Reproductive - Sperm effects (Beliles et al. 1980**)**	22	High-End	**7.4**	37	74	149	30
		Central Tendency	**22**	112	223	446	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**10**	52	105	210	30
		Central Tendency	**31**	157	314	629	
Industrial Uses							
CNS - Visual effects (U.S. EPA 2012c)	6.2	High-End	**3.2**	**16**	**32**	**64**	100
		Central Tendency	**9.6**	**48**	**96**	192	
Kidney - Histopathology (JISA 1993)	2.2	High-End	**1.1**	**5.7**	**11**	**23**	30
		Central Tendency	**3.4**	**17**	34	68	
Liver - Vessel dilation (JISA 1993)	24.5	High-End	**13**	63	127	253	30
		Central Tendency	38	190	380	760	
Reproductive - Sperm effects (Beliles et al. 1980**)**	22	High-End	**11**	57	114	227	30
		Central Tendency	34	171	341	682	
Developmental - Mortality/ CNS effects (Tinston 1994)	31	High-End	**16**	80	160	320	30
		Central Tendency	48	240	481	961	

Table 4-85. Risk Estimation for Chronic, Cancer Dermal Exposures for Adhesives, Sealants, Paints, and Coatings

Endpoint, Tumor Types[1]	Dermal slope factor (risk per mg/kg/day)	Exposure Level	Cancer Risk Estimates				Benchmark
			Worker No gloves	Worker PF 5	Worker PF 10	Worker PF 20	
Commercial Uses							
Cancer Risk liver tumors	2.0E-3	High-End	**3.0E-3**	**6.1E-4**	**3.0E-4**	**1.5E-4**	10^{-4}
		Central Tendency	**7.8E-4**	**1.6E-4**	7.8E-5	3.9E-5	
Industrial Uses							
Cancer Risk liver tumors	2.0E-3	High-End	**2.0E-3**	**4.0E-4**	**2.0E-4**	9.9E-5	10^{-4}
		Central Tendency	**5.1E-4**	1.0E-4	5.1E-5	2.6E-5	

[1] Based on route to route extrapolation from the oral slope factor using data from JISA (1993)

4.2.4 Risk Estimation for Exposures to Consumers

Risk estimates for consumers were calculated for consumers for acute inhalation and dermal exposures. Risk estimates for chronic exposures were not calculated because it is unknown how the available toxicological data relates to the human exposures expected in consumer exposure scenarios. The toxicity studies are based on human worker studies or continuous subchronic-to-chronic repeated dose animal studies. In contrast, the consumer exposure scenarios are expected to be intermittent and it is unlikely that the expected use patterns would cumulatively be equivalent to these scenarios. It therefore cannot be ruled out whether there is any risk for chronic non-cancer or cancer associated with regular, intermittent exposures at the very high end of use frequency, however this scenario cannot be adequately evaluated and is unlikely to apply to the vast majority of users.

Risk estimates were presented for differing acute exposure assumptions, categorized as high, moderate, or low intensity users based on variation in weight fraction, mass of product used, and duration of use/exposure duration. Risk estimates primarily utilized central tendency values for other modeling parameters (e.g., room volume, air exchange rate, building volume) and therefore do not necessarily represent an upper bound of possible exposures. For more details on the characterization of consumer exposure see Section 2.4.2.2. For MOE estimates of all modeled scenarios see supplemental files: *Draft Risk Evaluation for Perchloroethylene Consumer Inhalation Risk Calculations* (U.S. EPA 2020c) and *Draft Risk Evaluation for Perchloroethylene Consumer Dermal Risk Calculations* (U.S. EPA 2020b). The HEC (Table 3-7) and HED values (Table 3-10) for neurotoxicity from (Altmann et al. 1990) was used for estimating of all acute consumer risks.

4.2.4.1 Aerosol Cleaners for Motors, Coils, Electrical Parts, Cables, Stainless Steel and marine Equipment, and Wire and Ignition Demoisturants

Estimates of MOEs for acute inhalation and dermal exposures for the aerosol cleaners for motors, coils and electrical parts, etc. consumer use are presented in Table 4-86 and Table 4-87, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.1.1.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.1.1.

Table 4-86. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Aerosol Cleaners for Motors Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	**7.7**	39
Moderate Intensity User	**0.2**	**0.8**
High Intensity User	**1.3E-02**	**5.2E-02**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-87. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Aerosol Cleaners for Motors Consumer Use

Exposure Scenario	Consumer Receptor	`
		User MOE
Low Intensity User	Adult (≥21 years)	35
	Youth (16-20 years)	38
	Youth (11-15 years)	35
Moderate Intensity User	Adult (≥21 years)	**0.6**
	Youth (16-20 years)	**0.6**
	Youth (11-15 years)	**0.6**
High Intensity User	Adult (≥21 years)	**5.9E-02**
	Youth (16-20 years)	**6.3E-02**
	Youth (11-15 years)	**5.8E-02**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for high and moderate intensity users and bystanders by inhalation and dermal exposures. The MOEs are below the benchmark MOE for the low intensity user by inhalation not dermal exposure and not for the low-intensity bystander.

4.2.4.2 Aerosol Brake Cleaners

Estimates of MOEs for acute inhalation and dermal exposures for the aerosol brake cleaners consumer use are presented in Table 4-88 and Table 4-89, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.1.2.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.1.2.

Table 4-88. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Aerosol Brake Cleaners Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	**2.0**	**7.1**
Moderate Intensity User	**0.2**	**0.8**
High Intensity User	**4.5E-02**	**0.2**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-89. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Aerosol Brake Cleaner Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	22
	Youth (16-20 years)	23
	Youth (11-15 years)	21
Moderate Intensity User	Adult (≥21 years)	**0.6**
	Youth (16-20 years)	**0.7**
	Youth (11-15 years)	**0.6**
High Intensity User	Adult (≥21 years)	**7.2E-02**
	Youth (16-20 years)	**7.7E-02**
	Youth (11-15 years)	**7.1E-02**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for all users and bystanders by inhalation exposures. The MOEs are below the benchmark MOE for the high and Moderate Intensity Users by dermal exposure and not for low intensity dermal exposures.

4.2.4.3 Parts Cleaners

Estimates of MOEs for acute inhalation and dermal exposures for the immersive parts cleaner consumer use are presented in Table 4-90 and Table 4-91, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.2.

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.2.

Table 4-90. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Parts Cleaners Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	31	174
Moderate Intensity User	**0.6**	**3.3**
High Intensity User	**7.1E-02**	**0.4**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-91. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Parts Cleaners Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	**0.2**
	Youth (16-20 years)	**0.2**
	Youth (11-15 years)	**0.2**
Moderate Intensity User	Adult (≥21 years)	**1.4E-02**
	Youth (16-20 years)	**1.4E-02**
	Youth (11-15 years)	**1.3E-02**
High Intensity User	Adult (≥21 years)	**2.4E-03**
	Youth (16-20 years)	**2.3E-03**
	Youth (11-15 years)	**2.1E-03**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for high and moderate intensity users and bystanders by inhalation exposures and not for low intensity inhalation exposures. The MOEs are below the benchmark MOE for all users by dermal exposure.

4.2.4.4 Vandalism Stain Removers, Mold Cleaners, and Weld Splatter Protectants

Estimates of MOEs for acute inhalation exposures for the vandalism stain removers, mold cleaners, and weld splatter protectants consumer use are presented in Table 4-92. Dermal exposures to consumers are

not expected for vandalism stain removers, mold cleaners, and weld splatter protectants as described in Section 2.4.2.3.3. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.3.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.3.

Table 4-92. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Vandalism Stain Removers, Mold Cleaners, and Weld Splatter Protectants Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	15	77
Moderate Intensity User	**0.3**	**1.6**
High Intensity User	**1.3E-02**	**5.2E-02**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high and moderate intensity users and bystanders by inhalation exposures and not for low intensity inhalation exposures.

4.2.4.5 Marble Polish

Estimates of MOEs for acute inhalation and dermal exposures for the liquid-based marble polish consumer use 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 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.4.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.4.

Table 4-93. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Liquid-Based Marble Polish Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	**3.3**	17

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Moderate Intensity User	**6.8E-02**	**0.4**
High Intensity User	**1.2E-02**	**5.0E-02**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-94. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Liquid-Based Marble Polish Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	**3.5**
	Youth (16-20 years)	**3.8**
	Youth (11-15 years)	**3.5**
Moderate Intensity User	Adult (≥21 years)	**5.5E-02**
	Youth (16-20 years)	**5.9E-02**
	Youth (11-15 years)	**5.4E-02**
High Intensity User	Adult (≥21 years)	**5.8E-03**
	Youth (16-20 years)	**6.3E-03**
	Youth (11-15 years)	**5.8E-03**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for high and Moderate Intensity Users and bystanders by inhalation exposures and not for low intensity inhalation exposures. The MOEs are below the benchmark MOE for all users by dermal exposures.

4.2.4.6 Cutting Fluid

Estimates of MOEs for acute inhalation exposures for the cutting fluid consumer use are presented in Table 4-95. Dermal exposures for cutting fluid consumer use are not expected as described in Section 2.4.2.3.5. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.5.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.5.

Table 4-95. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Cutting Fluid Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10

	User MOE	Bystander MOE
Low Intensity User	**8.1**	39
Moderate Intensity User	**1.3**	**6.7**
High Intensity User	**0.1**	**0.6**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for all users and high and moderate intensity bystanders by inhalation exposures and not for low intensity bystanders.

4.2.4.7 Lubricants and Penetrating Oils

Estimates of MOEs for acute inhalation exposures for the lubricants and penetrating oils consumer use are presented in Table 4-96. Dermal exposures for the lubricants and penetrating oils consumer use are not expected as described in Section 2.4.2.3.6. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.6

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.6.

Table 4-96. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Lubricants and Penetrating Oils Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	90	435
Moderate Intensity User	**1.4**	**7.3**
High Intensity User	**8.0E-02**	**0.4**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high and moderate intensity users and bystanders by inhalation exposures and not for low intensity users and bystanders.

4.2.4.8 Adhesives

Estimates of MOEs for acute inhalation exposures for the adhesives consumer use are presented in Table 4-97. Dermal exposures for the adhesives consumer use are not expected as described in Section 2.4.2.3.7. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.7

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.7.

Table 4-97. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Adhesives Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	62	299
Moderate Intensity User	**2.3**	12
High Intensity User	**0.1**	**0.5**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high and moderate intensity users and high intensity bystanders by inhalation exposures and not for low intensity users and medium and low intensity bystanders.

4.2.4.9 Livestock Grooming Adhesive

Estimates of MOEs for acute inhalation exposures for the livestock grooming adhesive consumer use are presented in Table 4-98. Dermal exposures for the livestock grooming adhesive consumer use are not expected as described in Section 2.4.2.3.8. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.8

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.8.

Table 4-98. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Livestock Grooming Adhesives Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	112	539
Moderate Intensity User	12	64
High Intensity User	**0.8**	**3.0**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high intensity users and bystanders by inhalation exposures and not for medium and low intensity users and bystanders.

4.2.4.10 Caulks, Sealants and Column Adhesives

Estimates of MOEs for acute inhalation exposures for the caulks, sealants and column adhesives consumer use are presented in Table 4-99. Dermal exposures for the caulks, sealants and column adhesives consumer use are not expected and the area of use was assumed to be outdoors, so bystander

exposure was not estimated (see Section 2.4.2.3.9). Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.9.

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium for the consumer inhalation estimate, as discussed in Section 2.4.2.3.9.

Table 4-99. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Caulks, Sealants and Column Adhesives Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10
	User MOE
Low Intensity User	192
Moderate Intensity User	**2.3**
High Intensity User	**7.2E-02**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high and moderate intensity users by inhalation exposures and now for low intensity users.

4.2.4.11 Outdoor Water Shield

Estimates of MOEs for acute inhalation and dermal exposures for the outdoor water shield consumer use are presented in Table 4-100 and Table 4-101, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.10.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section2.4.2.3.4 2.4.2.3.10.

Table 4-100. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Outdoor Water Shield Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE

Low Intensity User	**7.6**	29
Moderate Intensity User	**1.1**	**3.3**
High Intensity User	**8.9E-02**	**0.4**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-101. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Outdoor Water Shield Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	**0.1**
	Youth (16-20 years)	**0.1**
	Youth (11-15 years)	**0.1**
Moderate Intensity User	Adult (≥21 years)	**2.6E-02**
	Youth (16-20 years)	**2.8E-02**
	Youth (11-15 years)	**2.5E-02**
High Intensity User	Adult (≥21 years)	**5.2E-03**
	Youth (16-20 years)	**5.5E-03**
	Youth (11-15 years)	**5.0E-03**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for all users and high and moderate intensity bystanders by inhalation exposures and not for low intensity bystanders. The MOEs are below the benchmark MOE for all users by dermal exposures.

4.2.4.12 Aerosol Coatings and Primers

Estimates of MOEs for acute inhalation exposures for the aerosol coatings and primers consumer use are presented in Table 4-102. Dermal exposures for the aerosol coatings and primers consumer use are not expected as described in Section 2.4.2.3.11. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.112.4.2.3.92.4.2.3.8.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate, as discussed in Section 2.4.2.3.11.

Table 4-102. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Aerosol Coatings and Primers Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE

Low Intensity User	522	13448
Moderate Intensity User	62	2143
High Intensity User	**5.9**	209

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are below the benchmark MOE for high intensity users by inhalation exposures. The MOEs are above the benchmark MOE for medium and low intensity users and all bystanders by inhalation exposures.

4.2.4.13 Liquid Primers and Sealants

Estimates of MOEs for acute inhalation and dermal exposures for the liquid primers and sealants consumer use are presented in Table 4-103 and Table 4-104, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.12.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.12.

Table 4-103. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Liquid Primers and Sealants Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	10600	128556
Moderate Intensity User	1163	12434
High Intensity User	36	229

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-104. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Liquid Primers and Sealants Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	**1.4**
	Youth (16-20 years)	**1.5**
	Youth (11-15 years)	**1.4**
Moderate Intensity User	Adult (≥21 years)	**1.8E-02**

	Youth (16-20 years)	**1.9E-02**
	Youth (11-15 years)	**1.8E-02**
High Intensity User	Adult (≥21 years)	**1.6E-02**
	Youth (16-20 years)	**1.7E-02**
	Youth (11-15 years)	**1.6E-02**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are above the benchmark MOE for all users and bystanders by inhalation exposures. The MOEs are below the benchmark MOE for all users by dermal exposures.

4.2.4.14 Metallic Overglaze

Estimates of MOEs for acute inhalation exposures for the metallic overglaze consumer use are presented in Table 4-105. Dermal exposures for the caulks, sealants and column adhesives consumer use are not expected as described in Section 2.4.2.3.13. Consumer inhalation exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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. Inhalation exposures are presented for users and bystanders for 24-hour TWAs are presented in Section 2.4.2.3.13.

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium for the consumer inhalation estimate, as discussed in Section 2.4.2.3.13.

Table 4-105. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Metallic Overglaze Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	4372	21107
Moderate Intensity User	337	1674
High Intensity User	21	81

[1] 24 hrs HEC based on data from Altmann et al. (1990)

The MOEs are above the benchmark MOE for all users and bystanders by inhalation exposures.

4.2.4.15 Metal and Stone Polish

Estimates of MOEs for acute inhalation and dermal exposures for the liquid wax-based metal and stone polish consumer use are presented in Table 4-106 and Table 4-107, respectively. Consumer inhalation and dermal exposures were modeled across a range of low, moderate, and high user intensities as described in detail in Section 2.4.2.2. 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 24-hour TWAs and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.3.14.

Considering the overall strengths and limitations of the data, EPA's overall confidence is high for the consumer inhalation estimate and medium for the dermal estimate, as discussed in Section 2.4.2.3.14.

Table 4-106. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Metal and Stone Polish Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User MOE	Bystander MOE
Low Intensity User	**1.1**	**5.3**
Moderate Intensity User	**0.2**	**0.8**
High Intensity User	**1.5E-02**	**6.1E-02**

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-107. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Metal and Stone Polish Consumer Use

Exposure Scenario	Consumer Receptor	Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10
		User MOE
Low Intensity User	Adult (≥21 years)	**1.0**
	Youth (16-20 years)	**1.0**
	Youth (11-15 years)	**1.0**
Moderate Intensity User	Adult (≥21 years)	**0.1**
	Youth (16-20 years)	**0.1**
	Youth (11-15 years)	**0.1**
High Intensity User	Adult (≥21 years)	**1.4E-02**
	Youth (16-20 years)	**1.5E-02**
	Youth (11-15 years)	**1.3E-02**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

The MOEs are below the benchmark MOE for all users and bystanders by inhalation and dermal exposures.

4.2.4.16 Dry Cleaned Clothing

Estimates of MOEs for acute inhalation and dermal exposures for the dry cleaned clothing consumer use are presented in Table 4-108 and Table 4-109, respectively. Consumer inhalation and dermal exposures were modeled as described in Section 2.4.2.4. Inhalation exposures are presented for users and bystanders for 24-hour TWAs in Section 2.4.2.4.3 and dermal exposure results are presented for users as acute ADRs in Section 2.4.2.4.2.

Considering the overall strengths and limitations of the data, EPA's overall confidence is medium to high for the consumer inhalation estimate and medium to high for the dermal estimate, as discussed in Section 2.4.2.4.2.

Table 4-108. Risk Estimation for Acute, Non-Cancer Inhalation Exposures for Dry Cleaned Clothing Consumer Use

Exposure Scenario	Acute HEC for CNS Effects[1] (11 mg/m^3) Benchmark MOE = 10	
	User (Adult) MOE	Bystander (Youth or Child) MOE
Stay-at-home Adult and Child	156	486

[1] 24 hrs HEC based on data from Altmann et al. (1990)

Table 4-109. Risk Estimation for Acute, Non-Cancer Dermal Exposures for Dry Cleaned Clothing Consumer Use

Acute HED for CNS Effects[1] (4.25 mg/kg/day) Benchmark MOE = 10				
Assumed dry cleaning technology	Dry Cleaning Events	Days After Dry Cleaning	User, Half-Body MOE	User, Full-Body MOE
2nd and 3rd generation	Single	1	**8.6**	**2.9**
		2	11	**3.7**
		3	15	**4.9**
4th and 5th generation	Single	1	49	16
		2	64	21
		3	83	28
4th and 5th generation	Repeat[2]	1	16	**5.2**
		2	20	**6.7**
		3	26	**8.7**

[1] HED extrapolated from inhalation exposures based on data from Altmann et al. (1990) described in Section 3.2.5.4.1

[2] Based on maximum average PCE concentration in wool after 6 dry cleaning cycles from Sherlach (2011); PCE concentration was still increasing in wool fabric after 6 cycles and had not yet reached saturation.

The MOEs are above the benchmark MOE for stay-at-home adults and children by inhalation. The MOEs are above the benchmark MOE for users exposed to half-body garments one day after dry cleaning, and full-body garments one to three days after dry cleaning for 2nd and 3rd generation dry cleaning technologies, and below the benchmark MOE for users exposed to half-body garments two and three days after dry cleaning for 2nd and 3rd generation dry cleaning technologies. The MOEs are above benchmark MOE for users exposed to full-body garments one to three days after multiple dry cleaning cycles for 4th and 5th generation dry cleaning technologies, and below the benchmarck MOE for users exposed to half- and full-body garments, one to three days after dry cleaning, for single event and multiple dry cleaning cycles, for 4th and 5th generation dry cleaning technologies.

4.3 Assumptions and Key Sources of Uncertainty for Risk Characterization

4.3.1 Environmental Risk Characterization Assumptions and Key Sources of Uncertainty

PCE is toxic to aquatic organisms. The EPA has determined that data are sufficient to characterize the environmental hazards of PCE and that the exposure pathways to the terrestrial environment are not likely. The following uncertainties are associated with the hazard characterization. Assessment factors (AFs) were used to calculate the acute and chronic COC for PCE. As described in Section 3.1.4, AFs address the inter- and intra-species variability, as well as laboratory-to-field variability and are routinely used within TSCA for assessing chemical hazards with limited environmental data. Additionally, AFs account for potential data gaps in the literature in which data for more sensitive species were not available. Use of AFs increases the confidence that the hazard characterizations were not underestimated, resulting in false negative conclusions. Although the toxicity values for fish, and invertebrates are relatively consistent, algae species tend to vary widely in their sensitivity to chemical pollutants. Data were only available for three algal species and may not represent the most sensitive species at a given site. Additionally, there were no PCE toxicity data available for amphibians.

Measured Surface Water Data and Watershed Analysis

The physical properties of PCE can lead to monitoring data showing limited occurrence in surface water. PCE in surface waters can be expected to volatilize into the atmosphere. However, PCE is denser than water and only slightly soluble in water. In soil and aquifers, it will tend to remain in the aqueous phase and be transported to ground water.

WQX surface water monitoring data for the following years of 2013-2017 showed that PCE occurrence was relatively low. For the 2016 data, only 4 monitoring sites had PCE concentrations above the monitoring detection limit. The concentrations ranged from 1.4E-2 to 5.2E-2 µg/L, which are below the lowest COC of 1.4 µg/L that is used in the ecological assessment.

When evaluating surface water monitoring data, it must be noted that EPA only looked at surface water data that excluded other major sources of water data, e.g., drinking water, superfund sites, and ground water. 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 specifically designed to evaluate PCE distribution across the U.S. As a result, there are uncertainties in the reported data that are difficult to quantify with regard to impacts on exposure estimates.

The available data represent a variety of discrete locations and time periods; therefore, it is unclear whether the data are representative of other locations in the U.S.; however, this limitation does not diminish the overall findings reported in this assessment, as the exposure data show very few instances (*i.e.*, less than 0.01 percent) where measured PCE levels in the ambient environment exceeded the identified hazard benchmarks for aquatic organisms.

The surface water monitoring results were further validated through data acquired via EPA's systematic review of surface water literature and biomonitoring data. Minimum results came from the systematic review on PCE in surface water. Data from three U.S. studies indicated that PCE occurrence and related concentrations in surface water were relatively low as well. The reported concentrations of PCE ranged from below the detection limit and reported central tendency values ranging from <0.2 to 0.7 µg/L which is below the lowest COC of 1.4 µg/L. The systematic review of biomonitoring data yielded three

viable studies that contained PCE concentration measurements in blood. These studies did indicate that PCE was detected moderately (37-60%) in samples evaluated. However, the concentration of PCE was not higher than the detection limits of the respective studies.

Modeled Surface Water Concentrations

To further evaluate PCE exposure in surface water EPA modeled indirect and direct releases of PCE in surface water by facilities. EPA modeled releasing facilities plus one industry with sites nationwide that was obtained by three data sources (TRI, DMRs, and CDR) for the 2016 calendar year.

The modeled estimations of PCE releases and surface water monitoring data were merged and mapped to reflect where PCE occurrence and related concentrations are with respect to each other in the U.S. The maps show that there is minimum PCE exposure at the respective COC in regard to environmental exposure assessment for aquatic species. The co-location of PCE releasing facilities and surface water monitoring stations in an HUC were also mapped via geospatial analysis to illustrate both measured and predicted concentrations PCE. The maps indicate that even though there are estimated releases from facilities, some of which have concentrations higher than the COC, the data from monitoring stations are not detecting PCE within the same HUC. It must be noted that the use geospatial analysis has a limitation with the accuracy of the latitudes and longitudes therefore affecting placement of facilities and monitoring stations.

4.3.2 Human Health Risk Characterization Key Assumptions and Uncertainties

4.3.2.1 Human Health Hazard Considerations

There is medium-high confidence in the acute non-cancer POD, high confidence in the chronic non-cancer PODs selected to represent each health domain, and medium confidence in the cancer POD. Confidence is reduced for dermal PODs due to the use of route-to-route extrapolation in the absence of a dermal compartment in the PBPK model (Section 3.2.6.4). Major uncertainties include the selection of cancer endpoint for IUR selection and inconclusive human evidence for a few health domains.

4.3.2.2 Occupational Risk Considerations

EPA estimated inhalation risk to workers and ONUs based on monitoring and/or modeling data, as reasonably available. For the majority of OES, only one source was available so the results could not be compared. Despite the absence of both types of data for most OES, overall confidence in worker inhalation estimates ranged from Medium to High for all OES (Table 2-15). For ONUs, modeling or monitoring data was available in 9 of 22 OES. For the other 13, in the absence of reasonably available data EPA applied the worker central tendency estimates to ONUs. When ONU data was not available, there is low confidence in ONU risk estimates. There is medium confidence in dermal exposure estimates, which are based on the *Dermal Exposure to Volatile Liquids Model* (Section 2.4.1.29).

There are significant uncertainties associated with PPE usage across OES. For the majority of OES, EPA assumes that workers will responsibly wear gloves and respirators and that employers implement a continuing, effective respiratory protection program according to the requirements of OSHA's Respiratory Protection Standard. This results in respiratory protection up to APF = 50 and glove protection up to PF = 20 (or PF = 10 for commercial scenarios). Respiratory protection factors can be confirmed through regular fit testing, however glove PFs represent a what-if scenario and EPA cannot confirm the actual frequency, type, and effectiveness of globe use in specific workplaces with PCE conditions of use. Risks may be underestimated by these assumptions. EPA also identified OES for

which regular respirator use is not expected (Table 4-8), and risks may be overestimated for these scenarios if even mild respiratory protection is employed.

4.3.2.3 Consumer Risk Considerations

There is medium to high or high confidence in both the consumer inhalation and dermal exposure estimates (Section 2.4.2.6). All exposure estimates are based on modeling, and there is uncertainty based on the application of surrogate product categories from the Westat survey (Westat 1987) when there was not an exact match for the COU. Professional judgement was also required for determining the most appropriate room of use, which affects the area volume and in turn inhalation exposure estimates. A key uncertainty for the dermal estimates is the accuracy of the assumption of which COUs are likely to result in exposure with impeded evaporation, and whether evaporation is truly fully impeded for those scenarios.

EPA only evaluated acute risks for consumer COUs. While the expected sparse and intermittent use frequency for the vast majority of users indicates that only acute risks are relevant to consumer uses, there is uncertainty whether chronic risks may be of concern for consumers at the very high end of the range for frequency of use, especially if a product is used several days consecutively. Without continued consecutive use, chronic hazards are unlikely due to the relatively short half life of TCE (Section 3.2.2.1.3).

4.4 Other Risk Related Considerations

4.4.1 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."

EPA identified workers, ONUs, consumers, and bystanders as potentially exposed populations. EPA provided risk estimates for workers and ONUs at both central tendency and high-end exposure levels for all COUs. Consumer and bystander risk estimates were provided for low, medium, and high intensities of use, accounting for differences in duration, weight fraction, and mass used. Occupational dermal risk estimates were calculated for both average workers and women of childbearing age (see *Draft Risk Evaluation for Perchloroethylene Supplemental File: Occupational Exposure Risk Calculator* (U.S. EPA 2020e)) and consumer dermal risk estimates were calculated for both adult and children (see *Draft Risk Evaluation for Perchloroethylene Consumer Dermal Risk Calculations* (U.S. EPA 2020b). EPA determined that bystanders may include lifestages of any age. These groups exhibit differences in delivered dose accounting for differing body weight and hand size, accounting for differences in exposure, and providing risk estimates for women of childbearing age protects the susceptible subpopulation of the developing fetus.

For inhalation exposures, risk estimates did not differ between sexes or across lifestages because both exposures and inhalation hazard values are expressed as an air concentration. EPA expects that variability in human physiological factors (e.g., breathing rate, body weight, tidal volume) which may affect internal delivered concentration or dose is sufficiently accounted for through the use of a 10x UF

for human intraspecies variability, although some differences among lifestages or between working and at-rest individuals may not have been accounted for by this value. EPA identified lifestage, biological sex, genetic polymorphisms, race/ethnicity, preexisting health status, and lifestyle factors and nutrition status as factors affecting biological susceptibility. Similarly, most but not all of these factors are expected to be covered by the inclusion of a 10x UF_H.

EPA was unable to directly account for all possible PESS considerations and subpopulations in the risk estimates. It is unknown whether the 10x UF to account for human variability will cover the full breadth of human responses, and subpopulations with particular disease states or genetic predispositions may fall outside of the range covered by this UF. As previously discussed, EPA also only considered acute effects from consumer exposure. While typical use patterns are unlikely to result in any chronic effects for the vast majority of consumers, EPA cannot rule out that consumers at very high frequencies of use may be at risk for chronic hazards, especially if those consumers also exhibit biological susceptibilities. EPA can also not rule out that certain subpopulations, whether due to very elevated exposure or biological susceptibility, may be at risk for hazards that were not fully supported by the weight of evidence or could not be quantified (e.g. immune and blood effects). However, in these circumstances EPA assumes that these effects are likely to occur at a higher dose than more sensitive endpoints that were accounted for by risk estimates.

4.4.2 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. Due to deference to existing environmental statutes, administered by EPA, a detailed analysis of environmental pathways to the general population was not deemed appropriate for this risk evaluation.

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." In terms of this risk evaluation, the EPA considered sentinel exposure in the form of a high-end screening level scenario for occupational exposure resulting from dermal and inhalation exposures, as these exposure routes are the most likely to result in the highest exposure given the details of the manufacturing process and the potential exposure scenarios discussed above. The calculation for dermal exposure is especially conservative given that it assumes full contact/immersion.

4.5 Risk Conclusions

4.5.1 Environmental Risk Conclusions

Aquatic Pathways

Table 4-110 displays risk quotients for each of the facilities by COU. No risks were identified for aquatic organisms from PCE release to surface water from the Maskants for Chemical Milling, Dry Cleaning (Industrial and Commercial), Other Industrial, and Other Commercial Uses COUs. *Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.*

Risks from acute PCE exposures were identified for aquatic organisms based on indirect releases from the Incorporation into Formulations COU. *Therefore, EPA concludes there is an acute risk to aquatic*

organisms from release of PCE to surface water from facilities using PCE from the Incorporation into Formulations COU. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.

Risks from chronic PCE exposures were identified for aquatic organisms based on direct releases from the Processing as a Reactant COU, and indirect releases from Incorporation into Formulations COU. *Therefore, EPA concludes there is a chronic risk to aquatic organisms from release of PCE to surface water from facilities using PCE for the COUs listed above. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.*

Risks from PCE exposures were identified for algae based on direct releases from the following COUs: Manufacturing; Processing as a Reactant; Open-Top Vapor Degreasing; and Industrial Processing Aid. In addition, indirect release (80% removal) from Manufacturing, Importing/Repackaging, Industrial Processing Aid; Incorporation into Formulations; and Waste Handling, Disposal, Treatment, and Recycling COUs resulted in risks to algae from PCE exposure. *Therefore, EPA concludes there is a risk to algae from release of PCE to surface water from facilities using PCE for the COUs listed above. Based on the data quality, uncertainties and weight of scientific evidence, confidence in the risk estimate is medium.*

Table 4-110. Modeled Facilities Showing RQs and Days of Exceedance from the Release of PCE to Surface Water as Modeled in E-FAST. Acute risk = RQs ≥ 1, chronic and algae risk = RQs ≥ 1 and ≥ 20 days of exceedance. Shaded areas show risk.

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
OES: Manufacturing											
Axiall Corporation Westlake, LA NPDES: LA0000761	Surface Water or POTW	Direct (0% WWT removal): LA0000761 Indirect (80% WWT removal): Organic Chemicals Mfg	Surface Water	350	0.1 (max)	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	0	2.2E-3
								Algae	1.4	0	7.9E-2
						80	2.3E-2	Acute	1,342	0	1.7E-5
								Chronic	50	0	4.5E-5
								Algae	1.4	0	1.6E-2
				350	3.0E-2 (avg)	0	3.4E-2	Acute	1,342	0	2.5E-5
								Chronic	50	0	6.8E-4
								Algae	1.4	0	2.4E-2
						80	1.1	Acute	1,342	0	8.2E-4
								Chronic	50	0	2.2E-2
								Algae	1.4	0	0.8
				20	0.5	0	0.6	Acute	1,342	0	4.6E-4
								Chronic	50	0	1.2E-2
								Algae	1.4	0	0.4
Greenchem West Palm Beach, FL NPDES: None (FRS 110056959634)	Surface Water or POTW	Direct and Indirect Surrogate: Organic Chemicals Mfg Receiving Facility: Unknown	Surface Water	350	0.1 (max)	0	18	Acute	1,342	0	1.4E-2
								Chronic	50	25	0.4
								Algae	1.4	189	13
						80	3.7	Acute	1,342	0	2.8E-3
								Chronic	50	7	7.5E-2
								Algae	1.4	77	2.7
				350	3.0E-2 (avg)	0	5.6	Acute	1,342	0	4.1E-3
								Chronic	50	11	0.1
								Algae	1.4	100	4.0
						80	1.1	Acute	1,342	0	8.3E-04
								Chronic	50	1	2.2E-2
								Algae	1.4	37	0.8
				20	0.5	0	100	Acute	1,342	0	7.4E-2
								Chronic	50	4	2.0
								Algae	1.4	17	71
Occidental Chemical Corp	Surface Water	LA0002933	Surface Water	350	2.0E-3	0	8.1E-6	Acute	1,342	0	6.0E-9
								Chronic	50	0	1.6E-7

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Geismar Plant Geismar, LA NPDES: LA0002933								Algae	1.4	0	5.8E-6
				20	3.0E-2	0	1.2E-4	Acute	1,342	0	9.0E-8
								Chronic	50	0	2.4E-6
								Algae	1.4	0	8.6E-5
Olin Blue Cube Freeport, TX NPDES: None (FRS 110066943605)	Non-POTW WWT	Receiving Facility: TX0006483	Surface Water	350	4.0E-2	80	3.1E-3	Acute	1,342	0	2.3E-6
								Chronic	50	0	2.3E-6
								Algae	1.4	0	6.1E-5
				20	0.7	80	5.6E-2	Acute	1,342	0	2.2E-3
								Chronic	50	0	1.1E-3
								Algae	1.4	0	4.2E-5
Solvents & Chemicals Pearland, TX NPDES: Not available (TRI: 77588SLVNT4704S)	Surface Water or POTW	Direct and Indirect Surrogate: Organic Chemicals Mfg Receiving Facility: Unknown	Surface Water	350	3.0E-4 (max)	0	5.6E-2	Acute	1,342	0	1.1E-3
								Chronic	50	0	4.0E-2
								Algae	1.4	2	4.0E-2
						80	1.1E-3	Acute	1,342	0	4.1E-5
								Chronic	50	0	1.1E-3
								Algae	1.4	0	4.0E-2
				350	1.0E-4 (avg)	0	1.9E-2	Acute	1,342	0	8.3E-7
								Chronic	50	0	2.2E-5
								Algae	1.4	0	7.9E-4
						80	3.7E-3	Acute	1,342	0	1.4E-5
								Chronic	50	0	3.7E-4
								Algae	1.4	0	1.3E-2
				20	2.0E-3	0	0.4	Acute	1,342	0	2.8E-6
								Chronic	50	0	7.4E-5
								Algae	1.4	1	0.3
Univar USA Inc Redmond, WA NPDES: None (FRS: 110036000000)	Surface Water or POTW	Direct and Indirect Surrogate: Organic Chemicals Mfg Receiving	Surface Water	350	0.1 (max)	0	18	Acute	1,342	0	1.4E-2
								Chronic	50	25	0.4
								Algae	1.4	189	13
						80	3.7	Acute	1,342	0	2.8E-3
								Chronic	50	7	7.4E-2
								Algae	1.4	77	2.6
				350	3.0E-2 (avg)	0	5.6	Acute	1,342	0	4.1E-3
								Chronic	50	11	0.1

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
		Facility: Unknown						Algae	1.4	100	4.0
						80	1.1	Acute	1,342	0	8.3E-4
								Chronic	50	1	2.2E-2
								Algae	1.4	37	0.8
				20	0.5	0	100	Acute	1,342	0	7.4E-2
								Chronic	50	4	2.0
								Algae	1.4	17	71
OES: Import/Repackaging											
Chemtool Rockton, IL NPDES: IL0064564	Surface Water	IL0064564	Surface Water	250	1.0E-3	0	1.5E-3	Acute	1,342	0	1.1E-6
								Chronic	50	0	2.9E-5
								Algae	1.4	0	1.0E-3
				20	1.5E-2	0	2.2E-2	Acute	1,342	0	1.6E-5
								Chronic	50	0	4.4E-4
								Algae	1.4	0	1.6E-2
Harvey Terminal Harvey, LA NPDES: LA0056600	Surface Water	Surrogate based on location: LA0005291	Surface Water	250	1.0E-4	0	4.1E-07	Acute	1,342	0	3.0E-10
								Chronic	50	0	8.1E-9
								Algae	1.4	0	2.9E-7
				20	1.0E-3	0	4.1E-06	Acute	1,342	0	3.0E-9
								Chronic	50	0	8.1E-8
								Algae	1.4	0	2.9E-6
Hubbard-Hall Inc Waterbury, CT NPDES: None (FRS 110000317194	Non-POTW WWT	Surrogate: Industrial POTW (for receiving facility FRS 110004250541)	Surface Water	250	1.1	80	29	Acute	1,342	0	2.2E-2
								Chronic	50	16	0.6
								Algae	1.4	230	21
				20	14	80	360	Acute	1,342	0	0.27
								Chronic	50	14	7.2
								Algae	1.4	20	257
Vopak Terminal Westwego Inc Westwego, LA NPDES: LA0124583	Surface Water	Surrogate based on location: LA0003093	Surface Water	250	5.0E-3	0	2.1E-05	Acute	1,342	0	1.5E-8
								Chronic	50	0	4.0E-7
								Algae	1.4	0	1.4E-5
				20	0.1	0	2.4E-04	Acute	1,342	0	1.8E-7
								Chronic	50	0	4.9E-6
								Algae	1.4	0	1.7E-4
OES: Processing as a Reactant											

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Akzo Nobel Surface Chemistry LLC Morris, IL NPDES: IL0026069	Surface Water	IL0026069	Surface Water	350	1.0E-4	0	2.1E-4	Acute	1,342	0	1.6E-7
								Chronic	50	0	4.2E-6
								Algae	1.4	0	1.49E-04
				20	2.5E-3	0	5.2E-3	Acute	1,342	0	3.88E-06
								Chronic	50	0	1.04E-04
								Algae	1.4	0	0.00372
Atkemix Ten Inc Louisville, KY NPDES: KY0002780	Surface Water	KY0002780	Surface Water	350	7.0E-2	0	3.8E-3	Acute	1,342	0	2.79E-06
								Chronic	50	0	7.50E-05
								Algae	1.4	0	0.0027
				20	1.3	0	6.9E-2	Acute	1,342	0	5.153E-05
								Chronic	50	0	0.0014
								Algae	1.4	0	0.049
Bayer Corporation Haledon, NJ NPDES: NJG104451	Surface Water	Surrogate: Organic Chemical Mfg SIC	Surface Water	350	4.0E-5	0	7.4E-3	Acute	1,342	0	5.51E-06
								Chronic	50	0	1.48E-04
								Algae	1.4	0	0.00528
				20	5.0E-4	0	9.2E-2	Acute	1,342	0	6.88525E-05
								Chronic	50	0	0.001848
								Algae	1.4	0	0.066
Bayer MaterialScience New Martinsville, WV NPDES: WV0005169	Surface Water	WV0005169	Surface Water	350	1.0E-3	0	1.2E-4	Acute	1,342	0	8.86736E-08
								Chronic	50	0	2.38E-06
								Algae	1.4	0	8.50E-05
				20	0.013	0	1.6E-3	Acute	1,342	0	1.15E-06
								Chronic	50	0	3.10E-05
								Algae	1.4	0	0.0011
Chemtura North and South Plants Morgantown, WV NPDES: WV0004740	Surface Water	WV0004740	Surface Water	350	2.0E-5	0	2.9E-5	Acute	1,342	0	2.16E-08
								Chronic	50	0	5.80E-07
								Algae	1.4	0	2.07E-05
				20	5.0E-4	0	7.3E-4	Acute	1,342	0	5.40E-07
								Chronic	50	0	1.45E-05
								Algae	1.4	0	5.18E-04
Dupont-Chemours Montague Site Montague, MI	Surface Water	MI0000884	Still Water	350	2.0E-2	0	2.4	Acute	1,342	0	0.0018
								Chronic	50	0	0.0484
								Algae	1.4	350	1.73

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: MI0000884				20	0.3	0	35	Acute	1,342	0	0.026
								Chronic	50	0	0.7014
								Algae	1.4	20	25.05
Eagle US 2 LLC - Lake Charles Complex Lake Charles, LA NPDES: LA0000761	Surface Water	LA0000761	Surface Water	350	1.3	0	1.5	Acute	1,342	0	1.1E-3
								Chronic	50	0	3.0E-2
								Algae	1.4	29	1.1
				20	23	0	26	Acute	1,342	0	2.0E-2
								Chronic	50	0	0.5
								Algae	1.4	17	19
Flint Hills Resources Corpus Christi LLC - West Plant Corpus Christi, TX NPDES: TXU001146, TX0006289	Surface Water	TX0006289	Still Water	350	7.0E-2	0	3.0	Acute	1,342	0	2.2E-3
								Chronic	50	0	6.0E-2
								Algae	1.4	350	2.15
				20	1.2	0	52	Acute	1,342	0	3.8E-2
								Chronic	50	20	1.0
								Algae	1.4	20	37
Flint Hills Resources Pine Bend LLC Rosemount, MN NPDES: MN0070246, MN0000418	Surface Water	MN0000418	Surface Water	350	1.0E-2	0	2.8E-3	Acute	1,342	0	2.1E-6
								Chronic	50	0	5.7E-5
								Algae	1.4	0	2.0E-3
				20	0.2	0	5.7E-2	Acute	1,342	0	4.2E-5
								Chronic	50	0	1.1E-3
								Algae	1.4	0	4.0E-2
Honeywell International Inc - Geismar Complex Geismar, LA NPDES: LA0006181	Surface Water	LA0006181	Surface Water	350	2.0E-2	0	8.1E-5	Acute	1,342	0	6.0E-8
								Chronic	50	0	1.6E-6
								Algae	1.4	0	5.8E-5
				20	0.36	0	1.5E-3	Acute	1,342	0	1.1E-6
								Chronic	50	0	2.9E-5
								Algae	1.4	0	1.0E-3
Honeywell International Inc- Baton Rouge Plant	Surface Water	LA0000329	Surface Water	350	5.0E-2	0	4.9	Acute	1,342	0	3.7E-3
								Chronic	50	0	9.9E-2
								Algae	1.4	193	3.53

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Baton Rouge, LA NPDES: LAR10E873, LA0000329				20	0.9	0	85	Acute	1,342	0	6.0E-2
								Chronic	50	7	1.7
								Algae	1.4	20	61
Indorama Ventures Olefins, LLC Sulphur, LA NPDES: LA0069850	Surface Water	Surrogate: Organic Chemical Mfg SIC	Surface Water	350	1.0E-5	0	1.9E-3	Acute	1,342	0	1.4E-6
								Chronic	50	0	3.7E-5
								Algae	1.4	0	1.3E-3
				20	2.0E-4	0	3.7E-2	Acute	1,342	0	2.8E-5
								Chronic	50	0	7.4E-4
								Algae	1.4	0	2.6E-2
Keeshan And Bost Chemical Co., Inc. Manvel, TX NPDES: TX0072168	Surface Water	TX0072168	Still Water	350	5.0E-5	0	5.0	Acute	1,342	0	3.7E-3
								Chronic	50	0	0.1
								Algae	1.4	350	3.6
				20	1.0E-3	0	100	Acute	1,342	0	7.5E-2
								Chronic	50	20	2.0
								Algae	1.4	20	71
Phillips 66 Lake Charles Refinery Westlake, LA NPDES: LAR05P540, LA0003026	Surface Water	LA0003026	Surface Water	350	6.0E-2	0	9.5E-2	Acute	1,342	0	7.0E-5
								Chronic	50	0	1.9E-3
								Algae	1.4	0	6.8E-2
				20	1.0	0	1.6	Acute	1,342	0	1.2E-3
								Chronic	50	0	3.2E-2
								Algae	1.4	1	1.2
Phillips 66 Los Angeles Refinery Wilmington Plant Wilmington, CA NPDES: CA0000035	POTW	Receiving Facility: CA0053856	Still Water	350	0.1	80	0.3	Acute	1,342	0	2.4E-4
								Chronic	50	0	6.4E-3
								Algae	1.4	0	0.2
Premcor Refining Group Inc Port Arthur Port Arthur, TX	Surface Water	TX0005991	Surface Water	350	0.1	0	2.0	Acute	1,342	0	1.5E-3
								Chronic	50	0	4.0E-2
								Algae	1.4	67	1.4
				20	2.3	0	34	Acute	1,342	0	2.6E-2
								Chronic	50	1	0.7

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES:								Algae	1.4	17	25
Solutia Nitro Site Nitro, WV NPDES: WV0116181	Surface Water	Surrogate: WV0000868	Surface Water	350	2.0E-4	0	5.9E-5	Acute	1,342	0	4.4E-8
								Chronic	50	0	1.2E-6
								Algae	1.4	0	4.2E-5
				20	3.0E-3	0	8.8E-4	Acute	1,342	0	6.6E-7
								Chronic	50	0	1.8E-5
								Algae	1.4	0	6.3E-4
Solvay - Houston Plant Houston, TX NPDES: TX0007072	Surface Water	TX0007072	Surface Water	350	2.0E-2	0	3.7	Acute	1,342	0	2.8E-3
								Chronic	50	0	7.4E-2
								Algae	1.4	8	2.6
				20	0.4	0	76	Acute	1,342	0	5.7E-2
								Chronic	50	0	1.5
								Algae	1.4	8	54
OES: Incorporation into Formulation											
Lord Corp Saegertown, PA NPDES: PA0101800	Non-POTW WWT	Surrogate: Industrial POTW	Surface Water	300	5.3	80	136	Acute	1,342	1	0.1
								Chronic	50	127	2.7
								Algae	1.4	299	97
				20	79	80	2034	Acute	1,342	5	1.5
								Chronic	50	20	41
								Algae	1.4	20	1453
Stepan Co Millsdale Road Elwood, IL NPDES: IL0002453	Surface Water	IL0002453	Surface Water	300	2.0E-3	0	8.4E-4	Acute	1,342	0	1.5E-6
								Chronic	50	0	4.0E-5
								Algae	1.4	0	1.4E-3
				20	2.5E-2	0	1.1E-2	Acute	1,342	0	7.8E-6
								Chronic	50	0	2.1E-4
								Algae	1.4	0	7.5E-3
Tesoro Los Angeles Refinery-Carson Operations Carson, CA	POTW	Receiving Facility: CA0053813	Still Water	300	0.3	80	2.7E-4	Acute	1,342	0	2.0E-7
								Chronic	50	0	5.3E-6
								Algae	1.4	0	1.9E-4

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: CA0000680											
Weatherford Aerospace LLC Weatherford, TX NPDES: None (FRS 110000743740)	POTW	Receiving Facility: TX0047724	Surface Water	300	2.0E-3	80	6.5E-2	Acute	1,342	0	4.9E-5
								Chronic	50	0	1.3E-3
								Algae	1.4	0	4.7E-2
OES: Open Top Vapor Degreasing											
601 Nassau St Assoc LLC North Brunswick Twp, NJ NPDES: NJG129127	Surface Water	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	1.0E-5	0	1.1E-3	Acute	1,342	0	8.3E-7
								Chronic	50	0	2.2E-5
								Algae	1.4	0	7.9E-4
				20	1.0E-3	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	0	2.2E-3
								Algae	1.4	2	7.9E-2
ASCO Valve Manufacturing Aiken, SC NPDES: SC0049026	Surface Water	SC0049026	Surface Water	260	1.0E-4	0	1.E-2	Acute	1,342	0	8.3E-6
								Chronic	50	0	2.2E-4
								Algae	1.4	7	7.9E-3
				20	1.9E-3	0	0.2	Acute	1,342	0	1.6E-4
								Chronic	50	0	4.2E-3
								Algae	1.4	2	0.2
Chemours - Beaumont Works Beaumont, TX NPDES: TX0004669	Surface Water	TX0004669	Surface Water	260	1.0E-2	0	1.4E-2	Acute	1,342	0	1.1E-5
								Chronic	50	0	2.8E-4
								Algae	1.4	0	1.0E-2
				20	8.4E-2	0	0.1	Acute	1,342	0	8.9E-5
								Chronic	50	0	2.4E-3
								Algae	1.4	0	8.6E-2
Delphi Harrison Thermal Systems Dayton, OH NPDES: OH0009431	Surface Water	OH0009431	Surface Water	260	1.0E-2	0	1.9E-2	Acute	1,342	0	1.4E-5
								Chronic	50	0	3.8E-4
								Algae	1.4	0	1.3E-2
				20	8.4E-2	0	0.2	Acute	1,342	0	1.2E-4
								Chronic	50	0	3.2E-3
								Algae	1.4	0	0.1
			Still Water	260	1.0E-2	0	0.2	Acute	1,342	0	1.5E-4

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Equistar Chemicals LP La Porte, TX NPDES: TX0119792		Surrogate: TX0002836						Chronic	50	0	4.0E-3
								Algae	1.4	0	0.1
				20	0.2	0	3.2	Acute	1,342	0	2.4E-3
								Chronic	50	0	6.5E-2
								Algae	1.4	20	2.3
Fairfield Works Fairfield, AL NPDES: AL0003646	Surface Water	AL0003646	Surface Water	260	4.0E-3	0	5.1E-3	Acute	1,342	0	3.7E-6
								Chronic	50	0	1.0E-4
								Algae	1.4	0	3.6E-3
				20	5.3E-2	0	6.7E-2	Acute	1,342	0	5.0E-5
								Chronic	50	0	1.3E-3
								Algae	1.4	0	4.8E-2
Gayston Corp Dayton, OH NPDES: OH0127043	POTW	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	3.0E-3	0	0.3	Acute	1,342	0	2.5E-4
								Chronic	50	5	6.6E-3
								Algae	1.4	25	0.2
				20	4.1E-2	0	4.6	Acute	1,342	0	3.4E-3
								Chronic	50	2	9.1E-2
								Algae	1.4	8	3.26
Getzen Co Inc Elkhorn, WI NPDES: None (FRS110000417291)	POTW	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	3.0E-4	80	6.7E-3	Acute	1,342	0	5.0E-6
								Chronic	50	0	1.3E-4
								Algae	1.4	3	4.8E-3
GM Components Holdings LLC Lockport, NY NPDES: NY0000558	Surface Water	NY0000558	Surface Water	260	7.0E-2	0	5.9	Acute	1,342	0	4.4E-3
								Chronic	50	0	0.1
								Algae	1.4	131	4.2
				20	0.9	0	78	Acute	1,342	0	5.8E-2
								Chronic	50	3	1.6
								Algae	1.4	20	55.46
HB Fuller Co Morris, IL	Surface Water	Surrogate: Primary Metal	Surface Water	260	1.0E-3	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	1	2.2E-3
								Algae	1.4	21	7.9E-2

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: IL0079758		Forming Manufacture		20	1.0E-2	0	1.1	Acute	1,342	0	8.3E-4
								Chronic	50	1	2.2E-2
								Algae	1.4	3	0.8
Hyster-Yale Group, Inc Sulligent, AL NPDES: AL0069787	Surface Water	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	1.0E-6	0	1.1E-4	Acute	1,342	0	8.3E-8
								Chronic	50	0	2.22E-6
								Algae	1.4	0	7.9E-05
				20	1.2E-5	0	1.3E-3	Acute	1,342	0	9.7E-7
								Chronic	50	0	2.6E-5
								Algae	1.4	0	9.3E-4
MEMC Electronic Materials Incorporated Moore, SC NPDES: SC0036145	Surface Water	SC0036145	Surface Water	260	3.0E-4	0	1.0E-2	Acute	1,342	0	7.5E-6
								Chronic	50	0	2.0E-4
								Algae	1.4	0	7.2E-3
				20	3.4E-3	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	0	2.2E-3
								Algae	1.4	0	7.9E-2
Piano Factory-Grand Haven Grand Haven, MI NPDES: MI0054399	Surface Water	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	1.0E-3	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	1	2.2E-3
								Algae	1.4	21	7.9E-2
				20	9.3E-3	0	1.0	Acute	1,342	0	7.7E-4
								Chronic	50	1	2.1E-2
								Algae	1.4	3	0.7
Rex Heat Treat Lansdale Inc Lansdale, PA NPDES: PA0052965	Surface Water	Surrogate: PA0026182	Surface Water	260	2.0E-3	0	5.4E-2	Acute	1,342	0	4.0E-5
								Chronic	50	0	1.1E-3
								Algae	1.4	0	0.03.9E-2
				20	2.5E-2	0	0.7	Acute	1,342	0	5.0E-4
								Chronic	50	0	1.3E-2
								Algae	1.4	0	0.5
Styrolution America LLC Channahon, IL	Surface Water	IL0001619	Surface Water	260	1.0E-5	0	3.5E-6	Acute	1,342	0	2.6E-9
								Chronic	50	0	6.9E-8
								Algae	1.4	0	2.5E-6

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: IL0001619				20	8.3E-3	0	2.9E-3	Acute	1,342	0	2.2E-6
								Chronic	50	0	5.8E-5
								Algae	1.4	0	2.1E-3
Trane Residential Solutions - Fort Smith Fort Smith, AR NPDES: AR0052477	Surface Water	Surrogate: Primary Metal Forming Manufacture	Surface Water	260	1.0E-5	0	1.1E-3	Acute	1,342	0	8.3E-7
								Chronic	50	0	2.2E-5
								Algae	1.4	0	7.9E-4
				20	1.7E-4	0	1.9E-2	Acute	1,342	0	1.4E-5
								Chronic	50	0	3.8E-4
								Algae	1.4	1	1.4E-2
US Steel Fairless Hills Facility Fairless Hills, PA NPDES: PA0013463	Surface Water	PA0013463	Surface Water	260	1.0E-3	0	1.7E-4	Acute	1,342	0	1.2E-7
								Chronic	50	0	3.3E-6
								Algae	1.4	0	1.2E-4
				20	1.3E-2	0	2.2E-3	Acute	1,342		1.6E-6
								Chronic	50	0	4.3E-5
								Algae	1.4	0	1.5E-3
OES: Dry Cleaning (Commercial and Industrial)											
12,822 Commercial Dry cleaning Sites		Surrogate: Laundry/Dry Cleaner SIC	Surface Water	307	2.0E-2 (high-end)	80	0.4	Acute	1,342	0	2.8E-4
								Chronic	50	0	7.6E-3
								Algae	1.4	0	0.3
				289	1.0E-3 (central tendency)	80	0.2	Acute	1,342	0	1.4E-4
								Chronic	50	0	3.8E-3
								Algae	1.4	0	0.1
Boise State University Boise, ID NPDES: IDG911006	Surface Water	Surrogate: Laundry/Dry Cleaner SIC	Surface Water	289	2.0E-4 (high-end)	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	0	2.2E-3
								Algae	1.4	0	7.9E-2
				307	2.0E-4 (central tendency)	0	0.1	Acute	1,342	0	8.2E-5
								Chronic	50	0	0.002.2E-3
								Algae	1.4	0	7.9E-2
				20	3.0E-3	0	1.7	Acute	1,342	0	1.3E-3
								Chronic	50	0	3.4E-2
								Algae	1.4	1	1.2

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Unifirst Williamstown, VT NPDES: VT0000850	Surface Water	Surrogate: Laundry/Dry Cleaner SIC	Surface Water	289	5.0E-5 (high-end)	0	2.8E-2	Acute	1,342	0	2.1E-5
								Chronic	50	0	5.7E-4
								Algae	1.4	0	0.2
				307	4.0E-5 (central tendency)	0	2.3E-2	Acute	1,342	0	1.7E-5
								Chronic	50	0	4.5E-4
								Algae	1.4	0	1.6E-2
				20	6.8E-4	0	0.4	Acute	1,342	0	2.9E-4
								Chronic	50	0	7.8E-3
								Algae	1.4	0	0.3
OES: Chemical Maskant											
Alliant Techsystems Operations LLC Elkton, MD NPDES: MD0000078	Surface Water	MD0000078	Surface Water	172	5.8E-6	0	5.3E-4	Acute	1,342	0	4.0E-7
								Chronic	50	0	1.1E-5
								Algae	1.4	0	3.8E-4
				20	5.0E-5	0	4.6E-3	Acute	1,342	0	3.4E-6
								Chronic	50	0	9.2E-5
								Algae	1.4	0	3.3E-3
Ducommun Aerostructures Inc Orange Facility Orange, CA NPDES: None (110070089239)	POTW	Surrogate: Metal Finishing SIC (surrogate for receiving facility CA0110604)	Surface Water	172	2.6E-3	80	6.8E-2	Acute	1,342	0	5.0E-5
								Chronic	50	0	1.4E-3
								Algae	1.4	0	4.8E-2
GE Aviation Lynn, MA NPDES: MA0003905	Surface Water	MA0003905	Still Water	172	8.7E-4	0	3.7E-3	Acute	1,342	0	2.8E-6
								Chronic	50	0	7.4E-5
								Algae	1.4	0	2.6E-3
				20	7.5E-3	0	3.2E-2	Acute	1,342	0	2.4E-5
								Chronic	50	0	6.4E-4
								Algae	1.4	0	2.2E-2
McCanna Inc. Carpentersville, IL	Surface Water	Surrogate: Metal Finishing SIC	Surface Water	172	4.1E-4	0	0.2	Acute	1,342	0	1.3E-4
								Chronic	50	0	3.4E-3
								Algae	1.4	0	0.1
				20	3.5E-3	0	1.3	Acute	1,342	0	9.9E-4

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: IL0071340								Chronic	50	0	2.7E-2
								Algae	1.4	0	1.0
Weatherford Aerospace LLC Weatherford, TX NPDES: None (FRS 110000743740)	POTW	Receiving Facility: TX0047724	Surface Water	208	1.1E-2	80	0.3	Acute	1,342	0	2.1E-4
								Chronic	50	0	5.6E-3
								Algae	1.4	0	0.2
OES: Industrial Processing Aid											
Chevron Products Co - Salt Lake Refinery *Salt Lake City, UT* NPDES: UTG070261, UT0000175	Surface Water	UT0000175	Surface Water	300	1.0E-2	0	0.3	Acute	1,342	0	2.3E-4
								Chronic	50	0	6.2E-3
								Algae	1.4	0	0.2
				20	8.7E-2	0	2.7	Acute	1,342	0	2.0E-3
								Chronic	50	0	5.4E-2
								Algae	1.4	0	1.9
Chevron Products Co Richmond Refinery Richmond, CA NPDES: CA0005134	Surface Water	CA0005134	Surface Water	300	3.0E-3	0	0.2	Acute	1,342	0	1.3E-4
								Chronic	50	0	3.4E-3
								Algae	1.4	0	0.1
				20	4.6E-2	0	2.7	Acute	1,342	0	2.0E-3
								Chronic	50	0	5.3E-2
								Algae	1.4	20	1.9
CHS McPherson Refinery McPherson, KS NPDES: KS0000337	Surface Water	KS0000337	Surface Water	300	3.0E-4	0	4.4E-2	Acute	1,342	0	3.3E-5
								Chronic	50	0	8.8E-4
								Algae	1.4	0	3.2E-2
				20	4.5E-3	0	0.7	Acute	1,342	0	4.9E-4
								Chronic	50	0	1.3E-2
								Algae	1.4	0	0.5
ExxonMobil Oil Beaumont Refinery Beaumont, TX NPDES: None	Surface Water	TX0068934	Surface Water	300	20E-2	0	5.5	Acute	1,342	0	4.1E-3
								Chronic	50	0	0.11
								Algae	1.4	55	4.0
				20	0.4	0	97	Acute	1,342	0	7.2E-2
								Chronic	50	2	1.9
								Algae	1.4	20	69

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
(FRS 110056963683)											
HollyFrontier El Dorado Refining LLC El Dorado, KS NPDES: KS0000761	Surface Water	KS0000761	Surface Water	300	3.0E-3	0	0.6	Acute	1,342	0	4.4E-4
								Chronic	50	0	1.2E-2
								Algae	1.4	2	0.4
				20	4.6E-2	0	9.1	Acute	1,342	0	6.8E-3
								Chronic	50	0	0.2
								Algae	1.4	6	6.5
Hunt Refining Co - Tuscaloosa Refinery Tuscaloosa, AL NPDES: AL0000973	Surface Water	AL0000973	Surface Water	300	1.1E-2	0	3.3E-2	Acute	1,342	0	2.5E-5
								Chronic	50	0	6.6E-4
								Algae	1.4	0	2.4E-2
				20	0.2	0	0.7	Acute	1,342	0	4.9E-4
								Chronic	50	0	1.3E-2
								Algae	1.4	0	0.5
Marathon Petroleum Co LP Garyville, LA NPDES: LAU009485, LA0045683	Surface Water	LA0045683	Still Water	300	1.0E-2	0	0.5	Acute	1,342	0	3.5E-4
								Chronic	50	0	9.4E-3
								Algae	1.4	0	0.3
				20	0.1	0	6.6	Acute	1,342	0	4.9E-3
								Chronic	50	0	0.1
								Algae	1.4	20	4.7
Occidental Chemical Corp Niagara Plant Niagara Falls, NY NPDES: NY0003336	Surface Water and POTW	Direct (0% WWT Removal): NY0003336 Indirect (80% WWT Removal): Organic Chemicals Mfg (surrogate for NY0026336)	Still Water	300	0.2	0	1.3	Acute	1,342	0	9.6E-4
								Chronic	50	0	2.6E-2
								Algae	1.4	0	0.9
			Surface Water	300	0.2	80	6.3	Acute	1,342	0	4.7E-3
								Chronic	50	11	0.1
								Algae	1.4	92	4.5
			Still Water	20	2.6	0	20	Acute	1,342	0	1.5E-2
								Chronic	50	0	0.4
								Algae	1.4	20	14
				300	3.0E-2	0	12	Acute	1,342	0	8.9E-3

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Tesoro Los Angeles Refinery-Carson Operations Carson, CA NPDES: CA0000680	Surface Water and POTW	Direct (0% WWT removal): Petroleum Refining Indirect (80% WWT removal): CA0053813	Surface Water					Chronic	50	17	0.2
								Algae	1.4	169	8.5
			Surface Water	300	3.0E-2	80	2.4E-5	Acute	1,342	0	1.8E-8
								Chronic	50	0	4.8E-7
								Algae	1.4	0	1.7E-5
			Surface Water	20	0.4	0	171	Acute	1,342	1	0.1
								Chronic	50	7	3.4
								Algae	1.4	19	122
The Dow Chemical Co Midland, MI NPDES: MI0000868	Surface Water	MI0000868	Surface Water	300	3.0E-2	0	4.8E-2	Acute	1,342	0	3.5E-5
								Chronic	50	0	9.5E-4
								Algae	1.4	0	3.4E-2
				20	0.5	0	0.8	Acute	1,342	0	6.1E-4
								Chronic	50	0	1.6E-2
								Algae	1.4	1	0.6
Valero Refining Co -Oklahoma Valero Ardmore Refinery Ardmore, OK NPDES: OK0001295	Surface Water	OK0001295	Surface Water	300	1.0E-2	0	0.7	Acute	1,342	0	4.8E-4
								Chronic	50	0	1.3E-2
								Algae	1.4	6	0.5
				20	0.1	0	7.1	Acute	1,342	0	5.3E-3
								Chronic	50	0	0.1
								Algae	1.4	9	5.1
Valero Refining Co -Oklahoma Valero Ardmore Refinery Ardmore, OK NPDES: OK0001295	Surface Water	Surrogate: Organic Chemicals Mfg	Surface Water	300	1.0E-2	0	1.9	Acute	1,342	0	1.4E-3
								Chronic	50	2	3.7E-2
								Algae	1.4	42	1.3
				20	0.1	0	26	Acute	1,342	0	1.9E-2
								Chronic	50	2	0.5
								Algae	1.4	12	18
OES: Other Industrial Uses											
ExxonMobil Oil Corp JoiIet Refinery Channahon, IL	Surface Water	ILR10H432	Surface Water	250	5.0E-3	0	1.7E-3	Acute	1,342	0	1.3E-6
								Chronic	50	0	3.5E-5
								Algae	1.4	0	1.2E-3
				20	5.9E-2	0	2.1E-2	Acute	1,342	0	1.5E-5

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: ILR10H432								Chronic	50	0	4.1E-4
								Algae	1.4	0	1.5E-2
Natrium Plant New Martinsville, WV NPDES: WV0004359	Surface Water	WV0004359	Surface Water	250	3.0E-2	0	3.6E-3	Acute	1,342	0	2.7E-6
								Chronic	50	0	7.1E-5
								Algae	1.4	0	2.6E-3
				20	0.4	0	4.6E-2	Acute	1,342	0	3.5E-5
								Chronic	50	0	9.3E-4
								Algae	1.4	0	3.3E-2
Oxy Vinyls LP - Deer Park PVC Deer Park, TX NPDES: TX0007412	Surface Water	TX0007412	Surface Water	250	0.3	0	1.0	Acute	1,342	0	7.5E-4
								Chronic	50	0	2.0E-2
								Algae	1.4	38	0.7
				20	3.9	0	13	Acute	1,342	0	9.4E-3
								Chronic	50	0	0.3
								Algae	1.4	17	9.0
Princeton Plasma Physics Lab (FF) Princeton, NJ NPDES: NJ0023922	Surface Water	Surrogate: Industrial POTW	Surface Water	250	1.0E-3	0	0.1	Acute	1,342	0	9.7E-5
								Chronic	50	0	2.6E-3
								Algae	1.4	0	9.3E-2
				20	6.6E-3	0	0.9	Acute	1,342	0	6.3E-4
								Chronic	50	0	1.7E-2
								Algae	1.4	1	0.6
Tree Top Inc Wenatchee Plant Wenatchee, WA NPDES: WA0051527	Surface Water	Industrial POTW	Surface Water	250	3.0E-5	0	3.9E-3	Acute	1,342	0	2.9E-6
								Chronic	50	0	7.7E-5
								Algae	1.4	0	2.8E-3
				20	3.8E-4	0	4.9E-2	Acute	1,342	0	3.6E-5
								Chronic	50	0	9.8E-4
								Algae	1.4	0	3.5E-2
Vesuvius USA Corp Buffalo Plant Buffalo, NY NPDES: NY0030881	Surface Water	Surrogate: Industrial POTW	Surface Water	250	1.0E-3	0	0.1	Acute	1,342	0	9.7E-5
								Chronic	50	0	2.6E-3
								Algae	1.4	0	9.3E-2
				20	1.5E-3	0	0.2	Acute	1,342	0	1.4E-4
								Chronic	50	0	3.8E-3
								Algae	1.4	0	0.1
		CA0059188		250	1.0E-6	0	0.1	Acute	1,342	0	7.5E-5

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
William E. Warne Power Plant Los Angeles County, CA NPDES: CA0059188	Surface Water		Surface Water					Chronic	50	0	2.0E-3
								Algae	1.4	0	7.1E-2
				20	1.4E-5	0	1.4	Acute	1,342	0	1.1E-3
								Chronic	50	0	2.8E-2
								Algae	1.4	0	1.1
OES: Other Commercial Uses											
Union Station North Wing Office Building Denver, CO NPDES: COG315293	Surface Water	Surrogate: Industrial POTW	Surface Water	250	3.0E-3	0	0.4	Acute	1,342	0	2.9E-4
								Chronic	50	0	7.8E-3
								Algae	1.4	4	0.3
				20	3.6E-2	0	4.6	Acute	1,342	0	3.5E-3
								Chronic	50	0	9.3E-2
								Algae	1.4	10	3.3
Confluence Park Apartments Denver, CO NPDES: COG315339	Surface Water	Surrogate: Industrial POTW	Surface Water	250	3.0E-4	0	3.9E-2	Acute	1,342	0	2.9E-5
								Chronic	50	0	7.7E-4
								Algae	1.4	0	2.8E-2
				20	3.7E-3	0	0.5	Acute	1,342	0	3.6E-4
								Chronic	50	0	9.6E-3
								Algae	1.4	0	0.3
Wynkoop Denver LLCP St Denver, CO NPDES: COG603115	Surface Water	Surrogate: Industrial POTW	Surface Water	250	2.0E-4	0	2.6E-2	Acute	1,342	0	1.9E-5
								Chronic	50	0	5.2E-4
								Algae	1.4	0	1.8E-2
				20	1.9E-3	0	0.2	Acute	1,342	0	1.8E-4
								Chronic	50	0	4.8E-3
								Algae	1.4	0	0.2
100 Saint Paul Denver County, CO NPDES: COG315289	Surface Water	Surrogate: Industrial POTW	Surface Water	250	4.0E-5	0	5.2E-3	Acute	1,342	0	3.8E-6
								Chronic	50	0	1.0E-4
								Algae	1.4	0	3.7E-3
				20	5.3E-4	0	6.8E-2	Acute	1,342	0	5.1E-5
								Chronic	50	0	1.4E-3
								Algae	1.4	0	4.9E-2
BPI-Westminster, LLC(Owner)/Arc	Surface Water		Surface Water	250	3.0E-5	0	3.9E-3	Acute	1,342	0	2.9E-6
								Chronic	50	0	7.7E-5

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
adis (Op) Denver, CO NPDES: COG315146		Surrogate: Industrial POTW						Algae	1.4	0	2.8E-3
				20	4.3E-4	0	5.5E-2	Acute	1,342	0	4.1E-5
								Chronic	50	0	1.1E-3
								Algae	1.4	0	4.0E-2
Safeway Inc Denver, CO NPDES: COG315260	Surface Water	Surrogate: Industrial POTW	Surface Water	250	2.0E-5	0	2.6E-3	Acute	1,342	0	1.9E-6
								Chronic	50	0	5.2E-5
								Algae	1.4	0	1.8E-3
				20	2.0E-4	0	2.6E-2	Acute	1,342	0	1.9E-5
								Chronic	50	0	5.2E-4
								Algae	1.4	0	1.8E-2
Illinois Central Railroad Thompsonville, IL NPDES: IL0070696	Surface Water	Surrogate: Industrial POTW	Surface Water	250	1.0E-5	0	1.3E-3	Acute	1,342	0	9.6E-7
								Chronic	50	0	2.6E-5
								Algae	1.4	0	9.2E-4
				20	1.6E-4	0	2.1E-2	Acute	1,342	0	1.5E-5
								Chronic	50	0	4.1E-4
								Algae	1.4	0	1.5E-2
OES: Waste Handling, Disposal, Treatment, and Recycling											
Clean Harbors Deer Park LLC La Porte, TX NPDES: TX0005941	Non-POTW WWT	Surrogate: Industrial POTW	Surface Water	250	0.4	80	9.1	Acute	1,342	0	6.7E-3
								Chronic	50	2	0.2
								Algae	1.4	172	6.4
				20	4.4	80	113	Acute	1,342	0	8.4E-2
								Chronic	50	7	2.3
								Algae	1.4	20	80
Clean Harbors El Dorado LLC El Dorado, AR NPDES: AR0037800	Non-POTW WWT	Surrogate: Industrial POTW	Surface Water	250	4.0E-2	80	1.0	Acute	1,342	0	7.7E-4
								Chronic	50	0	2.1E-2
								Algae	1.4	24	0.7
				20	0.5	80	12	Acute	1,342	0	8.8E-3
								Chronic	50	0	0.2
								Algae	1.4	15	8.5
Clean Harbors Recycling Services of Ohio LLC Hebron, OH	POTW	Receiving Facility: OH0021539	Surface Water	250	3.0E-5	80	3.2E-4	Acute	1,342	0	2.4E-7
								Chronic	50	0	6.4E-6

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
NPDES: None (FRS 110070118494)								Algae	1.4	0	2.3E-4
Clean Water Of New York Inc Staten Island, NY NPDES: NY0200484	Surface Water	Surrogate: Industrial POTW SIC code	Surface Water	250	4.0E-3	0	0.5	Acute	1,342	0	3.9E-4
								Chronic	50	0	1.0E-2
								Algae	1.4	7	0.4
				20	4.7E-2	0	6.1	Acute	1,342	0	4.5E-3
								Chronic	50	0	0.1
								Algae	1.4	11	4.3
Clifford G Higgins Disposal Service Inc SLF Kingston, NJ NPDES: NJG160946	Surface Water	Surrogate: Industrial POTW SIC code	Surface Water	250	2.0E-4	0	2.6E-2	Acute	1,342	0	1.9E-5
								Chronic	50	0	5.2E-4
								Algae	1.4	0	1.8E-2
				20	2.5E-3	0	0.3	Acute	1,342	0	2.4E-4
								Chronic	50	0	6.4E-3
								Algae	1.4	0	0.2
Durez North Tonawanda Occidental Chemical Corporation North Tonawanda, NY NPDES: NY0001198	Surface Water	NY0001198	Surface Water	250	1.0E-4	0	5.3E-2	Acute	1,342	0	4.0E-5
								Chronic	50	0	1.1E-3
								Algae	1.4	0	3.8E-2
				20	5.0E-4	0	0.3	Acute	1,342	0	2.0E-4
								Chronic	50	0	5.4E-3
								Algae	1.4	0	0.2
Heritage Thermal Services East Liverpool, OH NPDES: OH0107298	POTW	Receiving Facility: OH0024970	Surface Water	250	3.6E-7	80	9.7E-9	Acute	1,342	0	7.2E-12
								Chronic	50	0	1.9E-10
								Algae	1.4	0	6.9E-9
Oiltanking Houston Inc Houston, TX NPDES: TX0091855	Surface Water	Surrogate location: TX0005941	Surface Water	250	3.0E-3	0	0.3	Acute	1,342	0	2.5E-4
								Chronic	50	0	6.6E-3
								Algae	1.4	0	0.2
				20	4.2E-2	0	4.6	Acute	1,342	0	3.4E-3
								Chronic	50	0	9.2E-2
								Algae	1.4	1	3.3

Name, Location, and ID of Active Releaser Facility[a]	Release Media[b]	Modeled Facility or Industry Sector in E-FAST[c]	EFAST Waterbody Type[d]	Days of Release[e]	Release (kg/day)[f]	WWT removal %	7Q10 SWC (ppb)[g]	COC Type	COC (ppb)	Days of Exceedance (days/year)[h]	Risk Quotient
Pinewood Site Custodial Trust Pinewood, SC NPDES: SC0042170	Surface Water	Surrogate: Industrial POTW SIC code	Surface Water	250	1.0E-3	0	0.1	Acute	1,342	0	9.7E-5
								Chronic	50	0	2.6E-3
								Algae	1.4	0	9.3E-2
				20	7.5E-3	0	1.0	Acute	1,342	0	7.2E-4
								Chronic	50	0	1.9E-2
								Algae	1.4	2	0.7
Safety-Kleen Systems Inc Smithfield, KY NPDES: KY0098345	Non-POTW WWT	Surrogate: Industrial POTW SIC code (surrogate for receiving facility MDU000011)	Surface Water	250	1.4	80	35	Acute	1,342	0	2.6E-2
								Chronic	50	22	0.7
								Algae	1.4	235	25
				20	17	80	436	Acute	1,342	0	0.3
								Chronic	50	15	8.7
								Algae	1.4	20	311
Safety-Kleen Systems Inc, East Chicago, IN NPDES: Unknown	POTW	Receiving Facility: IN0022829	Surface Water	250	0.3	80	0.8	Acute	1,342	3	6.0E-4
								Chronic	50	10	1.6E-2
								Algae	1.4	148	0.6
Tier Environmental LLC Bedford, OH NPDES: None (FRS 110000388232)	POTW	Surrogate: Industrial POTW SIC code	Surface Water	250	0.1	80	3.1	Acute	1,342	0	2.3E-3
								Chronic	50	0	6.2E-2
								Algae	1.4	90	2.2
Tradebe Treatment & Recycling LLC East Chicago, IN NPDES: None (FRS 110070334821)	Non-POTW WWT	Surrogate: Industrial POTW SIC code (surrogate for FRS 110020159852	Surface Water	250	5.0E-3	80	0.1	Acute	1,342	0	9.7E-5
								Chronic	50	0	2.6E-3
								Algae	1.4	0	9.3E-2
				20	6.8E-2	80	1.8	Acute	1,342	0	1.3E-3
								Chronic	50	0	3.5E-2
								Algae	1.4	4	1.3

a. Facilities actively releasing PCE were identified via DMR, TRI and CDR 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 80% 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 industry sector. If available in TRI, the NPDES of the receiving facility is provided.
d. E-FAST 2014 (U.S. EPA 2014b) uses the "surface water" model for free-flowing water bodies such as rivers and streams, and the "still water" model for lakes, bays, and oceans. The surface water model uses stream flow values to calculate the concentration, whereas the still water model uses dilution factors. The dilution factor used in E-FAST is provided in parenthesis.
e. Modeling was conducted with the maximum days of release per year estimated. 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. The harmonic mean is not applicable for discharges to still water. For discharges to free-flowing water using an industry sector flow, the 10^{th} percentile harmonic mean is reported.
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. For discharges to free-flowing water using an industry sector flow, the 10^{th} percentile 7Q10 is reported.

4.5.2 Human Health Risk Conclusions

4.5.2.1 Summary of Risk Estimates for Inhalation and Dermal Exposures to Workers and ONUs

Table 4-112 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 both with and without assumed PPE. The PPE protection factor is listed in parentheticals beneath the risk value. The lowest APF/glove PF that eliminated risk (or APF 50/glove PF 20 if risk was not eliminated) was presented. The risk characterization is described in more detail in Section 4.2.2 and specific links to the exposure and risk characterization sections are listed in Table 4-112 in the column headed Occupational Exposure Scenario.

Of note, the risk summary below is based on the most sensitive acute and chronic non-cancer endpoints (neurotoxicity) as well as cancer. For the majority of exposure scenarios, when risks were identified for the chronic non-cancer endpoint (neurotoxicity), risks were also identified for kidney (urinary markers of nephrotoxicity) and immune system toxicity.

EPA made OES-specific determinations of assumed respirator use (see Section 4.2.2.2). When respirator use was considered plausible for the use scenario, the following PPE protection limits were considered for purposes of risk determination (Section 5.3), displayed in Table 4-111. Risk estimates are shown for all OES in Table 4-112 as a what-if scenario, even if those limits are not used for risk determination. Footnotes indicate for which individual OES respirator use is not assumed.

Table 4-111. PPE Protection Limits Considered for Risk Determination by Sector

Sector	APF	Glove PF
Manufacturing	50	20
Import/Processing/Disposal	25	20
Industrial	25	10
Commercial	10	5
Consumer	None	None

10098 **Table 4-112 Summary of Risk Estimates for Inhalation and Dermal Exposures to Workers by Condition of Use**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (bench-mark MOE = 10)	Chronic Non-cancer (bench-mark MOE = 100)	Cancer (bench-mark = 10^{-4})	Acute Non-cancer (bench-mark MOE = 10)	Chronic Non-cancer (bench-mark MOE = 100)	Cancer (bench-mark = 10^{-4})
Manufacture/ Domestic manufacture	Domestic manufacture	Section 2.4.1.6 – Manufacturing and Section 4.2.2.3 for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**1.9**	**8.7**	**6.1E-4**	19 (APF 10)	218 (APF 25)	6.1E-5 (APF 10)
					Central Tendency	154	701	5.9E-6	1538 (APF 10)	17,520 (APF 25)	5.9E-7 (APF 10)
				Inhalation 12 hr	High-End	16	**72**	7.5E-5	156 (APF 10)	716 (APF 10)	7.5E-6 (APF 10)
					Central Tendency	161	741	5.6E-6	1610 (APF 10)	7407 (APF 10)	5.6E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.9**	**8.7**	**6.1E-4**	N/A	N/A	N/A
					Central Tendency	154	701	5.9E-6	N/A	N/A	N/A
				Inhalation 12 hr	High-End	16	**72**	7.5E-5	N/A	N/A	N/A
					Central Tendency	161	741	5.6E-6	N/A	N/A	N/A
Manufacture/ Import	Import	Section 2.4.1.7 - Repackaging and Section 0 - 2 EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown. Repackaging for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**6.1**	**28**	**1.9E-4**	61 (APF 10)	278 (APF 10)	1.9E-5 (APF 10)
					Central Tendency	11.5	**52**	7.9E-5	115 (APF 10)	523 (APF 10)	7.9E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.1**	**28**	**1.9E-4**	N/A	N/A	N/A
					Central Tendency	11.5	**52**	7.9E-5	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
Processing/ Processing as a reactant/ intermediate	Intermediate in industrial gas manufacturing	Section 2.4.1.8– Processing as a Reactant and Section 4.2.2.5 - Processing as Reactant for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**1.9**	**8.7**	**6.1E-4**	19 (APF 10)	218 (APF 25)	6.1E-5 (APF 10)
					Central Tendency	154	701	5.9E-6	1538 (APF 10)	17520 (APF 25)	5.9E-7 (APF 10)
	Intermediate in basic organic chemical manufacturing			Inhalation 12 hr	High-End	15.6	**72**	7.5E-5	156 (APF 10)	716 (APF 10)	7.5E-6 (APF 10)
					Central Tendency	161	741	5.6E-6	1610 (APF 10)	7407 (APF10)	5.6E-7 (APF 10)
	Intermediate in petroleum refineries			Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
	Residual or byproduct reused as a reactant [a]				Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.9**	**8.7**	**6.1E-4**	N/A	N/A	N/A
					Central Tendency	154	701	5.9E-6	N/A	N/A	N/A
				Inhalation 12 hr	High-End	15.6	**72**	7.5E-5	N/A	N/A	N/A
					Central Tendency	161	741	5.6E-6	N/A	N/A	N/A
Processing/ Incorporated into formulation mixture or reaction product	Cleaning and degreasing products	Section 2.4.1.9 – Incorporation into Formulation, Mixture, or Reactant Product and Section 4.2.2.6 - Incorporation into Formulation, Mixture, or Reactant Product Based on Aerosol Packing for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.38**	**1.7**	**3.1E-3**	19 (APF 50)	**84** (APF 50)	6.2E-5 (APF 50)
	Adhesive and sealant products				Central Tendency	**0.60**	**2.7**	**1.5E-3**	30 (APF 50)	132 (APF 50)	3.0E-5 (APF 50)
	Paint and coating products			Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
	Other chemical products and preparations				Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.38**	**1.7**	**3.1E-3**	N/A	N/A	N/A
					Central Tendency	**0.60**	**2.7**	**1.5E-3**	N/A	N/A	N/A
		Section 2.4.1.9 – Incorporation into	Worker	Inhalation 8 hr	High-End	**1.9**	**92**	1.7E-5	19 (APF 10)	918 (APF 10)	1.7E-6 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Formulation, Mixture, or Reactant Product and Section 4.2.2.6 - Incorporation into Formulation, Mixture, or Reactant Product Based on Degreasing Solvent for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	**6.9**	328	4.7E-6	69 (APF 10)	3277 (APF 10)	4.7E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.9**	**92**	1.7E-5	N/A	N/A	N/A
					Central Tendency	**6.9**	328	4.7E-6	N/A	N/A	N/A
		Section 2.4.1.9 – Incorporation into Formulation, Mixture, or Reactant Product and Section 4.2.2.6 - Incorporation into Formulation, Mixture, or Reactant Product Based on Dry Cleaning Solvent for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.35**	**17**	9.1E-5	18 (APF 50)	169 (APF 10)	9.1E-6 (APF 10)
					Central Tendency	**1.3**	**60**	2.5E-5	63 (APF 50)	604 (APF 10)	2.5E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.35**	**17**	9.1E-5	N/A	N/A	N/A
					Central Tendency	**1.3**	**60**	2.5E-5	N/A	N/A	N/A
		Section 2.4.1.9 – Incorporation into Formulation, Mixture, or Reactant Product and Section 4.2.2.6 - Incorporation into Formulation, Mixture, or Reactant Product Based on Miscellaneous for inhalation risks and Section 4.2.34.2.3.1 for dermal risks	Worker	Inhalation 8 hr	High-End	**3.5**	169	9.1E-6	89 (APF 25)	1693 (APF 10)	9.1E-7 (APF 10)
					Central Tendency	13	602	2.6E-6	315 (APF 25	6017 (APF 10)	2.6E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**3.5**	169	9.1E-6	N/A	N/A	N/A
					Central Tendency	13	602	2.6E-6	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
Processing/ Incorporated into articles	Plastic and rubber products	Not assessed – after further review, EPA determined that PCE is not incorporated into plastic articles but rather is used as a degreasing solvent at plastic manufacture sites which are assessed in Sections 2.4.1.10 through 2.4.1.15									
Processing/ Repackaging	Solvent for cleaning or degreasing Intermediate	Section 2.4.1.7 – Repackaging and Section 0 - 2 EPA is unable to estimate ONU exposures separately from workers. EPA used worker central tendency values as a surrogate to assess risk for ONUs; however, the statistical representativeness of this value for ONUs is unknown. Repackaging for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**6.1**	**28**	**1.9E-4**	61 (APF 10)	278 (APF 10)	1.9E-5 (APF 10)
					Central Tendency	11.5	**52**	7.9E-5	115 (APF 10)	523 (APF 10)	7.9E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.1**	**28**	**1.9E-4**	N/A	N/A	N/A
					Central Tendency	11.5	**52**	7.9E-5	N/A	N/A	N/A
Processing/ Recycling	Recycling	Section 2.4.1.26 – Waste Handling, Disposal, Treatment, and Recycling and Section 4.2.2.23 - Waste Handling, Disposal, Treatment, and Recycling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	139	633	8.4E-6	1390 (APF 10)	6331 (APF 10)	8.4E-7 (APF 10)
					Central Tendency	628	2862	1.4E-6	6284 (APF 10)	28,624 (APF 10)	1.4E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	139	633	8.4E-6	N/A	N/A	N/A
					Central Tendency	628	2862	1.4E-6	N/A	N/A	N/A
Distribution in commerce	Distribution	Activities related to distribution (e.g., loading, unloading) are considered throughout the life cycle, rather than using a single distribution scenario.									

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
Industrial use/ Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)	Section 2.4.1.10 – Batch Open-Top Vapor Degreasing and Section 4.2.2.7 - Batch Open-Top Vapor Degreasing for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.16**	**0.71**	**7.5E-3**	**7.8** (APF 50)	**35** (APF 50)	**1.5E-4** (APF 50)
					Central Tendency	**2.4**	**11**	**3.8E-4**	119 (APF 50)	542 (APF 50)	7.6E-6 (APF 50)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.96**	**4.4**	**1.2E-3**	N/A	N/A	N/A
					Central Tendency	**8.3**	**38**	**1.1E-4**	N/A	N/A	N/A
		Section 2.4.1.11 – Batch Closed-Loop Vapor Degreasing And Section 4.2.2.8 - Batch Closed-Loop Vapor Degreasing for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	20	**90**	5.9E-5	198 (APF 10)	238 (APF 10)	5.9E-6 (APF 10)
					Central Tendency	69	316	1.3E-5	693 (APF 10)	348 (APF 10)	1.3E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	52	238	2.2E-5	N/A	N/A	N/A
					Central Tendency	76	348	1.2E-5	N/A	N/A	N/A
	In-line vapor degreaser (e.g., conveyorized, web cleaner)	Section 2.4.1.12– Conveyorized Vapor Degreasing and Section 4.2.2.9 - Conveyorized Vapor Degreasing for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.03**	**0.12**	**3.5E-2**	**1.3** (APF 50)	**6.1** (APF 50)	**7.0E-4** (APF 50)
					Central Tendency	**0.06**	**0.29**	**1.3E-2**	**3.2** (APF 50)	**15** (APF 50)	**2.7E-** (APF 50)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs		High-End	**0.04**	**0.18**	**2.3E-2**	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
				Inhalation 8 hr	Central Tendency	**0.12**	**0.56**	**7.0E-3**	N/A	N/A	N/A
		Section 2.4.1.13 - Web Degreasing and Section 4.2.2.10 - Web Degreasing for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**2.8**	**13**	**3.3E-4**	139 (APF 10)	126 (APF 10)	3.3E-05 (APF 10)
					Central Tendency	**8.2**	**37**	**1.1E-4**	409 (APF 10)	373 (APF 10)	1.1E-05 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**4.3**	**19**	**2.1E-4**	N/A	N/A	N/A
					Central Tendency	16	**71**	5.5E-5	N/A	N/A	N/A
	Cold cleaner	Section 2.4.1.14– Cold Cleaning and Section 4.2.2.11 - Cold Cleaning Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**1.2**	**5.5**	**9.7E-4**	12 (APF 10)	138 (APF 25)	9.7E-5 (APF 10)
					Central Tendency	**3.6**	**16**	**2.5E-4**	36 (APF 10)	407 (APF 25)	2.4E-05 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.2**	**5.5**	**9.7E-4**	N/A	N/A	N/A
					Central Tendency	**3.6**	**16**	**2.5E-4**	N/A	N/A	N/A
		Section 2.4.1.14– Cold Cleaning and Section 4.2.2.11 - Cold Cleaning Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**3.3**	**15**	**2.6E-4**	33 (APF 10)	148 (APF 10)	2.6E-5 (APF 10)
					Central Tendency	2086	9501	4.1E-7	20857 (APF 10)	95,007 (APF 10)	4.1E-8 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
			ONUs	Inhalation 8 hr	High-End	**6.4**	**29**	**1.3E-4**	N/A	N/A	N/A
					Central Tendency	4029	18,354	2.1E-7	N/A	N/A	N/A
	Aerosol spray degreaser/cleaner	Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	32 (APF 50)	146 (APF 50)	3.6E-5 (APF 50)
					Central Tendency	**3.5**	**16**	**2.6E-4**	174 (APF 50)	792 (APF 50)	5.2E-6 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	N/A	N/A	N/A
					Central Tendency	**3.5**	**16**	**2.6E-4**	N/A	N/A	N/A
		Section 2.4.1.15 - Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.29**	**1.3**	**3.1E-3**	15 (APF 50)	**66** (APF 50)	6.3E-5 (APF 50)
					Central Tendency	**0.91**	**4.2**	**9.4E-4**	46 (APF 50)	208 (APF 50)	1.9E-5 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.8**	**31**	**1.4E-4**	N/A	N/A	N/A
					Central Tendency	50	260	2.0E-5	N/A	N/A	N/A
	Dry cleaning solvent	Section 2.4.1.16 – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c]	Worker	Inhalation 8 hr	High-End	**0.26**	**1.0**	**5.4E-3**	13 (APF 50)	**50** (APF 50)	**1.1E-4** (APF 50)
	Spot cleaner				Central Tendency	**1.4**	**6.1**	**6.8E-4**	69 (APF 50)	303 (APF 50)	1.4E-5 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	14	**56**	9.5E-5	N/A	N/A	N/A
					Central Tendency		**64**	6.5E-5	N/A	N/A	N/A
		Section 2.4.1.16– Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.17**	**0.50**	**8.1E-2**	**8.4** (APF 50)	**25** (APF 50)	**1.6E-4** (APF 50)
					Central Tendency	**3.6**	**11**	**3.8E-4**	179 (APF 50)	527 (APF 50)	7.6E-6 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**3.2**	**9.5**	**4.3E-4**	N/A	N/A	N/A
					Central Tendency	46	136	2.9E-5	N/A	N/A	N/A
		Section 2.4.1.16– Dry Cleaning and Spot Cleaning 4th/5th Gen Only Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.89**	**3.5**	**1.5E-3**	45 (APF 50)	174 (APF 50)	3.1E-5 (APF 50)
					Central Tendency	**5.1**	**23**	**1.8E-4**	256 (APF 50)	1129 (APF 50)	3.7E-6 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	41	158	3.4E-5	N/A	N/A	N/A
					Central Tendency	358	1582	2.6E-6	N/A	N/A	N/A
Industrial use/ Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Section 2.4.1.15 - Aerosol Degreasing and Aerosol Lubricants	Worker	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	32 (APF 50)	146 (APF 50)	3.6E-5 (APF 50)
					Central Tendency	**3.5**	**16**	**2.6E-4**	174 (APF 50)	792 (APF 50)	5.2E-6 (APF 50)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks		Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	N/A	N/A	N/A
					Central Tendency	**3.5**	**16**	**2.6E-4**	N/A	N/A	N/A
		Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.29**	**1.3**	**3.1E-3**	15 (APF 50)	**66** (APF 50)	6.3E-5 (APF 50)
					Central Tendency	**0.91**	**4.2**	**9.4E-4**	46 (APF 50)	208 (APF 50)	1.9E-5 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.8**	**31**	**1.4E-4**	N/A	N/A	N/A
					Central Tendency	50	260	2.0E-5	N/A	N/A	N/A
		Section2.4.1.20– Metalworking Fluids and Section 4.2.2.17 - Metalworking Fluids [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	239	1087	4.9E-6	2387 (APF 10)	10,875 (APF 10)	4.9E-7 (APF 10)
					Central Tendency	869	3960	1.0E-6	8692 (APF 10)	39,595 (APF 10)	1.0E-7 (APF 10)
				Dermal	High-End	12	**26**	**2.5E-4**	60 (PF 5)	128 (PF 5)	5.0E-5 (PF 5)
					Central Tendency	36	**77**	6.4E-5	181 (PF 5)	384 (PF 5)	1.3E-5 (PF 5)
			ONUs	Inhalation 8 hr	High-End	239	1087	4.9E-6	N/A	N/A	N/A
					Central Tendency	869	3960	1.0E-6	N/A	N/A	N/A
	Solvent-based adhesives and sealants	Section 2.4.1.17– Adhesive, Sealants, Paints, and	Worker	Inhalation 8 hr	High-End	**6.2**	**28**	**1.9E-4**	62 (APF 10)	281 (APF 10)	1.9E-5 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
Industrial use/ Adhesives and sealants		Coatings and Section 4.2.2.14 Adhesives, Sealants, Paints, and Coatings Based on Adhesives for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	57	257	1.6E-5	565 (APF 10)	2574 (APF 10)	1.6E-6 (APF 10)
				Dermal Commercial use	High-End	**0.98**	**2.1**	**3.0E-3**	20 (PF 20)	**42** (PF 20)	**1.5E-4** (PF 20)
					Central Tendency	**3.0**	**6.3**	**7.8E-4**	59 (PF 20)	126 (PF 20)	3.9E-5 (PF 20)
				Dermal Industrial use	High-End	**1.5**	**3.2**	**2.0E-3**	30 (PF 20)	**64** (PF 20)	9.9E-5 (PF 20)
					Central Tendency	**4.5**	**9.6**	**5.1E-4**	90 (PF 20)	192 (PF 20)	2.6E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.2**	**28**	**1.9E-4**	N/A	N/A	N/A
					Central Tendency	57	257	1.6E-5	N/A	N/A	N/A
Industrial use/ Paints and coatings including paint and coating removers	Solvent-based paints and coatings, including for chemical milling	Section 2.4.1.17– Adhesive, Sealants, Paints, and Coatings and Section 4.2.2.14 Adhesives, Sealants, Paints, and Coatings Based on Paints/ Coatings for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**1.1**	**5.0**	**1.1E-3**	11 (APF 10)	125 (APF 25)	4.3E-5 (APF 25)
					Central Tendency	21	**98**	4.2E-5	214 (APF 10)	2440 (APF 25)	1.7E-6 (APF 25)
				Dermal Commercial use	High-End	**0.98**	**2.1**	**3.0E-3**	20 (PF 20)	**42** (PF 20)	**1.5E-4** (PF 20)
					Central Tendency	**3.0**	**6.3**	**7.8E-4**	59 (PF 20)	126 (PF 20)	3.9E-5 (PF 20)
				Dermal Industrial use	High-End	**1.5**	**3.2**	**2.0E-3**	30 (PF 20)	**64** (PF 20)	9.9E-5 (PF 20)
					Central Tendency	**4.5**	**9.6**	**5.1E-4**	90 (PF 20)	192 (PF 20)	2.6E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.1**	**5.0**	**1.1E-3**	N/A	N/A	N/A
					Central Tendency	21	**98**	4.2E-5	N/A	N/A	N/A
		Section 2.4.1.18 – Maskant for Chemical Milling and Section 4.2.2.15 - Maskant for Chemical	Worker	Inhalation 8 hr	High-End	**2.4**	**11**	**4.9E-4**	24 (APF 10)	108 (APF 10)	4.9E-5 (APF 10)
					Central Tendency	**4.1**	**19**	**2.2E-4**	41 (APF 10)	188 (APF 10)	2.2E-5 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (bench-mark MOE = 10)	Chronic Non-cancer (bench-mark MOE = 100)	Cancer (bench-mark = 10^{-4})	Acute Non-cancer (bench-mark MOE = 10)	Chronic Non-cancer (bench-mark MOE = 100)	Cancer (bench-mark = 10^{-4})
		Milling for inhalation risks and Section 4.2.3 for dermal risks		Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**2.4**	**11**	**4.9E-4**	N/A	N/A	N/A
					Central Tendency	**4.1**	**19**	**2.2E-4**	N/A	N/A	N/A
Industrial use/ Processing aids, not otherwise listed	Pesticide, fertilizer and other agricultural chemical manufacturing	Section 2.4.1.19 – Industrial Processing Aid And Section 4.2.2.16 - Industrial Processing Aid for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**4.2**	**19**	**2.8E-4**	42 (APF 10)	193 (APF 10)	2.8E-5 (APF 10)
					Central Tendency	83	380	1.1E-5	833 (APF 10)	3796 (APF 10)	1.1E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**4.2**	**19**	**2.8E-4**	N/A	N/A	N/A
					Central Tendency	83	380	1.1E-5	N/A	N/A	N/A
Industrial use/ Processing aids, specific to petroleum production	Catalyst regeneration in petrochemical manufacturing	Section 2.4.1.19 – Industrial Processing Aid And Section 4.2.2.16 - Industrial Processing Aid for inhalation risks and Section4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**4.2**	**19**	**2.8E-4**	42 (APF 10)	193 (APF 10)	2.8E-5 (APF 10)
					Central Tendency	83	380	1.1E-5	833 (APF 10)	3796 (APF 10)	1.1E-6 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**4.2**	**19**	**2.8E-4**	N/A	N/A	N/A
					Central Tendency	83	380	1.1E-5	N/A	N/A	N/A
Industrial use/ Other uses	Textile processing	Section 2.4.1.22 – Other Spot Cleaning/Spot	Worker	Inhalation 8 hr	High-End	22	**99**	5.4E-5	217 (APF 10)	987 (APF 10)	5.4E-6 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Removers (Including Carpet Cleaning) and Section 4.2.2.19 - Other Spot Cleaning/Spot Removers (Including Carpet Cleaning) [c] for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	29	133	3.1E-5	291 (APF 10)	1325 (APF 10)	3.1E-6 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	167	759	7.0E-6	N/A	N/A	N/A
					Central Tendency			5.4E-6			
		Section 2.4.1.23 – Other Industrial Uses and Section 4.2.2.20 - Other Industrial Uses for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	139	633	8.4E-6	1390 (APF 10)	6331 (APF 10)	8.4E-7 (APF 10)
					Central Tendency	628	2862	1.4E-6	6284 (APF 10)	28,624 (APF 10)	1.4E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	139	633	8.4E-6	N/A	N/A	N/A
					Central Tendency	628	2862	1.4E-6	N/A	N/A	N/A
	Wood furniture manufacturing	Section 2.4.1.23 – Other Industrial Uses and Section 4.2.2.20 - Other Industrial Uses for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	139	633	8.4E-6	1390 (APF 10)	6331 (APF 10)	8.4E-7 (APF 10)
					Central Tendency	628	2862	1.4E-6	6284 (APF 10)	28,624 (APF 10)	1.4E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	139	633	8.4E-6	N/A	N/A	N/A
					Central Tendency	628	2862	1.4E-6	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
	Laboratory chemicals	Section 2.4.1.25 – Laboratory Chemicals	N/A – qualitative assessment								
	Foundry applications	Section 2.4.1.23 – Other Industrial Uses and Section 4.2.2.20 - Other Industrial Uses for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	139	633	8.4E-6	1390 (APF 10)	6331 (APF 10)	8.4E-7 (APF 10)
					Central Tendency	628	2862	1.4E-6	6284 (APF 10)	28,624 (APF 10)	1.4E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51** (PF 20)	**1.2E-4** (PF 20)
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20)	154 (PF 20)	3.2E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	139	633	8.4E-6	N/A	N/A	N/A
					Central Tendency	628	2862	1.4E-6	N/A	N/A	N/A
Commercial use/ Cleaning and furniture care products	Cleaners and degreasers (other)	Section 2.4.1.21 – Wipe Cleaning and Metal/Stone Polishes and Section 4.2.2.18 - Wipe Cleaning and Metal/Stone Polishes [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.02**	**0.10**	**5.3E-2**	**1.1** (APF 50)	**5.0** (APF 50)	**1.1E-3** (APF 50)
					Central Tendency	**0.04**	**0.17**	**2.4E-2**	**1.9** (APF 50)	**8.6** (APF 50)	**4.8E-4** (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.22**	**0.98**	**5.4E-3**	N/A	N/A	N/A
					Central Tendency	229	1043	4.0E-6	N/A	N/A	N/A
		Section 2.4.1.22 – Other Spot Cleaning/Spot Removers (Including Carpet Cleaning) and Section 4.2.2.19 - Other Spot Cleaning/Spot Removers (Including Carpet Cleaning) [c] for inhalation	Worker	Inhalation 8 hr	High-End	22	**99**	5.4E-5	217 (APF 10)	987 (APF 10)	5.4E-6 (APF 10)
					Central Tendency	29	133	3.1E-5	291 (APF 10)	1325 (APF 10)	3.1E-6 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		risks and Section 4.2.3 for dermal risks	ONUs	Inhalation 8 hr	High-End	167	759	7.0E-6	N/A	N/A	N/A
					Central Tendency			5.4E-6			
		Section 2.4.1.24 – Other Commercial Uses and Section 4.2.2.21 - Other Commercial Uses Based on Mold Release [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	25	114	4.7E-5	250 (APF10)	1139 (APF 10)	4.7E-6 (APF 10)
					Central Tendency	50	228	1.8E-5	500 (APF10)	2278 (APF 10)	1.8E-6 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	25	114	4.7E-5	N/A	N/A	N/A
					Central Tendency	50	228	1.8E-5	N/A	N/A	N/A
	Dry cleaning solvent	Section 2.4.1.16 – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.26**	**1.0**	**5.4E-3**	13 (APF 50)	**50** (APF 50)	**1.1E-4** (APF 50)
	Spot cleaner				Central Tendency	**1.4**	**6.1**	**6.8E-4**	69 (APF 50)	303 (APF 50)	1.4E-5 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	14	**56**	9.5E-5	N/A	N/A	N/A
					Central Tendency		**64**	6.5E-5	N/A	N/A	N/A
		Section 2.4.1.16 – Dry Cleaning and Spot Cleaning	Worker	Inhalation 8 hr	High-End	**0.17**	**0.50**	**8.1E-2**	**8.4** (APF 50)	**25** (APF 50)	**1.6E-4** (APF 50)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Post-2006 Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	**3.6**	**11**	**3.8E-4**	179 (APF 50)	527 (APF 50)	7.6E-6 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**3.2**	**9.5**	**4.3E-4**	N/A	N/A	N/A
					Central Tendency	46	136	2.9E-5	N/A	N/A	N/A
		Section 2.4.1.16 – Dry Cleaning and Spot Cleaning 4th/5th Gen Only Dry Cleaning (including spot cleaning) and Section 4.2.2.13 - Dry Cleaning and Spot Cleaning [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.89**	**3.5**	**1.5E-3**	45 (APF 50)	174 (APF 50)	3.1E-5 (APF 50)
					Central Tendency	**5.1**	**23**	**1.8E-4**	256 (APF 50)	1129 (APF 50)	3.7E-6 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	41	158	3.4E-5	N/A	N/A	N/A
					Central Tendency	358	1582	2.6E-6	N/A	N/A	N/A
	Automotive care products (e.g., engine degreaser and brake cleaner)	Section 2.4.1.15 – Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	32 (APF 50)	146 (APF 50)	3.6E-5 (APF 50)
	Aerosol cleaner				Central Tendency	**3.5**	**16**	**2.6E-4**	174 (APF 50)	792 (APF 50)	5.2E-6 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs		High-End	**0.64**	**2.9**	**1.8E-3**	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
				Inhalation 8 hr	Central Tendency	**3.5**	**16**	**2.6E-4**	N/A	N/A	N/A
		Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.29**	**1.3**	**3.1E-3**	15 (APF 50)	**66** (APF 50)	6.3E-5 (APF 50)
					Central Tendency	**0.91**	**4.2**	**9.4E-4**	46 (APF 50)	208 (APF 50)	1.9E-5 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.8**	**31**	**1.4E-4**	N/A	N/A	N/A
					Central Tendency	50	260	2.0E-5	N/A	N/A	N/A
	Non-aerosol cleaner	Section 2.4.1.21 – Wipe Cleaning and Metal/Stone Polishes and Section 4.2.2.18 Wipe Cleaning and Metal/Stone Polishes [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.02**	**0.10**	**5.3E-2**	**1.1** (APF 50)	**5.0** (APF 50)	**1.1E-3** (APF 50)
					Central Tendency	**0.04**	**0.17**	**2.4E-2**	**1.9** (APF 50)	**8.6** (APF 50)	**4.8E-4** (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.22**	**0.98**	**5.4E-3**	N/A	N/A	N/A
					Central Tendency	229	1043	4.0E-6	N/A	N/A	N/A
Commercial use/ Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Section 2.4.1.15 – Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure monitoring data for	Worker	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	32 (APF 50)	146 (APF 50)	3.6E-5 (APF 50)
					Central Tendency	**3.5**	**16**	**2.6E-4**	174 (APF 50)	792 (APF 50)	5.2E-6 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		inhalation risks and Section 4.2.3 for dermal risks	ONUs	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	N/A	N/A	N/A
					Central Tendency	**3.5**	**16**	**2.6E-4**	N/A	N/A	N/A
		Section 2.4.1.15 – Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.29**	**1.3**	**3.1E-3**	15 (APF 50)	**66** (APF 50)	6.3E-5 (APF 50)
					Central Tendency	**0.91**	**4.2**	**9.4E-4**	46 (APF 50)	208 (APF 50)	1.9E-5 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.8**	**31**	**1.4E-4**	N/A	N/A	N/A
					Central Tendency	50	260	2.0E-5	N/A	N/A	N/A
		Section 2.4.1.20 – Metalworking Fluids and Section 4.2.2.17 - Metalworking Fluids [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	239	1087	4.9E-6	2387 (APF 10)	10,875 (APF 10)	4.9E-7 (APF 10)
					Central Tendency	869	3960	1.0E-6	8692 (APF 10)	39,595 (APF 10)	1.0E-7 (APF 10)
				Dermal	High-End	12	**26**	**2.5E-4**	60 (PF 5)	128 (PF 5)	5.0E-5 (PF 5)
					Central Tendency	36	**77**	6.4E-5	181 (PF 5)	384 (PF 5)	1.3E-5 (PF 5)
			ONUs	Inhalation 8 hr	High-End	239	1087	4.9E-6	N/A	N/A	N/A
					Central Tendency	869	3960	1.0E-6	N/A	N/A	N/A
Commercial use/ Adhesives and sealant chemicals	Light repair adhesives	Section 2.4.1.17 – Adhesive, Sealants, Paints, and Coatings and Section 4.2.2.14 Adhesives, Sealants, Paints, and Coatings	Worker	Inhalation 8 hr	High-End	**6.2**	**28**	**1.9E-4**	62 (APF 10)	281 (APF 10)	1.9E-5 (APF 10)
					Central Tendency	57	257	1.6E-5	565 (APF 10)	2574 (APF 10)	1.6E-6 (APF 10)
					High-End	**0.98**	**2.1**	**3.0E-3**	20 (PF 20)	**42** (PF 20)	**1.5E-4** (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Based on Adhesives for inhalation risks and Section 4.2.3 for dermal risks		Dermal Commercial use	Central Tendency	**3.0**	**6.3**	**7.8E-4**	59 (PF 20)	126 (PF 20)	3.9E-5 (PF 20)
				Dermal Industrial use	High-End	**1.5**	**3.2**	**2.0E-3**	30 (PF 20)	**64** (PF 20)	9.9E-5 (PF 20)
					Central Tendency	**4.5**	**9.6**	**5.1E-4**	90 (PF 20)	192 (PF 20)	2.6E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.2**	**28**	**1.9E-4**	N/A	N/A	N/A
					Central Tendency	57	257	1.6E-5	N/A	N/A	N/A
Commercial use/ Paints and coatings	Solvent-based paints and coatings	Section 2.4.1.17– Adhesive, Sealants, Paints, and Coatings and Section 4.2.2.14 Adhesives, Sealants, Paints, and Coatings Based on Paints/ Coatings for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**1.1**	**5.0**	**1.1E-3**	11 (APF 10)	125 (APF 25)	4.3E-5 (APF 25)
					Central Tendency	21	**98**	4.2E-5	214 (APF 10)	2440 (APF 25)	1.7E-6 (APF 25)
				Dermal Commercial use	High-End	**0.98**	**2.1**	**3.0E-3**	20 (PF 20)	**42** (PF 20)	**1.5E-4** (PF 20)
					Central Tendency	**3.0**	**6.3**	**7.8E-4**	59 (PF 20)	126 (PF 20)	3.9E-5 (PF 20)
				Dermal Industrial use	High-End	**1.5**	**3.2**	**2.0E-3**	30 (PF 20)	**64** (PF 20)	9.9E-5 (PF 20)
					Central Tendency	**4.5**	**9.6**	**5.1E-4**	90 (PF 20)	192 (PF 20)	2.6E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**1.1**	**5.0**	**1.1E-3**	N/A	N/A	N/A
					Central Tendency	21	**98**	4.2E-5	N/A	N/A	N/A
Commercial use/ Other uses	Carpet cleaning	Section 2.4.1.22– Other Spot Cleaning/Spot Removers (Including Carpet Cleaning) and Section 4.2.2.19 - Other Spot Cleaning/Spot Removers (Including Carpet	Worker	Inhalation 8 hr	High-End	22	**99**	5.4E-5	217 (APF 10)	987 (APF 10)	5.4E-6 (APF 10)
					Central Tendency	29	133	3.1E-5	291 (APF 10)	1325 (APF 10)	3.1E-6 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		Cleaning) [c] for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	167	759	7.0E-6	N/A	N/A	N/A
					Central Tendency			5.4E-6	N/A	N/A	N/A
	Laboratory chemicals	Section 2.4.1.25– Laboratory Chemicals	N/A – qualitative assessment								
	Metal (e.g., stainless steel) and stone polishes	Section 2.4.1.21– Wipe Cleaning and Metal/Stone Polishes and Section 4.2.2.18 - Wipe Cleaning and Metal/Stone Polishes [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.02**	**0.10**	**5.3E-2**	**1.1** (APF 50)	**5.0** (APF 50)	**1.1E-3** (APF 50)
					Central Tendency	**0.04**	**0.17**	**2.4E-2**	**1.9** (APF 50)	**8.6** (APF 50)	**4.8E-4** (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.22**	**0.98**	**5.4E-3**	N/A	N/A	N/A
					Central Tendency	229	1043	4.0E-6	N/A	N/A	N/A
	Inks and ink removal products	Section 2.4.1.24– Other Commercial Uses and Section 4.2.2.21 - Other Commercial Uses Based on Printing [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.84**	**3.8**	**1.4E-3**	21 (APF 25)	192 (APF 50)	5.6E-5 (APF 25)
					Central Tendency	**2.6**	**12**	**3.5E-4**	65 (APF 25)	594 (APF 50)	1.4E-5 (APF 25)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.84**	**3.8**	**1.4E-3**	N/A	N/A	N/A
					Central Tendency	**2.6**	**12**	**3.5E-4**	N/A	N/A	N/A
		Section 2.4.1.24– Other Commercial Uses	Worker	Inhalation 8 hr	High-End	10,000	45,552	1.17E-7	100,000 (APF 10)	455,520 (APF 10)	1.17E-8 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
		and Section 4.2.2.21 - Other Commercial Uses Based on Photocopying [c] for inhalation risks and Section 4.2.3 for dermal risks			Central Tendency	26,667	121,472	3.40E-8	266,667 (APF 10)	1214,720 (APF 10)	3.40E-9 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	10,000	45,552	1.17E-7	N/A	N/A	N/A
					Central Tendency	26,667	121,472	3.40E-8	N/A	N/A	N/A
	Welding	Section 2.4.1.15 – Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure monitoring data for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	32 (APF 50)	146 (APF 50)	3.6E-5 (APF 50)
					Central Tendency	**3.5**	**16**	**2.6E-4**	174 (APF 50)	792 (APF 50)	5.2E-6 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.64**	**2.9**	**1.8E-3**	N/A	N/A	N/A
					Central Tendency	**3.5**	**16**	**2.6E-4**	N/A	N/A	N/A
		Section 2.4.1.15– Aerosol Degreasing and Aerosol Lubricants and Section 4.2.2.12 - Aerosol Degreasing and Aerosol Lubricants [c] Based on inhalation* exposure modeling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.29**	**1.3**	**3.1E-3**	15 (APF 50)	**66** (APF 50)	6.3E-5 (APF 50)
					Central Tendency	**0.91**	**4.2**	**9.4E-4**	46 (APF 50)	208 (APF 50)	1.9E-5 (APF 50)
				Dermal	High-End	**0.80**	**1.7**	**3.7E-3**	16 (PF 20)	**34** (PF 20)	**1.9E-4** (PF 20)
					Central Tendency	**2.4**	**5.1**	**9.6E-4**	48 (PF 20)	103 (PF 20)	4.8E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**6.8**	**31**	**1.4E-4**	N/A	N/A	N/A
					Central Tendency	50	260	2.0E-5	N/A	N/A	N/A

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
	Photographic film	Section 2.4.1.24 – Other Commercial Uses and Section 4.2.2.21 - Other Commercial Uses Based on Photographic Film [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	**0.089**	**0.40**	**1.3E-2**	**4.4** (APF 50)	**20** (APF 50)	**2.6E-4** (APF 50)
					Central Tendency	**0.79**	**3.6**	**1.1E-3**	40 (APF 50)	181 (APF 50)	2.3E-5 (APF 50)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	**0.089**	**0.40**	**1.3E-2**	N/A	N/A	N/A
					Central Tendency	**0.79**	**3.6**	**1.1E-3**	N/A	N/A	N/A
	Mold cleaning, release and protectant products	Section 2.4.1.24– Other Commercial Uses and Section 4.2.2.21 - Other Commercial Uses Based on Mold Release [c] for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	25	114	4.7E-5	250 (APF10)	1139 (APF 10)	4.7E-6 (APF 10)
					Central Tendency	50	228	1.8E-5	500 (APF10)	2278 (APF 10)	1.8E-6 (APF 10)
				Dermal	High-End	**0.79**	**1.7**	**4.4E-3**	16 (PF 20)	**34** (PF 20)	**2.2E-4** (PF 20)
					Central Tendency	**2.4**	**5.0**	**1.0E-3**	47 (PF 20)	101 (PF 20)	5.1E-5 (PF 20)
			ONUs	Inhalation 8 hr	High-End	25	114	4.7E-5	N/A	N/A	N/A
					Central Tendency	50	228	1.8E-5	N/A	N/A	N/A
Disposal/ Disposal	Industrial pre-treatment Industrial wastewater treatment Publicly owned treatment works (POTW) Underground injection Municipal landfill Hazardous landfill Other land disposal	Section 2.4.1.26– Waste Handling, Disposal, Treatment, and Recycling and Section 4.2.2.23 - Waste Handling, Disposal, Treatment, and Recycling for inhalation risks and Section 4.2.3 for dermal risks	Worker	Inhalation 8 hr	High-End	139	633	8.4E-6	1390 (APF 10)	6331 (APF 10)	8.4E-7 (APF 10)
					Central Tendency	628	2862	1.4E-6	6284 (APF 10)	28,624 (APF 10)	1.4E-7 (APF 10)
				Dermal	High-End	**1.2**	**2.6**	**2.5E-3**	24 (PF 20)	**51 (PF 20)**	**1.2E-4 (PF 20)**
					Central Tendency	**3.6**	**7.7**	**6.4E-4**	72 (PF 20))	154 (PF 20)	3.2E-5 (PF 20)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario and Exposure and Risk Section Numbers	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})	Acute Non-cancer (benchmark MOE = 10)	Chronic Non-cancer (benchmark MOE = 100)	Cancer (benchmark = 10^{-4})
	Municipal waste incinerator		ONUs	Inhalation 8 hr	High-End	139	633	8.4E-6	N/A	N/A	N/A
	Hazardous waste incinerator										
	Off-site waste transfer				Central Tendency	628	2862	1.4E-6	N/A	N/A	N/A

N/A = not assessed because ONUs are not assumed to be wearing PPE

* exposure scenarios with both inhalation exposure monitoring data and inhalation exposure modeling present risk calculations for both exposure results, note that all dermal exposures were modeled

[a] EPA assessed PCE as a reactant where it was produced as a byproduct from EDC manufacture and reused as a reactant

[b] Identified welding products were anti-spatter aerosol products; therefore, the assessment is included with the assessment of other aerosol products.

[c] EPA believes that small commercial facilities using PCE for aerosol degreasing and lubrication, dry cleaning, metalworking fluid, wipe cleaning, spot cleaning, or other commercial uses are unlikely to have a respiratory protection program. Therefore, the use of respirators is unlikely for workers in these facilities.

4.5.2.2 Summary of Risk Estimates for Inhalation and Dermal Exposures to Consumers and Bystanders

Table 4-113 summarizes the risk estimates for 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 Section 4.2.2 and specific links to the exposure and risk characterization sections are listed in Table 4-113 in the column headed Consumer Exposure Scenario.

Dermal risk estimates for all three consumer age groups (11-15 years, 16 – 20 years) and adults (≥21) are presented for each exposure scenario in Section 4.2.4. Overall the differences in the MOEs between age groups are approximately 10% or less and none of the exposure scenarios have MOEs close enough to the benchmark MOE to result in different risk results depending on the age group selected. Table 4-113 presents dermal exposures for the most sensitive age group (11-15 years).

10121 **Table 4-113 Summary of Risk Estimates for CNS effects from Acute Inhalation and Dermal Exposures to Consumers by Conditions**
10122 **of Use**

Category	Sub Category	Consumer Exposure Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 10)	Bystander MOE (benchmark MOE = 10)
Cleaning and furniture care products	Cleaners and degreasers (other)	Section 2.4.2.3.1- Aerosol Degreasers (includes: marine cleaner, degreaser, coil cleaner, electric motor cleaner, parts cleaner, cable cleaner, stainless steel polish, electrical/energized cleaner, wire and ignition demoisturants, electric motor cleaner; brake cleaners) Section 4.2.4.1 Aerosol Cleaners for Motors, Coils, Electrical Parts, Cables, Stainless Steel and marine Equipment, and Wire and Ignition Demoisturants	Inhalation 24-hr	Low Intensity User	**7.7**	39
				Moderate Intensity User	**0.2**	**0.8**
				High Intensity User	**1.3E-02**	**5.2E-02**
			Dermal[1]	Low Intensity User	35	N/A
				Moderate Intensity User	**0.6**	N/A
				High Intensity User	**5.8E-02**	N/A
	Dry cleaning solvent	Section 2.4.2.4.2 and Section 2.4.2.4.3- Dry Cleaned Articles Section 4.2.4.16 Dry Cleaned Clothing	Inhalation 24-hr	Stay-at-home Adult and Child	156	486
			Dermal[1]	Assumed dry cleaning Technology (Events, days after cleaning)	User, Half-Body MOE	User, Full-Body MOE
				2nd and 3rd genearation (single, 1 day)	**8.6**	**2.9**
				2nd and 3rd genearation (single, 2 day)	11	**3.7**
				2nd and 3rd genearation (single, 3 day)	15	**4.9**
				4nd and 5th genearation (single, 1 day)	49	16
				4nd and 5th genearation (single, 2 day)	64	21
				4nd and 5th genearation (single, 3 day)	83	28
				4nd and 5th genearation (repeat, 1 day)	16	**5.2**
				4nd and 5th genearation (repeat, 2 day)	20	**6.7**

Category	Sub Category	Consumer Exposure Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 10)	Bystander MOE (benchmark MOE = 10)
				4nd and 5th genearation (repeat, 3 day)	26	**8.7**
	Automotive care products (e.g., engine degreaser and brake cleaner)	Section 2.4.2.3.1 - Brake Cleaner Section 4.2.4.2 Aerosol Brake Cleaners	Inhalation 24-hr	Low Intensity User	**2.0**	**7.1**
				Moderate Intensity User	**0.2**	**0.8**
				High Intensity User	**4.5E-02**	**0.2**
			Dermal[1]	Low Intensity User	21	N/A
				Moderate Intensity User	**0.6**	N/A
				High Intensity User	**7.1E-02**	N/A
		Section 2.4.2.3.2 - Parts Cleaner Section 4.2.4.3 Parts Cleaners	Inhalation 24-hr	Low Intensity User	31	174
				Moderate Intensity User	**0.6**	**3.3**
				High Intensity User	**7.1E-02**	**0.4**
			Dermal[1]	Low Intensity User	**0.2**	N/A
				Moderate Intensity User	**1.3E-02**	N/A
				High Intensity User	**2.1E-02**	N/A
	Aerosol cleaner	Section 2.4.2.3.3 - Vandalism Mark & Stain Remover, Mold Cleaner, Weld Splatter Protectant Section 4.2.4.4 Vandalism Stain Removers, Mold Cleaners, and Weld Splatter Protectants	Inhalation 24-hr	Low Intensity User	15	77
				Moderate Intensity User	**0.3**	**1.6**
				High Intensity User	**1.3E-02**	**5.2E-02**
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
	Non-aerosol cleaner	Section 2.4.2.3.4 - Marble and Stone Polish (liquid) Section 4.2.4.5 Marble Polish	Inhalation 24-hr	Low Intensity User	**3.3**	17
				Moderate Intensity User	**6.8E-02**	**0.4**
				High Intensity User	**1.2E-02**	**5.0E-02**
			Dermal[1]	Low Intensity User	**3.5**	N/A
				Moderate Intensity User	**5.4E-02**	N/A
				High Intensity User	**5.8E-03**	N/A
		Section 2.4.2.3.5-Cutting Fluid	Inhalation 24-hr	Low Intensity User	**8.1**	39

Category	Sub Category	Consumer Exposure Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 10)	Bystander MOE (benchmark MOE = 10)
Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Section 4.2.4.6 Cutting Fluid		Moderate Intensity User	**1.3**	**6.7**
				High Intensity User	**0.1**	**0.6**
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
		Section 2.4.2.3.6- Spray Lubricant and Penetrating Oil Section 4.2.4.7 Lubricants and Penetrating Oils	Inhalation 24-hr	Low Intensity User	90	435
				Moderate Intensity User	**1.4**	**7.3**
				High Intensity User	**8.0E-02**	**0.4**
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
Adhesives and sealant chemicals	Adhesives for arts and crafts	Section 2.4.2.3.7-Adhesives (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant) Section 4.2.4.8 Adhesives	Inhalation 24-hr	Low Intensity User	62	29
				Moderate Intensity User	**2.3**	12
				High Intensity User	**0.1**	**0.5**
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
		Section 2.4.2.3.8-Livestock Grooming Adhesive Section 4.2.4.9 Livestock Grooming Adhesive	Inhalation 24-hr	Low Intensity User	112	539
				Moderate Intensity User	12	64
				High Intensity User	**0.8**	**3.0**
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
	Light repair adhesives	Section 2.4.2.3.9-Column Adhesive, Caulk and Sealant Section 4.2.4.10 Caulks, Sealants and Column Adhesives	Inhalation 24-hr	Low Intensity User	192	N/E
				Moderate Intensity User	**2.3**	N/E
				High Intensity User	**7.2E-02**	N/E
			Dermal[1]	Low Intensity User	N/E	N/A

Category	Sub Category	Consumer Exposure Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 10)	Bystander MOE (benchmark MOE = 10)
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
Paints and coatings	Solvent-based paints and coatings	Section 2.4.2.3.10-Outdoor Water Shield (liquid) Section 4.2.4.11 Outdoor Water Shield	Inhalation 24-hr	Low Intensity User	**7.6**	29
				Moderate Intensity User	**1.1**	**3.3**
				High Intensity User	**8.9E-02**	**0.4**
			Dermal[1]	Low Intensity User	**0.1**	N/A
				Moderate Intensity User	**2.5E-02**	N/A
				High Intensity User	**5.0E-02**	N/A
		Section 2.4.2.3.11 - Coatings and primers (aerosol) Section 4.2.4.12 Aerosol Coatings and Primers	Inhalation 24-hr	Low Intensity User	522	13448
				Moderate Intensity User	62	2143
				High Intensity User	**5.9**	209
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
		Section 2.4.2.3.12 - Rust Primer and Sealant (liquid) Section 4.2.4.13 Liquid Primers and Sealants	Inhalation 24-hr	Low Intensity User	10600	128556
				Moderate Intensity User	1163	12434
				High Intensity User	36	229
			Dermal[1]	Low Intensity User	**1.4**	N/A
				Moderate Intensity User	**1.8E-02**	N/A
				High Intensity User	**1.6E-02**	N/A
		Section 2.4.2.3.13-Metallic Overglaze Section 4.2.4.14 Metallic Overglaze	Inhalation 24-hr	Low Intensity User	4372	21107
				Moderate Intensity User	337	1674
				High Intensity User	21	81
			Dermal[1]	Low Intensity User	N/E	N/A
				Moderate Intensity User	N/E	N/A
				High Intensity User	N/E	N/A
Other Uses			Inhalation 24-hr	Low Intensity User	**1.1**	**5.3**

Category	Sub Category	Consumer Exposure Scenario	Exposure Route and Duration	Scenario Description	User MOE (benchmark MOE = 10)	Bystander MOE (benchmark MOE = 10)
	Metal (e.g., stainless steel) and stone polishes	Section 2.4.2.3.14-Marble and Stone Polish (wax) Section 4.2.4.15 Metal and Stone Polish		Moderate Intensity User	**0.2**	**0.8**
				High Intensity User	**1.5E-02**	**6.1E-02**
			Dermal[1]	Low Intensity User	**1.0**	N/A
				Moderate Intensity User	**0.1**	N/A
				High Intensity User	**1.3E-02**	N/A
	Inks and ink removal products	Ink removal combined under Aerosol Cleaner (vandalism and stain remover); use in printing inks discussed as "other use"				
	Welding	Identified welding products were anti-spatter aerosol products; therefore, the assessment is included with the assessment of other aerosol products combined under Aerosol Cleaner (weld splatter protectant)				
	Mold cleaning, release and protectant products	Combined under Aerosol Cleaner (mold cleaner)				

[1] Dermal exposure presented here are the youth age group (11-15 years). Three age groups are presented for each COU in section 4.2.4. Overall the differences in the MOEs between age groups are approximately 10% or less.
N/A = not assessed because bystanders are assumed to not have dermal contact with liquid PCE
N/E = not evaluated because dermal exposures to consumers are not expected for these uses because for the caulks, sealants and column adhesives consumer use the area of use was assumed to be outdoors, so bystander exposure was not estimated.

5 RISK DETERMINATION

5.1 Unreasonable Risk

5.1.1 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 estimate 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, (U.S. EPA 2017h)).[19]

Under TSCA, conditions of use are defined as the circumstances, as determined by the Administrator, under which the substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of. TSCA §3(4).

An unreasonable risk of injury to health may be indicated when health risks under the conditions of use are identified by comparing the estimated risks with the risk benchmarks and where the risks affect the general population or PESS, identified as relevant. For workers (which are one example of PESS), an unreasonable risk may be indicated when risks are not adequately addressed through expected use of workplace practices and exposure controls, including engineering controls or use of personal protective equipment (PPE). An unreasonable risk of injury to the environment may be indicated when environmental risks under the conditions of use are greater than environmental risk benchmarks. The risk estimates contribute to the evidence EPA uses to determine unreasonable risk.

EPA uses the term "indicates unreasonable risk" to indicate EPA concern for potential unreasonable risk. For non-cancer endpoints, "less than the MOE benchmark" is used to indicate potential unreasonable risk; this occurs if an MOE value is less than the benchmark MOE (e.g., MOE 0.3 < benchmark MOE 30). For cancer endpoints, EPA uses the term "greater than risk benchmark" to indicate potential unreasonable risk; this occurs, for example, if the lifetime cancer risk value is greater than 1 in 10,000 (e.g., cancer risk value is $5x10^{-2}$ which is greater than the standard range of acceptable cancer risk benchmarks of $1x10^{-4}$ to $1x10^{-6}$). For environmental endpoints, to indicate potential unreasonable risk EPA uses a risk quotient (RQ) value "greater than 1" (i.e., RQ >1). Conversely, EPA uses the term "does not indicate unreasonable risk" to indicate that it is unlikely that EPA has a concern for potential unreasonable risk. More details are described below.

[19] 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.

The degree of uncertainty surrounding the MOEs, cancer risk or RQs is a factor in determining whether or not unreasonable risk is present. Where uncertainty is low, and EPA has high confidence in the hazard and exposure characterizations (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), the Agency has a higher degree of confidence in its risk determination. EPA may also consider other risk factors, such as severity of endpoint, reversibility of effect, or exposure-related considerations, such as magnitude or number of exposures, in determining that the risks are unreasonable under the conditions of use. Where EPA has made assumptions in the scientific evaluation, whether or not those assumptions are protective will also be a consideration. Additionally, EPA considers the central tendency and high-end scenarios when determining the unreasonable risk. High-end risk estimates (i.e., 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.

EPA may make a no unreasonable risk determination for conditions of use where the substance's hazard and exposure potential, or where the risk-related factors described previously, lead EPA to determine that the risks are not unreasonable.

5.1.2 Risks to Human Health

5.1.2.1 Determining Non-Cancer Risks

Margins of exposure (MOEs) are used in EPA's risk evaluations as a starting point to estimate non-cancer risks for acute and chronic exposures. The non-cancer evaluation refers to potential 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. The benchmark for the MOE that is used 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. MOEs can provide a non-cancer risk profile by presenting a range of estimates for different non-cancer health effects for different exposure scenarios and are a widely recognized point estimate method for evaluating a range of potential non-cancer health risks from exposure to a chemical.

A calculated MOE that is less than the benchmark MOE indicates the possibility of non-cancer risk to human health. Whether those risks are unreasonable will depend upon other risk-related factors, such as severity of endpoint, reversibility of effect, exposure-related considerations (e.g., duration, magnitude, frequency of exposure, population exposed), and the confidence in the information used to inform the hazard and exposure values. If the calculated MOE is greater than the benchmark MOE, generally it is less likely that there is non-cancer risk.

Uncertainty factors (UFs) also play an important role in the risk estimation approach and in determining unreasonable risk. 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 in risk estimation and extrapolation for the MOE for specific endpoints and scenarios. However, these are often not the only uncertainties in a risk evaluation.

5.1.2.2 Determining Cancer Risks

EPA estimates cancer risks by determining 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 under specified use scenarios. 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., $1x10^{-6}$ to $1x10^{-4}$) depending on the subpopulation exposed. Generally, EPA considers 1×10^{-6} to $1x\ 10^{-4}$ as the appropriate benchmark for the general population, consumer users, and non-occupational PESS.[20]

For the subject chemical substance, the EPA, consistent with 2017 NIOSH guidance,[21] used 1×10^{-4} as the benchmark for the purposes of this risk determination for individuals in industrial and commercial work environments subject to Occupational Safety and Health Act (OSHA) requirements. It is important to note that $1x10^{-4}$ is not a bright line and EPA has discretion to make risk determinations based on other benchmarks as appropriate. It is important to note that exposure-related considerations (duration, magnitude, population exposed) can affect EPA's estimates of the excess lifetime cancer risk.

5.1.3 Determining Environmental Risk

To assess environmental risk, EPA identifies and evaluates environmental hazard data for aquatic, sediment-dwelling, and terrestrial organisms exposed under acute and chronic exposure conditions. The environmental risk includes any risks that exceed benchmarks to the aquatic environment from levels of the evaluated 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 and hazard data.

Environmental risks are estimated by calculating a RQ. The RQ is defined as:

$$\text{RQ} = \text{Environmental Concentration} / \text{Effect Level}$$

An RQ equal to 1 indicates that the exposures are the same as the concentration that causes effects. If the RQ is greater than 1, the exposure is greater than the effect concentration and there is potential for risk. If the RQ is less than 1, the exposure is less than the effect concentration and unreasonable risk is not likely. The Concentrations of Concern (COC) or hazard value for certain aquatic organisms are used to calculate RQs for acute and chronic exposures. For environmental risk, EPA is more likely to determine that there is unreasonable risk if the RQ exceeds 1 for the conditions of use being evaluated. Consistent with EPA's human health evaluations, the RQ is not treated as a bright line and other risk-based factors

[20] 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 (U.S. EPA 2017d). 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, (Federal Register 1989)).

[21] NIOSH Current intelligence bulletin 68: NIOSH chemical carcinogen policy (Whittaker et al. 2016).

may be considered (e.g., exposure scenario, uncertainty, severity of effect) for purposes of making a risk determination.

5.2 Risk Determinations for PCE

EPA's draft determinations of unreasonable risk for specific conditions of use of PCE listed below are based on environmental risks to aquatic organisms, health risks to workers and occupational non-users (ONUs) during occupational exposures, and health risks to consumers and bystanders during exposures to consumer uses.

For risks to the environment, as described in Section 4, EPA identified environmental risks to aquatic organisms (aquatic invertebrates, fish, and aquatic plants). In Table 5-1 and Section 5.3 below, the driver endpoints for EPA's preliminary determination of unreasonable risks to aquatic organisms are immobilization from acute exposure, growth effects from chronic exposure, and mortality to algae.

For risks to health, as described in Section 4, significant risks associated with more than one adverse effect (e.g. central nervous system, kidney, liver, immune system and developmental toxicity) were identified for particular conditions of use. The evaluation of cancer included estimates of risk of lung and liver tumors. In Table 5-1 and Section 5.3 below, EPA identifies neurotoxicity as the driver endpoint for the conditions of use that EPA has preliminarily determined present unreasonable risks. This is the effect that is most sensitive, and it is expected that addressing risks for this effect would address other identified risks.

- Workers: EPA evaluated workers' acute and chronic inhalation and dermal exposures for cancer and non-cancer risks and determined whether any risks are unreasonable. The drivers for EPA's determination of unreasonable risk for workers are neurotoxicity from acute and chronic inhalation and dermal exposures and cancer from chronic inhalation and dermal exposures. The determinations reflect the effects associated with the occupational exposures to PCE and incorporate consideration of assumed PPE. EPA expects there is compliance with federal and state laws, such as 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 consistent with the applicable SDSs. Estimated numbers of workers are in Section 2.4.1.2. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data were not reasonably available for the conditions of use.

- Occupational Non-Users (ONUs): EPA considers occupational non-users to be a subset of workers for whom the potential inhalation exposures may differ based on proximity to the exposure source. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance. EPA evaluated ONU acute and chronic inhalation exposures for cancer and non-cancer risks and determined whether any risks are unreasonable. The drivers for EPA's determination of unreasonable risks to ONUs are neurotoxicity from acute and chronic inhalation exposures and cancer from chronic inhalation exposures. The determinations reflect the effects associated with the occupational exposures to PCE and the assumed absence of PPE for ONUs. For dermal exposures, because ONUs are not expected to be dermally exposed to PCE, dermal risks to ONUs were not evaluated. For inhalation exposures, EPA, where possible, used monitoring or modeling information to estimate ONU exposures and to describe the risks separately from workers directly exposed. For some

conditions of use, EPA did not separately calculate risk estimates for ONUs and workers. For these conditions of use, there is uncertainty in the ONU risk estimates since the data or modeling did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency risk estimate for workers when determining ONU risk for those conditions of use for which ONU exposures were not separately estimated. Estimated numbers of occupational non-users are in Section 2.4.1.2.

- Consumers: EPA evaluated consumer acute inhalation and dermal exposures for non-cancer risks and determined whether any risks are unreasonable. The driver for EPA's determination of unreasonable risk is neurotoxicity from acute inhalation and dermal exposures. Generally, risks for consumers were indicated by acute inhalation and dermal exposure at low, medium, and high intensity use. For nearly half of the consumer uses, dermal exposure was not evaluated because PCE is a volatile solvent and is expected to quickly evaporate from skin. However, for certain consumer use scenarios product evaporation may be limited (e.g., handling/wiping using a solvent soaked rag). For these conditions of use, consumer dermal exposure was evaluated. Estimated numbers of consumers are in Section 2.4.2.2.

- Bystanders (from consumer uses): EPA evaluated bystander acute inhalation exposures for non-cancer risks and determined whether any risks are unreasonable. The driver for EPA's determination of unreasonable risk are neurotoxicity from acute inhalation exposure. Generally, risks for bystanders were indicated by acute inhalation exposure scenarios at low, medium, and high intensity use. Because bystanders are not expected to be dermally exposed to PCE, dermal non-cancer risks to bystanders were not evaluated. Estimated numbers of bystanders are in Section 2.4.2.2.

- Environmental risks**:** EPA determined that environmental exposures are expected for aquatic organisms for the conditions of use within the scope of the risk evaluation. EPA's evaluation assessed risks to aquatic organisms because PCE has low bioconcentration potential and moderate potential to accumulate in wastewater biosolids, soil, or sediment. The drivers for EPA's draft determination of unreasonable risks to aquatic organisms are immobilization from acute exposure, growth effects from chronic exposure, and mortality to algae. Algae was assessed separately and not incorporated into acute or chronic COCs, because durations normally considered acute for other species (e.g. 48, 72 hours) can encompass several generations of algae. Confidence in acute and chronic COCs for fish and invertebrates are high. The confidence in algae COC is medium given that the COC for algae is based on a single study and that data were only available for three algal species that may not represent the most sensitive species at a given site. Algae species tend to vary widely in their sensitivity to chemical pollutants and the sites assessed included both free-flowing water bodies (i.e., rivers and streams) and still water bodies (i.e., bays, lakes, and estuaries). Because current regulations do not require facilities to report the number of days associated with reported releases, EPA estimated site-specific surface water concentrations for discharges using upper and lower bounds for the range of predicted surface water concentrations. Details of EPA's estimates are in Section 4.1.2 and include consideration of the number of facility operating days per year, partial removal of PCE from industrial wastes or wastewater following treatment, and the impacts of any direct releases of wastes to surface waters without treatment. Site-specific surface water concentration estimates

for free-flowing water bodies were reported for both the 7Q10 (the lowest consecutive 7-day average flow during any 10-year period) and harmonic mean stream flows. Based on the estimated surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium. In general, the majority of releases of PCE to the aquatic environment do not exceed the aquatic benchmark. However, there are specific facilities for given COUs where estimated or reported releases result in modeled surface water concentrations that exceed the aquatic benchmark (see Section 4.1.2). While nine COUs had RQs ≥ 1, indicating risk, no risks were identified for aquatic organisms for all other COUs. EPA's preliminary determination regarding unreasonable risks for each of the nine COUs indicating risks is discussed further under the specific COU in Section 5.3.

As described below, risks to the general population were not evaluated.

- **General population:** Exposure pathways to the general population are covered by other statutes and consist of: the ambient air pathway (i.e., PCE is listed as a hazardous air pollutant (HAP) in the Clean Air Act (CAA)), the drinking water pathway (i.e., National Primary Drinking Water Regulations (NPDWRs) are promulgated for PCE under the Safe Drinking Water Act), ambient water pathways (i.e., PCE is a priority pollutant with recommended water quality criteria for protection of human health under the CWA), biosolids pathways (i.e., PCE has been identified in biosolids biennial reviews under the CWA), disposal pathways (PCE disposal is managed and prevented from further environmental release by RCRA and SDWA regulations). As described above, other environmental statutes administered by EPA adequately assess and effectively manage these exposures. EPA believes that the TSCA risk evaluation should focus on those exposure pathways associated with TSCA conditions of use that are not subject to the regulatory regimes discussed above because those pathways are likely to represent the greatest areas of concern to EPA. Therefore, EPA did not evaluate hazards or exposures to the general population in this risk evaluation, and there is no risk determination for the general population.

Table 5-1 below presents an overview of risk determinations by condition of use. An in-depth explanation of each determination follows the table, in Section 5.3. For the conditions of use where EPA found no unreasonable risk, EPA describes the estimated risks in Section 4.4 (or Section 2.4.3).

Table 5-1. Summary of Unreasonable Risk Determinations by Condition of Use

Condition of Use	Unreasonable Risk Determination
Manufacture – Domestic Manufacture	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present an unreasonable risk of injury to the environment (aquatic organisms).
Manufacture – Import (includes repackaging and loading/unloading)	**Presents an unreasonable risk of injury to health (workers and occupational non-users (ONUs)).** Does not present an unreasonable risk of injury to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Processing – Processing as a reactant/intermediate in industrial gas manufacturing; intermediate in basic organic chemical manufacturing; intermediate in petroleum refineries; residual or byproduct reused as a reactant	**Presents an unreasonable risk of injury to health (workers).** **Presents an unreasonable risk to the environment (aquatic organisms).** Does not present an unreasonable risk of injury to health (occupational non-users).
Processing – Incorporation into formulation, mixture or reaction product – Cleaning and degreasing products	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Processing – Incorporation into formulation, mixture or reaction product – Adhesive and sealant products	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Processing – Incorporation into formulation, mixture or reaction product – Paint and coating products	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Processing – Incorporation into formulation, mixture or reaction product – Other chemical products and preparations	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Processing – Repackaging – Solvents (for cleaning or degreasing); intermediate	**Presents an unreasonable risk of injury to health (workers and occupational non-users (ONUs)).** Does not present an unreasonable risk of injury to the environment (aquatic organisms).
Processing – Recycling	**Presents an unreasonable risk of injury to health (workers).** **Presents an unreasonable risk to the environment (aquatic organisms).** Does not present an unreasonable risk of injury to health (occupational non-users).

Condition of Use	Unreasonable Risk Determination
Distribution in Commerce	Does not present an unreasonable risk of injury to health (workers and occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (open-top)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (closed-loop)	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (conveyorized)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (web cleaner)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – Cold cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Industrial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning 4th/5th Gen Only Dry Cleaning	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Lubricants and greases – Lubricants and greases (aerosol lubricants)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Lubricants and greases – Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants)	Does not present an unreasonable risk of injury to health (workers and occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Adhesives and sealants – Solvent-based adhesives and sealants	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Paints and coatings – Solvent-based paints and coatings	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Paints and coatings – Maskant for Chemical Milling	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Processing aids, not otherwise listed – Pesticide, fertilizer and other agricultural chemical manufacturing	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Industrial use – Processing aids, specific to petroleum production – Catalyst regeneration in petrochemical manufacturing	**Presents an unreasonable risk of injury to health (workers).** **Presents an unreasonable risk to the environment (aquatic organisms).** Does not present an unreasonable risk of injury to health (occupational non-users).
Industrial use – Other uses – Textile processing (spot cleaning)	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Other uses – Textile processing (other)	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Other uses – Wood furniture manufacturing	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Other uses – Laboratory chemicals	Does not present an unreasonable risk of injury to health (workers and ONUs). Does not present unreasonable risk to the environment (aquatic organisms).
Industrial use – Other uses – Foundry applications	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (wipe cleaning)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (Other Spot Cleaning/Spot Removers (Including Carpet Cleaning))	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (Mold Release)	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning $4^{th}/5^{th}$ Gen Only Dry Cleaning	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Automotive care products (e.g., engine degreaser and brake cleaner)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Aerosol cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Cleaning and furniture care products – Non-aerosol cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Commercial Use – Lubricants and greases – Lubricants and greases (aerosol lubricants)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Lubricants and greases – Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)	Does not present an unreasonable risk of injury to health (workers and occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Adhesives and sealant chemicals – Light repair adhesives	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial Use – Paints and coatings – Solvent-based paints and coatings	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Carpet cleaning	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Laboratory chemicals	Does not present an unreasonable risk of injury to health (workers and occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Metal (e.g., stainless steel) and stone polishes	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Inks and ink removal products (based on printing)	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).

Condition of Use	Unreasonable Risk Determination
Commercial use – Other uses – Inks and ink removal products (based on photocopying)	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Welding	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Photographic film	**Presents an unreasonable risk of injury to health (workers and occupational non-users).** Does not present unreasonable risk to the environment (aquatic organisms).
Commercial use – Other uses – Mold cleaning, release and protectant products	**Presents an unreasonable risk of injury to health (workers).** Does not present an unreasonable risk of injury to health (occupational non-users). Does not present unreasonable risk to the environment (aquatic organisms).
Consumer Use – Cleaning and furniture care products – Cleaners and degreasers (other)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Cleaning and furniture care products – Dry cleaning solvent	**Presents an unreasonable risk of injury to health (consumers).** Does not present an unreasonable risk of injury to health (bystanders).
Consumer Use – Cleaning and furniture care products – Automotive care products (Brake cleaner)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Cleaning and furniture care products – Automotive care products (Parts cleaner)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Cleaning and furniture care products – Aerosol cleaner (Vandalism Mark & Stain Remover, Mold Cleaner, Weld Splatter Protectant)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Cleaning and furniture care products – Non-aerosol cleaner (e.g., marble and stone polish)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Lubricants and greases – Lubricants and greases (Cutting Fluid)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**

Condition of Use	Unreasonable Risk Determination
Consumer Use – Lubricants and greases – Lubricants and greases (Lubricants and Penetrating Oils)	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant)	**Presents an unreasonable risk of injury to health (consumers).** Does not present an unreasonable risk of injury to health (bystanders).
Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (Livestock Grooming Adhesive)	Does not present an unreasonable risk of injury to health (consumers and bystanders).
Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (Column Adhesive, Caulk and Sealant)	**Presents an unreasonable risk of injury to health (consumers).** Does not present an unreasonable risk of injury to health (bystanders).
Consumer Use – Paints and coatings – Solvent-based paints and coatings (Outdoor water shield (liquid))	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Paints and coatings – Solvent-based paints and coatings (Coatings and primers (aerosol))	Does not present an unreasonable risk of injury to health (consumers and bystanders).
Consumer Use – Paints and coatings – Solvent-based paints and coatings (Rust Primer and Sealant (liquid))	**Presents an unreasonable risk of injury to health (consumers).** Does not present an unreasonable risk of injury to health (bystanders).
Consumer Use – Paints and coatings – Solvent-based paints and coatings (Metallic Overglaze)	Does not present an unreasonable risk of injury to health (consumers and bystanders).
Consumer Use – Other Uses – Metal (e.g., stainless steel) and stone polishes	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Consumer Use – Other Uses – Inks and ink removal products; welding; mold cleaning, release and protectant products	**Presents an unreasonable risk of injury to health (consumers and bystanders).**
Disposal	**Presents an unreasonable risk of injury to health (workers).** **Presents an unreasonable risk to the environment (aquatic organisms).** Does not present an unreasonable risk of injury to health (occupational non-users).

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5.3 Detailed Risk Determinations by Condition of Use

5.3.1 Manufacture – Domestic manufacture

Section 6(b)(4)(A) unreasonable risk determination of domestic manufacture of PCE:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present an unreasonable risk of injury to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. EPA assessed inhalation exposures during manufacturing using monitoring data submitted by the Halogenated Solvents Industry Alliance (HSIA).

While EPA identified environmental risk for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the six facilities assessed as manufacturing PCE, there were two facilities with releases indicating risk to aquatic organisms (RQ ≥ 1 and 20 days or more of exceedance for algae). RQ values ranged from 2.64 (100 days of exceedance, indirect discharge) to 13.2 (189 days of exceedance, direct discharge). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. Exceedances occurred using direct and indirect release scenarios but were highest for direct release scenarios. Four of the six facilities assessed as manufacturing PCE did not have NPDES permits. EPA identified risk to algae from direct and indirect release of PCE to surface water from two of the facilities without NPDES permits. Lack of a NPDES permit increases the uncertainty in the surface water release estimate for a facility. Based on the surface water PCE concentration and COC

confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Manufacture	Domestic manufacture	Domestic manufacture

5.3.2 Manufacture – Import (includes repackaging and loading/unloading)

Section 6(b)(4)(A) unreasonable risk determination for manufacture – import of PCE (includes repackaging and loading/unloading):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users (ONUs)).**
- Does not present an unreasonable risk of injury to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate – ONUs:

- Neurotoxicity:
  - Chronic inhalation MOE 52 (central tendency). (Table 4-8)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for chronic inhalation exposures indicated non-cancer risk at the central tendency, while acute inhalation exposures did not indicate risk. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as importing or repackaging PCE, a single facility had releases indicating risk to aquatic organisms (RQ ≥ 1 and 20 days or more of exceedance for algae). RQ values were 20.62 (230 days of exceedance, indirect release) and 256.8 (20 days of exceedance, indirect release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. An exceedance from indirect release indicates that risk can exist even with waste water treatment if the rate of PCE release to surface water is high. One of the facilities assessed as manufacturing PCE did not have NPDES permits. EPA only identified risk to algae from the one facility lacking a NPDES permit. Lack of a NPDES permit increases the uncertainty in the surface water release estimate for a facility. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Manufacture	Import	Import

5.3.3 Processing – Processing as a reactant/intermediate in industrial gas manufacturing; intermediate in basic organic chemical manufacturing; intermediate in petroleum refineries; residual or byproduct reused as a reactant

Section 6(b)(4)(A) unreasonable risk determination for processing of PCE as a reactant/intermediate in industrial gas manufacturing; intermediate in basic organic chemical manufacturing; intermediate in petroleum refineries; and as a residual or byproduct and reused as a reactant:

- **Presents an unreasonable risk of injury to health (workers).**
- **Presents an unreasonable risk to the environment (aquatic organisms).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers and aquatic organisms:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.
- Growth effects to aquatic invertebrates from chronic exposure.
- Algae mortality from exposure.

Driver benchmarks – workers and aquatic organisms:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.
- Growth effects: Chronic (aquatic invertebrates) RQ ≥ 1.
- Mortality: Algae RQ ≥ 1.

Risk estimate - workers:

- Neurotoxicity:

- Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate for facilities with exceedances – aquatic organisms: (Table 4-110)

- Growth effects to aquatic invertebrates from chronic exposure:
  - RQ = 1.0 (chronic, aquatic invertebrates, 20 days of exceedance, direct release).
  - RQ = 2.0 (chronic, aquatic invertebrates, 20 days of exceedance, direct release).
- Algae mortality from exposure: (some facilities had exceedances for multiple scenarios)
  - RQ = 1.7 (algae, 350 days of exceedance, direct release).
  - RQ = 25 (algae, 20 days of exceedance, direct release).
  - RQ = 1.1 (algae, 29 days of exceedance, direct release).
  - RQ = 2.2 (algae, 350 days of exceedance, direct release).
  - RQ = 37 (algae, 20 days of exceedance, direct release).
  - RQ = 3.5 (algae, 193 days of exceedance, direct release).
  - RQ = 61 (algae, 20 days of exceedance, direct release).
  - RQ = 3.6 (algae, 350 days of exceedance, direct release).
  - RQ = 71 (algae, 20 days of exceedance, direct release).
  - RQ = 1.4 (algae, 67 days of exceedance, direct release).

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. Exposure is assessed using PCE personal breathing zone monitoring data collected at facilities manufacturing PCE as a surrogate for facilities processing PCE as reactant. The data were determined to have a "high" confidence rating through EPA's systematic review process. Although these data are not directly applicable to processing of PCE as a reactant, EPA expects a high degree of overlap of worker tasks at both manufacturing sites and sites processing PCE as a reactant. EPA assessed PCE as a reactant where it was produced as a byproduct from manufacture of 1,2-dichloroethane (CASRN 107-06-2) and reused as a reactant.

Environmental releases for this condition of use indicate chronic risk to aquatic organisms and risk to algae. Of the 18 facilities processing PCE as a reactant, six facilities had releases indicating risk to aquatic organisms (RQs ≥ 1 and 20 days or more of exceedance for aquatic organisms) with the highest RQ being 71 (algae, 20 days of exceedance, direct release). For the six facilities indicating risk, EPA identified risk to algae from all six facilities and chronic risk to aquatic organisms from two facilities. Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. All exceedances occurred using the direct release to surface water scenario. All of the facilities assessed as processing PCE as a reactant had NPDES permits. Based on the surface water PCE concentration and

COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Processing	Processing as a reactant or intermediate	• Intermediate in industrial gas manufacturing • Intermediate in basic organic chemical manufacturing • Intermediate in petroleum refineries • Residual or byproduct as a reactant

5.3.4 Processing – Incorporation into formulation, mixture or reaction product – Cleaning and degreasing products

Section 6(b)(4)(A) unreasonable risk determination for processing PCE for incorporation into a formulation, mixture, or reaction product – cleaning and degreasing products:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 1.3 (central tendency). (Table 4-13) (dry cleaning solvent)
  - Chronic inhalation MOEs 60 (central tendency). (Table 4-14) (dry cleaning solvent)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for

ONUs for acute and chronic inhalation exposures (central tendency) indicate risk. Two exposure scenarios, degreasing solvent and dry cleaning solvent, apply to this condition of use. EPA made its draft determination based on the dry cleaning solvent scenario, which was more representative of the condition of use. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as using PCE for incorporation into formulations, a single facility had releases indicating RQs ≥ 1 for acute, chronic, and algae risks. RQ values for algae were 96.84 (299 days of exceedance, indirect release) and 1,453.06 (20 days of exceedance, indirect release). RQ values for chronic effects to aquatic organisms were 2.71 (127 days of exceedance, indirect release) and 40.69 (20 days of exceedance, indirect release). The RQ value for the acute effect to aquatic organisms was 1.52 (acute, aquatic invertebrates, 20 days of exceedance, direct release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. The facility indicating risk had the highest surface water concentrations for all indirect releases evaluated (both maximum days of release and 20 days of release scenarios). The annual release at this facility was the highest of all active releasers, and generally was an order of magnitude higher than all other releases. The facility showing risk has a NPDES permit.

Life Cycle Stage	Category	Subcategory
Processing	Incorporated into formulation, mixture or reaction product	Cleaning and degreasing products

5.3.5 Processing – Incorporation into formulation, mixture or reaction product – Adhesive and sealant products

Section 6(b)(4)(A) unreasonable risk determination for processing PCE for incorporation into a formulation, mixture, or reaction product – adhesive and sealant products:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.

- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as using PCE for incorporation into formulations, a single facility had releases indicating RQs ≥ 1 for acute, chronic, and algae risks. RQ values for algae were 96.84 (299 days of exceedance, indirect release) and 1,453.06 (20 days of exceedance, indirect release). RQ values for chronic effects to aquatic organisms were 2.71 (127 days of exceedance, indirect release) and 40.69 (20 days of exceedance, indirect release). The RQ value for the acute effect to aquatic organisms was 1.52 (acute, aquatic invertebrates, 20 days of exceedance, direct release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. The facility indicating risk had the highest surface water concentrations for all indirect releases evaluated (both maximum days of release and 20 days of release scenarios). The annual release at this facility was the highest of all active releasers, and generally was an order of magnitude higher than all other releases. The facility showing risk has a NPDES permit.

Life Cycle Stage	Category	Subcategory
Processing	Incorporated into formulation, mixture or reaction product	Adhesive and sealant products

5.3.6 Processing – Incorporation into formulation, mixture or reaction product – Paint and coating products

Section 6(b)(4)(A) unreasonable risk determination for processing PCE for incorporation into a formulation, mixture, or reaction product – adhesive and sealant products:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as using PCE for incorporation into formulations, a single facility had releases indicating RQs ≥ 1 for acute, chronic, and algae risks. RQ values for algae were 96.84 (299 days of exceedance, indirect release) and 1,453.06 (20 days of exceedance, indirect release). RQ values for chronic effects to aquatic organisms were 2.71 (127 days of exceedance, indirect release) and 40.69 (20 days of exceedance, indirect release). The RQ value for the acute effect to aquatic organisms was 1.52 (acute, aquatic invertebrates, 20 days of exceedance, direct release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. The facility indicating risk had the highest surface water concentrations for all indirect releases evaluated (both maximum days of release and 20 days of release scenarios). The annual release at this facility was the highest of all active releasers, and generally was an order of magnitude higher than all other releases. The facility showing risk has a NPDES permit.

Life Cycle Stage	Category	Subcategory
Processing	Incorporated into formulation, mixture or reaction product	Paint and coating products

5.3.7 Processing – Incorporation into formulation, mixture or reaction product – Other chemical products and preparations

Section 6(b)(4)(A) unreasonable risk determination for processing PCE for incorporation into a formulation, mixture, or reaction product – other chemical products and preparations:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic inhalation MOEs 69 and 43 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-14) (aerosol packing)
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 0.6 (central tendency). (Table 4-13) (aerosol packing)
  - Chronic inhalation MOEs 2.7 (central tendency). (Table 4-14) (aerosol packing)
- Cancer (liver tumors):
  - Inhalation: 1.5E-03 (central tendency) without PPE. (Table 4-15) (aerosol packing)

Risk Considerations: For workers, all pathways of occupational exposure for this condition of use indicate risk, even with assumed respiratory protection (APF 25) and dermal protection (PF 20). Risk estimates for ONUs for acute, chronic, and cancer inhalation exposures (central tendency) indicate risk. EPA made its determination based on the aerosol packing scenario, which used personal breathing zone monitoring data. While aerosol packing may not be representative of other formulation, EPA has a high level of confidence in the assessed exposures based on the strength of the monitoring data. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as using PCE for incorporation into formulations, a single facility had releases indicating RQs ≥ 1 for acute, chronic, and algae risks. RQ values for algae were 96.84 (299 days of exceedance, indirect release) and 1,453.06 (20 days of exceedance, indirect release). RQ values for chronic effects to aquatic organisms were 2.71 (127 days of exceedance, indirect release) and 40.69 (20 days of exceedance, indirect release). The RQ value for the acute effect to aquatic organisms was 1.52 (acute, aquatic invertebrates, 20 days of exceedance, direct release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. The facility indicating risk had the highest surface water concentrations for all indirect releases evaluated (both maximum days of release and 20 days of release scenarios). The annual release at this facility was the highest of all active releasers, and generally was an order of magnitude higher than all other releases. The facility showing risk has a NPDES permit.

Life Cycle Stage	Category	Subcategory
Processing	Incorporated into formulation, mixture or reaction product	Other chemical products and preparations

5.3.8 Processing – Repackaging – Solvents (for cleaning or degreasing); intermediate

Section 6(b)(4)(A) unreasonable risk determination for processing PCE by repackaging – solvent for cleaning or degreasing; intermediate:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users (ONUs)).**
- Does not present an unreasonable risk of injury to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:

- Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate – ONUs:

- Neurotoxicity:
  - Chronic inhalation MOE 52 (central tendency). (Table 4-8)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for chronic inhalation exposures indicated non-cancer risk at the central tendency, while acute inhalation exposures did not indicate risk. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

While EPA identified environmental risks for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the four facilities assessed as importing or repackaging PCE, a single facility had releases indicating risk to aquatic organisms (RQ ≥ 1 and 20 days or more of exceedance for algae). RQ values were 20.62 (230 days of exceedance, indirect release) and 256.8 (20 days of exceedance, indirect release). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for indirect release. An exceedance from indirect release indicates that risk can exist even with waste water treatment if the rate of PCE release to surface water is high. One of the facilities assessed as manufacturing PCE did not have NPDES permits. EPA only identified risk to algae from the one facility lacking a NPDES permit. Lack of a NPDES permit increases the uncertainty in the surface water release estimate for a facility. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Processing	Repackaging	• Solvent for cleaning or degreasing • Intermediate

5.3.9 Processing – Recycling

Section 6(b)(4)(A) unreasonable risk determination for processing PCE by recycling:

- **Presents an unreasonable risk of injury to health (workers).**
- **Presents an unreasonable risk to the environment (aquatic organisms).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers and aquatic organisms:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.
- Growth effects to aquatic invertebrates from chronic exposure.
- Algae mortality from exposure.

Driver benchmarks – workers and aquatic organisms:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.
- Mortality: Algae RQ ≥ 1.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)

Risk estimate for facilities with exceedances – aquatic organisms: (Table 4-110)

- Algae mortality from exposure: (some facilities had exceedances for multiple scenarios)
  - RQ = 6.4 (algae, 172 days of exceedance, indirect release).
  - RQ = 80 (algae, 20 days of exceedance, indirect release).
  - RQ = 25 (algae, 235 days of exceedance, indirect release).
  - RQ = 311 (algae, 20 days of exceedance, indirect release).
  - RQ = 2.2 (algae, 90 days of exceedance, indirect release).

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), the dermal chronic non-cancer risk estimate (high-end) indicates risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

Environmental releases for this condition of use indicate chronic risk to aquatic organisms and risk to algae. Of the 13 facilities assessed for the waste handling, disposal, treatment, and recycling of PCE, three facilities had releases indicating risk to aquatic organisms (RQs ≥ 1 and 20 days of exceedance for algae). RQ values ranged from 2.2 (90 days of exceedance, indirect discharge) to 311 (20 days of exceedance, indirect discharge). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. Exceedances occurred using indirect release scenarios. An exceedance from indirect release indicates that risk can exist even with waste water treatment if the rate of PCE release to surface water is high. Four of the 13 facilities assessed for the waste handling, disposal, treatment, and recycling of PCE did not have NPDES permits. EPA identified risk to algae from indirect release of PCE to surface water from one of the facilities without a NPDES permit. Lack of a NPDES permit

increases the uncertainty in the surface water release estimate for a facility. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Processing	Recycling	Recycling

5.3.10 Distribution in Commerce

Section 6(b)(4)(A) unreasonable risk determination of distribution of PCE in commerce:

1 Does not present an unreasonable risk of injury to health (workers and occupational non-users).
2 Does not present unreasonable risk to the environment (aquatic organisms).

Risk Considerations: A quantitative evaluation of the distribution of PCE was not included in the risk evaluation because exposures and releases from distribution were considered within each condition of use.

Life Cycle Stage	Category	Subcategory
Distribution in commerce	Distribution	Distribution

5.3.11 Industrial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (open-top)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or decreasing) – batch vapor degreaser (open-top):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and chronic dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 60 and 3.9 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-16)
  - Chronic inhalation MOEs 271 and 18 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-17)
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):
  - Inhalation: 1.5E-05 and 3.0E-04 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-18)
  - Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (
  - Table 4-73)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 8.2 and 1.0 (central tendency and high-end). (Table 4-16)
  - Chronic inhalation MOEs 38 and 4.4 (central tendency and high-end). (Table 4-17)
- Cancer (liver tumors):
  - Inhalation: 1.1E-04 and 1.2E-03 (central tendency and high-end). (Table 4-18)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. For workers, non-cancer and cancer risk estimates for inhalation and dermal exposures indicate risks even with assumed respiratory protection (APF 25) and dermal protection (PF 10). EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Risk estimates for ONUs for acute (high-end), chronic (high-end and central tendency), and cancer (high-end) inhalation exposures indicate risk. EPA defined ONU as an employee who does not regularly handle PCE or operate the degreaser but performs work in the area around the degreaser. Samples from employees determined not to be operating the degreasing equipment were designated as ONU samples. EPA identified inhalation exposure monitoring data from NIOSH investigations at five sites using PCE as a degreasing solvent in OTVDs. Due to the large variety in shop types that may use PCE as a vapor degreasing solvent, there is some uncertainty in how representative these data are of a "typical" shop.

While EPA identified environmental risk for this COU, given the uncertainties in the data, EPA does not consider these risks unreasonable. Of the 17 facilities assessed for this COU, two facilities had releases indicating risk to risk to aquatic organisms (RQs ≥ 1 and 20 days or more of exceedance for algae). RQ values ranged from 2.3 (20 days of exceedance, direct discharge) to 55.5 (20 days of exceedance, direct discharge). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. The exceedance occurred for direct release. All of the facilities assessed as using PCE in open top vapor degreasing had NPDES permits. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)

5.3.12 Industrial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (closed-loop)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or decreasing) – batch vapor degreaser (closed-loop):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):
  - Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (
  - Table 4-73)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risks even with assumed dermal protection (PF 10). EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency or high-end. Worker samples were determined to be any sample taken on a person while performing the degreasing tasks. ONUs samples were determined to be any sample taken on a person in the same location as the degreaser but not performing the degreasing themselves. EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE as a degreasing solvent in batch closed-loop vapor degreasers. Due to the large variety in shop types that may use PCE as a vapor degreasing solvent, there is some uncertainty in how representative these data are of a "typical" shop. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)

5.3.13 Industrial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (conveyorized)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or degreasing) – in-line vapor degreaser (conveyorized):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and chronic dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 1.6 and 0.7 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-22)
  - Chronic inhalation MOEs 7.3 and 3.1 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-23)
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):
  - Inhalation: 5.4E-04 and 1.4E-03 (central tendency and high-end) with PPE (respirator APF 25). (Table 4-24)

Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (

  - Table 4-73)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 0.1 and 4.0E-02 (central tendency and high-end). (Table 4-22)
  - Chronic inhalation MOEs 0.6 and 0.2 (central tendency and high-end). (Table 4-23)
- Cancer (liver tumors):

- Inhalation: 7.0E-03 and 2.3E-02 (central tendency and high-end). (Table 4-24)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. For workers, non-cancer and cancer risk estimates for inhalation and dermal exposures indicate risks even with assumed respiratory protection (APF 25) and dermal protection (PF 10). EPA separately calculated risk estimates for ONUs and workers. Risks for ONUs for acute, chronic, and cancer inhalation exposures are indicated at the high-end and central tendency estimates. EPA assessed inhalation exposures during conveyorized degreasing using the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model. Workers' risk estimates are based on concentrations in the near-field where the conveyorized degreasing work occurs, and ONU exposures are based on concentrations in the far-field, away from the conveyorized degreaser. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	In-line vapor degreaser (e.g., conveyorized, web cleaner)

5.3.14 Industrial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (web degreaser)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or decreasing) – in-line vapor degreaser (web degreaser):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):

- Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (
- Table 4-73)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 16 and 4.3 (central tendency and high-end). (Table 4-25)
  - Chronic inhalation MOEs 71 and 19 (central tendency and high-end). (Table 4-26)
- Cancer (liver tumors):
  - Inhalation: 5.5E-05 and 2.1E-04 (central tendency and high-end). (Table 4-27)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). EPA separately calculated risk estimates for ONUs and workers. Risk estimates for ONUs for acute (high-end), chronic (high-end and central tendency), and cancer (high-end) inhalation exposures indicate risk. EPA assessed inhalation exposures during web degreasing using the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model. Workers' estimates are based on concentrations in the near-field where the web degreasing work occurs, and ONU exposures are based on concentrations in the far-field, away from the web degreaser. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	In-line vapor degreaser (e.g., conveyorized, web cleaner)

5.3.15 Industrial Use – Solvents (for cleaning or degreasing) – Cold cleaner

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or degreasing) – cold cleaner:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.

- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):
  - Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (
  - Table 4-73)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 3.6 (central tendency). (Table 4-28) (monitoring)
  - Chronic inhalation MOEs 16 (central tendency). (Table 4-29) (monitoring)
- Cancer (liver tumors):
  - Inhalation: 2.5E-04 (central tendency). (Table 4-30) (monitoring)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risks for ONUs for acute, chronic, and cancer inhalation exposures are indicated at the central tendency. For workers and ONUs, EPA used monitoring data to make the risk determination on the use of PCE in cold cleaners. While EPA modeled the use of PCE in cold cleaning, the model showed large variation in modeled results as a result of the large variation in unit emissions reported in NEI. There is uncertainty in the ONU risk estimate since the monitoring data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk from the monitoring data. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	Cold cleaner

5.3.16 Industrial Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or degreasing) – aerosol spray degreaser/cleaner:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
  - Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
  - Acute dermal MOEs 24 and 8.0 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-74)
  - Chronic dermal MOEs 51 and 17 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-75)
- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)
  - Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs.

Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. EPA separately evaluated risks to consumers from dry cleaned articles as part of the COU, Consumer Use – Cleaning and furniture care products – Dry cleaning solvent, in Section 5.3.52. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	Aerosol spray degreaser/cleaner

5.3.17 Industrial Use – Solvents (for cleaning or degreasing) – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or degreasing) – dry cleaning and spot cleaning post-2006 dry cleaning:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 1.4 and 0.3 (central tendency and high-end) without PPE. (Table 4-34) (monitoring)
  - Chronic inhalation MOEs 6.1 and 1.0 (central tendency and high-end) without PPE. (Table 4-35) (monitoring)
  - Acute dermal MOEs 24 and 7.9 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-77)
  - Chronic dermal MOEs 50 and 17 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 6.8E-04 and 5.4E-03 (central tendency and high-end) without PPE. (Table 4-36) (monitoring)

- Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 30 and 2.1 (central tendency and high-end). (Table 4-34) (modeling)
  - Chronic inhalation MOEs 136 and 9.5 (central tendency and high-end). (Table 4-35) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.9E-05 and 4.3E-04 (central tendency and high-end). (Table 4-36) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory PPE. While EPA does not assume routine use of respiratory PPE with this exposure scenario, risk was still present to workers with APF 50 for chronic inhalation at the high-end, for monitoring and modeled data. Because the monitoring data only contained one data point representing an ONU for this scenario, EPA made its determination on ONUs using modeled data. Modeled ONU exposures are based on concentrations in the far-field which corresponds to any area outside the near-field zones. Risk estimates for ONUs for acute (high-end), chronic (high-end and central tendency), and cancer (high-end) inhalation exposures indicate risk. EPA separately evaluated risks to consumers from dry cleaned articles as part of the COU, Consumer Use – Cleaning and furniture care products – Dry cleaning solvent, in Section 5.3.52. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	• Dry cleaning solvent • Spot cleaner

5.3.18 Industrial Use – Solvents (for cleaning or degreasing) – Dry Cleaning and Spot Cleaning 4th/5th Gen Only Dry Cleaning

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a solvent (for cleaning or degreasing) – dry cleaning and spot cleaning 4th/5th Gen only dry cleaning:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 5.1 and 0.9 (central tendency and high-end) without PPE. (Table 4-34) (monitoring)
  - Chronic inhalation MOEs 23 and 3.5 (central tendency and high-end) without PPE. (Table 4-35) (monitoring)
  - Acute dermal MOEs 24 and 7.9 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-77)
  - Chronic dermal MOEs 50 and 17 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 1.8E-04 and 1.5E-03 (central tendency and high-end) without PPE. (Table 4-36) (monitoring)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-79)

Risk Considerations: For workers, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory PPE. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency and high-end. EPA based its risk determination on monitoring data. EPA does not assume routine use of respiratory PPE with this exposure scenario. When comparing the model results to the fourth/fifth generation monitoring data results for workers, the model high-end and central tendency are both an order of magnitude greater than the monitoring data. This is expected as the model captures exposures from facilities with third and fourth/fifth generation machines. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	• Dry cleaning solvent • Spot cleaner

5.3.19 Industrial Use – Lubricants and greases – Lubricants and greases (aerosol lubricants)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in lubricants and greases – lubricants and greases (aerosol lubricants):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
  - Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
  - Acute dermal MOEs 24 and 8.0 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-74)
  - Chronic dermal MOEs 51 and 17 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-75)
- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)
  - Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Solvents (for cleaning or degreasing)	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)

5.3.20 Industrial Use – Lubricants and greases – Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in lubricants and greases – lubricants and greases (e.g., penetrating lubricants, cutting tool coolants):

- Does not present an unreasonable risk of injury to health (workers and occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 869 and 239 (central tendency and high-end) without PPE. (Table 4-46)
  - Chronic inhalation MOEs 3,960 and 1,087 (central tendency and high-end) without PPE. (Table 4-47)
  - Acute dermal MOEs 361 and 120 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-80)
  - Chronic dermal MOEs 769 and 256 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-81)
- Cancer (liver tumors):
  - Inhalation: 1.0E-06 and 4.9E-06 (central tendency and high-end) without PPE. (Table 4-48)
  - Dermal: 6.4E-05 and 2.5E-04 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-82)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 869 and 239 (central tendency and high-end). (Table 4-46)
  - Chronic inhalation MOEs 3,960 and 1,087 (central tendency and high-end). (Table 4-47)
- Cancer (liver tumors):
  - Inhalation: 1.0E-06 and 4.9E-06 (central tendency and high-end). (Table 4-48)

Risk Considerations: Risk estimates for workers and ONUs for acute and chronic exposures do not indicate acute or chronic risks from any route of exposure, including cancer risks, in the absence of

respiratory PPE and with assumed dermal protection (PF 10) for workers. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants, aerosol lubricants)

5.3.21 Industrial Use – Adhesives and sealants – Solvent-based adhesives and sealants

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in adhesives and sealants – solvent-based adhesives and sealants:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 96 and 32 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-84)
- Cancer (liver tumors):
  - Dermal: 5.1E-05 and 2.0E-04 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-85)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency or high-end. EPA identified inhalation exposure monitoring data related to the use of PCE-based adhesives, sealants, paints, and coatings. The results in the monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for

this uncertainty when using monitoring data, EPA considered the central tendency estimate when determining ONU risk. Due to the large variety in shop types that may use PCE-based adhesives and coatings, it is unclear how representative these data are of a "typical" site using these products. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Adhesives and sealant chemicals	Solvent-based adhesives and sealants

5.3.22 Industrial Use – Paints and coatings – Solvent-based paints and coatings

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in paints and coatings – solvent-based paints and coatings:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 96 and 32 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-84)
- Cancer (liver tumors):
  - Dermal: 5.1E-05 and 2.0E-04 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-85)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end and central tendency) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA identified inhalation exposure monitoring data related to the use of PCE-based adhesives, sealants, paints, and coatings. The results in the monitoring data only include values for workers as monitoring data for ONUs were not identified. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance but the relative exposure of ONUs to workers in these cases were not quantifiable. To account for this uncertainty when using monitoring data, EPA considered the central tendency estimate when determining ONU risk. Due to the large variety in shop types that may use

PCE-based adhesives and coatings, it is unclear how representative these data are of a "typical" site using these products. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Paints and coatings including paint and coating removers	Solvent-based paints and coatings, including for chemical milling

5.3.23 Industrial Use – Paints and coatings – Maskant for Chemical Milling

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in paints and coatings – maskant for chemical milling:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-72)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (
  -
  - Table **4-73**)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 4.1 (central tendency). (Table 4-40)
  - Chronic inhalation MOEs 19 (central tendency). (Table 4-41)
- Cancer (liver tumors):

Inhalation: 2.2E-04 (central tendency). (

  - Table 4-42)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risks for ONUs for acute, chronic, and cancer inhalation exposures are indicated at the central tendency. EPA identified inhalation exposure monitoring data from a single NIOSH investigation and samples collected by the DoD. EPA did not separately calculate risk estimates for ONUs and workers. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. Due to the variety in industry types and typical per site maskant use rates and the uncertainty of the PCE concentration in the maskant, it is unclear if these data are representative of a "typical" site. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Paints and coatings including paint and coating removers	Solvent-based paints and coatings, including for chemical milling

5.3.24 Industrial Use – Processing aids, not otherwise listed – Pesticide, fertilizer and other agricultural chemical manufacturing

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE in processing aids, not otherwise listed – pesticide, fertilizer and other agricultural chemical manufacturing:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-70)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic cancer and non-cancer risk estimates (high-end and central tendency) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA identified inhalation exposure monitoring data from four studies submitted to EPA. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Processing aids, not otherwise listed	Pesticide, fertilizer, and other agricultural chemical manufacturing

5.3.25 Industrial Use – Processing aids, specific to petroleum production – Catalyst regeneration in petrochemical manufacturing

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE as a processing aids, specific to petroleum production – catalyst regeneration in petrochemical manufacturing processing aid:

- **Presents an unreasonable risk of injury to health (workers).**
- **Presents an unreasonable risk to the environment (aquatic organisms).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers and aquatic organisms:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.
- Algae mortality from exposure.

Driver benchmarks – workers and aquatic organisms:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.
- Mortality: Algae RQ ≥ 1.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-70)

Risk estimate for facilities with exceedances – aquatic organisms: (Table 4-110)

- Algae mortality from exposure: (some facilities had exceedances for multiple scenarios)
  - RQ = 1.9 (algae, 20 days of exceedance, direct release).
  - RQ = 4 (algae, 55 days of exceedance, direct release).
  - RQ = 69 (algae, 20 days of exceedance, direct release).
  - RQ = 4.7 (algae, 20 days of exceedance, direct release).
  - RQ = 4.5 (algae, 92 days of exceedance, indirect release).
  - RQ = 14 (algae, 20 days of exceedance, direct release).
  - RQ = 8.5 (algae, 169 days of exceedance, direct release).
  - RQ = 1.3 (algae, 42 days of exceedance, direct release).

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic cancer and non-cancer risk estimates (high-end and central tendency) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA identified inhalation exposure monitoring data from four studies submitted to EPA. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Environmental releases for this condition of use indicate chronic risk to aquatic organisms and risk to algae. Of the 12 facilities assessed as using PCE as an industrial processing aid, six facilities had releases indicating risk to aquatic organisms (RQs ≥ 1 and 20 days or more of exceedance for algae). RQ values ranged from 1.3 (42 days of exceedance, direct discharge) to 69 (20 days of exceedance, direct discharge). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. Exceedances occurred using direct and indirect release scenarios but were highest for direct release scenarios. All of the facilities assessed as processing PCE as a reactant had NPDES permits. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
Industrial use	Processing aids, specific to petroleum production	Catalyst regeneration in petrochemical manufacturing

5.3.26 Industrial Use – Other uses – Textile processing (spot cleaning)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE for other uses – textile processing (spot cleaning):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute dermal MOEs 24 and 7.9 (central tendency and high-end) with PPE (gloves PF = 10) (Table 4-77)
  - Chronic dermal MOEs 50 and 17 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-78)
- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks, dermal acute non-cancer (high-end), dermal chronic non-cancer (high-end and central tendency), and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). EPA does not assume routine use of respiratory PPE with this exposure scenario. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk. EPA identified inhalation exposure monitoring data from a single NIOSH investigation at a garment manufacturer. Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. ONU exposure data did not distinguish central tendency and high-end. There is some uncertainty in how representative this data are of exposure at other facilities performing carpet cleaning or spot remover tasks. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Other uses	Textile processing

5.3.27 Industrial Use – Other uses – Textile processing (other)

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE for other uses – textile processing (other):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-70)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not identify any inhalation exposure monitoring data for other industrial uses, and therefore assessed inhalation exposures for workers and ONUs using the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model. Due to other potential sources of exposure at industrial facilities, there are some model uncertainties that could result in an underestimate of worker exposure. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Other uses	Textile processing

5.3.28 Industrial Use – Other uses – Wood furniture manufacturing

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE for other uses – wood furniture manufacturing:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-70)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not identify any inhalation exposure monitoring data for other industrial uses, and therefore assessed inhalation exposures for workers and ONUs using the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model. Due to other potential sources of exposure at industrial facilities, there are some model uncertainties that could result in an underestimate of worker exposure. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial use	Other uses	Wood furniture manufacturing

5.3.29 Industrial Use – Other uses – Laboratory chemicals

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE for other uses - laboratory chemical:

- Does not present an unreasonable risk of injury to health (workers and ONUs).
- Does not present unreasonable risk to the environment (aquatic organisms).

Risk Considerations: As discussed in Section 2.4.1.25, EPA does not have data to assess worker exposures to PCE during laboratory use. However, due to the expected safety practices when using chemicals in a laboratory setting, PCE is expected to be applied in small amounts under a fume hood, thus reducing the potential for inhalation exposures. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Other uses	Laboratory chemicals

5.3.30 Industrial Use – Other uses – Foundry applications

Section 6(b)(4)(A) unreasonable risk determination for industrial use of PCE for other uses – foundry applications:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 77 and 26 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 6.4E-04 and 2.5E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-70)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 10). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not identify any inhalation exposure monitoring data for other industrial uses, and therefore assessed inhalation exposures for workers and ONUs using the Tank Truck and Railcar Loading and Unloading Release and Inhalation Exposure Model. Due to other potential sources of exposure at industrial facilities, there are some model uncertainties that could result in an underestimate of worker exposure. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower

than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Other uses	Foundry applications

5.3.31 Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (wipe cleaning)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – cleaners and degreasers (other)(wipe cleaning):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.8E-02 and 2.2E-02 (central tendency and high-end) without PPE. (Table 4-49)
  - Chronic inhalation MOEs 0.2 and 0.1 (central tendency and high-end) without PPE. (Table 4-50)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 2.4E-02 and 5.3E-02 (central tendency and high-end) without PPE. (Table 4-51)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 229 and 0.2 (central tendency and high-end). (Table 4-49)
  - Chronic inhalation MOEs 1043 and 1.0 (central tendency and high-end). (Table 4-50)
- Cancer (liver tumors):
  - Inhalation: 4.0E-06 and 5.4E-03 (central tendency and high-end). (Table 4-51)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory and dermal PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for chronic inhalation at the high-end. EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE for wipe cleaning. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Due to the large variety in shop types that may use PCE as a wipe cleaning solvent, it is unclear how representative these data are of a "typical" shop. EPA does not have a model for estimating exposures from wipe cleaning; therefore, the assessment is based on the identified monitoring data. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Cleaners and degreasers (other) (wipe cleaning)

5.3.32 Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (Other Spot Cleaning/Spot Removers (Including Carpet Cleaning))

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – cleaners and degreasers (other)(other spot cleaning/spot removers (including carpet cleaning)):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:

- Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
- Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)

- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-04 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks, dermal acute non-cancer (high-end), dermal chronic non-cancer (high-end and central tendency), and dermal cancer risk estimates (high-end) indicate risk. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk. EPA identified inhalation exposure monitoring data from a single NIOSH investigation at a garment manufacturer. Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. ONU exposure data did not distinguish central tendency and high-end. There is some uncertainty in how representative this data are of exposure at other facilities performing carpet cleaning or spot remover tasks. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Cleaners and degreasers (other) (other spot cleaning/spot removers (including carpet cleaning))

5.3.33 Commercial Use – Cleaning and furniture care products – Cleaners and degreasers (other) (Mold Release)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – cleaners and degreasers (other) (mold release):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures
- Cancer resulting from chronic dermal exposures

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate – workers:

- Neurotoxicity:
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-04 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks, dermal acute non-cancer (high-end), dermal chronic non-cancer (high-end and central tendency), and dermal cancer risk estimates (high-end) indicate risk. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. Data for this condition of use are area samples, not worker breathing zone samples. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Cleaners and degreasers (other) (mold release)

5.3.34 Commercial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning Post-2006 Dry Cleaning

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – dry cleaning and spot cleaning post-2006 dry cleaning:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 1.4 and 0.3 (central tendency and high-end) without PPE. (Table 4-34) (monitoring)
  - Chronic inhalation MOEs 6.1 and 1.0 (central tendency and high-end) without PPE. (Table 4-35) (monitoring)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 6.8E-04 and 5.4E-03 (central tendency and high-end) without PPE. (Table 4-36) (monitoring)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 30 and 2.1 (central tendency and high-end). (Table 4-34) (modeling)
  - Chronic inhalation MOEs 136 and 9.5 (central tendency and high-end). (Table 4-35) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.9E-05 and 4.3E-04 (central tendency and high-end). (Table 4-36) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory and dermal PPE. While EPA does not assume routine use of respiratory PPE with this exposure scenario, risk was still present to workers with APF 50 for chronic inhalation at the high-end, for monitoring and modeled data. Because the monitoring data only contained one data point representing an ONU for this scenario, EPA made its determination on ONUs using modeled data. Modeled ONU exposures are based on concentrations in the far-field which corresponds to any area outside the near-field zones. Risk estimates for ONUs for acute (high-end), chronic (high-end and central tendency), and cancer (high-end) inhalation exposures indicate risk. EPA separately evaluated risks to consumers from dry cleaned articles as part of the COU, Consumer Use – Cleaning and furniture care products – Dry cleaning solvent, in Section 5.3.52. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Dry cleaning and spot cleaning post-2006 dry cleaning

5.3.35 Commercial Use – Cleaning and furniture care products – Dry Cleaning and Spot Cleaning 4th/5th Gen Only Dry Cleaning

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – dry cleaning and spot cleaning 4th/5th Gen only dry cleaning:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and chronic dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 5.1 and 0.9 (central tendency and high-end) without PPE. (Table 4-34) (monitoring)
  - Chronic inhalation MOEs 23 and 3.5 (central tendency and high-end) without PPE. (Table 4-35) (monitoring)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 1.8E-04 and 1.5E-03 (central tendency and high-end) without PPE. (Table 4-36) (monitoring)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory and dermal PPE. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency and high-end. EPA based its risk determination on monitoring data. When comparing the model results to the fourth/fifth generation monitoring data results for workers, the model high-end and central tendency are both an order of magnitude greater than the monitoring data. This is expected as the model captures exposures from facilities with third and fourth/fifth generation machines. EPA separately evaluated risks to consumers from dry cleaned articles as part of the COU, Consumer Use – Cleaning and furniture care products – Dry cleaning solvent, in Section 5.3.52. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Dry cleaning and spot cleaning 4th/5th Gen only dry cleaning

5.3.36 Commercial Use – Cleaning and furniture care products – Automotive care products (e.g., engine degreaser and brake cleaner)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – automotive care products (e.g., engine degreaser and brake cleaner):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
  - Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE (Table 4-74)
  - Chronic dermal MOEs 5.1 and 1.7 (central tendency and high-end) without PPE. (Table 4-75)
- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-03 (central tendency and high-end) without PPE. (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)

- Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory and dermal PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Automotive care products (e.g. engine degreaser and brake cleaner)

5.3.37 Commercial Use – Cleaning and furniture care products – Aerosol cleaner

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in cleaning and furniture care products – aerosol cleaner:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:

- Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
- Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
- Acute dermal 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-74)
- Chronic dermal MOEs 5.1 and 1.7 (central tendency and high-end) without PPE. (Table 4-75)

- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-04 (central tendency and high-end) without PPE. (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)
  - Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of respiratory and dermal PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Aerosol cleaner

5.3.38 Commercial Use – Cleaning and furniture care products – Non-aerosol cleaner

Section 6(b)(4)(A) unreasonable risk determination of PCE for commercial use – cleaning and furniture care products – non-aerosol cleaner:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.8E-02 and 2.2E-02 (central tendency and high-end) without PPE. (Table 4-49)
  - Chronic inhalation MOEs 0.2 and 0.1 (central tendency and high-end) without PPE. (Table 4-50)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 2.4E-02 and 5.3E-02 (central tendency and high-end) without PPE. (Table 4-51)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 229 and 0.2 (central tendency and high-end). (Table 4-49)
  - Chronic inhalation MOEs 1043 and 1.0 (central tendency and high-end). (Table 4-50)
- Cancer (liver tumors):
  - Inhalation: 4.0E-06 and 5.4E-03 (central tendency and high-end). (Table 4-51)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for chronic inhalation at the high-end. EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE for wipe cleaning. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Due to the large variety in shop types that may use PCE as a wipe cleaning solvent, it is unclear how representative these data are of a "typical" shop. EPA does not have a model for estimating exposures from wipe cleaning; therefore, the assessment is based on the identified monitoring data. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Cleaning and furniture care products	Non-aerosol cleaner

5.3.39 Commercial Use – Lubricants and greases – Lubricants and greases (aerosol lubricants)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in lubricants and greases – lubricants and greases (aerosol lubricants):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
  - Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-74)
  - Chronic dermal MOEs 5.1 and 1.7 (central tendency and high-end) without PPE. (Table 4-75)
- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-03 (central tendency and high-end) without PPE. (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)

- Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Lubricants and greases	Lubricants and greases (aerosol lubricants)

5.3.40 Commercial Use – Lubricants and greases – Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in lubricants and greases – lubricants and greases (e.g., penetrating lubricants, cutting tool coolants):

- Does not present an unreasonable risk of injury to health (workers and occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 869 and 239 (central tendency and high-end) without PPE. (Table 4-46)
  - Chronic inhalation MOEs 3,960 and 1,087 (central tendency and high-end) without PPE. (Table 4-47)
  - Acute dermal MOEs 181 and 60 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-80)

- Chronic dermal MOEs 384 and 128 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-81)
- Cancer (liver tumors):
  - Inhalation: 1.0E-06 and 4.9E-06 (central tendency and high-end) without PPE. (Table 4-48)
  - Dermal: 1.3E-05 and 5.0E-05 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-82)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 869 and 239 (central tendency and high-end). (Table 4-46)
  - Chronic inhalation MOEs 3,960 and 1,087 (central tendency and high-end). (Table 4-47)
- Cancer (liver tumors):
  - Inhalation: 1.0E-06 and 4.9E-06 (central tendency and high-end). (Table 4-48)

Risk Considerations: Risk estimates for workers and ONUs for acute and chronic exposures do not indicate acute or chronic risks from any route of exposure, including cancer risks. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial use	Lubricants and greases	Lubricants and greases (e.g., penetrating lubricants, cutting tool coolants)

5.3.41 Commercial Use – Adhesives and sealant chemicals – Light repair adhesives

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in adhesives and sealant chemicals – light repair adhesives:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.

- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute dermal MOEs 15 and 4.9 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-83)
  - Chronic dermal MOEs 31 and 10 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-84)
- Cancer (liver tumors):
  - Dermal: 1.6E-04 and 6.1E-04 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-85)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 10), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 5). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency or high-end. EPA identified inhalation exposure monitoring data related to the use of PCE-based adhesives, sealants, paints, and coatings. The results in the monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for this uncertainty when using monitoring data, EPA considered the central tendency estimate when determining ONU risk. Due to the large variety in shop types that may use PCE-based adhesives and coatings, it is unclear how representative these data are of a "typical" site using these products. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial use	Adhesives and sealant chemicals	Light repair adhesives

5.3.42 Commercial Use – Paints and coatings – Solvent-based paints and coatings

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in paints and coatings – solvent-based paints and coatings:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from chronic inhalation and acute and chronic dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic inhalation MOEs 976 and 50 (central tendency and high-end) with PPE (respirator APF 10). (Table 4-38)
  - Acute dermal MOEs 15 and 4.9 (central tendency and high-end) with PPE (gloves = 5). (Table 4-83)
  - Chronic dermal MOEs 31 and 10 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-84)
- Cancer (liver tumors):
  - Dermal: 1.6E-04 and 6.1E-04 (central tendency and high-end) with PPE (gloves PF = 5). (Table 4-85)

Risk Considerations: For workers, while acute non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 10), chronic non-cancer (high-end), dermal chronic non-cancer (high-end and central tendency) and dermal cancer risk estimates (high-end and central tendency) indicate risk even with assumed dermal protection (PF 5). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA identified inhalation exposure monitoring data related to the use of PCE-based adhesives, sealants, paints, and coatings. The results in the monitoring data only include values for workers as monitoring data for ONUs were not identified. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance but the relative exposure of ONUs to workers in these cases were not quantifiable. To account for this uncertainty when using monitoring data, EPA considered the central tendency estimate when determining ONU risk. Due to the large variety in shop types that may use PCE-based adhesives and coatings, it is unclear how representative these data are of a "typical" site using these products. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial use	Paints and coatings	Solvent-based paints and coatings

5.3.43 Commercial Use – Other uses – Carpet cleaning

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – carpet cleaning:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks, dermal acute non-cancer, dermal chronic non-cancer, and dermal cancer risk estimates (high-end and central tendency) indicate risk. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk. EPA identified inhalation exposure monitoring data from a single NIOSH investigation at a garment manufacturer. Worker samples were determined to be any sample taken on a person while directly handling PCE. ONUs samples were determined to be any sample taken on a person in the same location as the PCE use but not handling PCE. ONU exposure data did not distinguish central tendency and high-end. There is some uncertainty in how representative this data are of exposure at other facilities performing carpet cleaning or spot remover tasks. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial use	Other uses	Carpet cleaning

5.3.44 Commercial Use – Other uses – Laboratory chemicals

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – laboratory chemicals:

- Does not present an unreasonable risk of injury to health (workers and ONUs).
- Does not present unreasonable risk to the environment (aquatic organisms).

Risk Considerations: As discussed in Section 2.4.1.25, EPA does not have data to assess worker exposures to PCE during laboratory use. However, due to the expected safety practices when using chemicals in a laboratory setting, PCE is expected to be applied in small amounts under a fume hood,

thus reducing the potential for inhalation exposures. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial use	Other uses	Laboratory Chemicals

5.3.45 Commercial Use – Other uses – Metal (e.g., stainless steel) and stone polishes

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – metal (e.g., stainless steel) and stone polishes:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.8E-02 and 2.2E-02 (central tendency and high-end) without PPE. (Table 4-49)
  - Chronic inhalation MOEs 0.2 and 0.1 (central tendency and high-end) without PPE. (Table 4-50)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 2.4E-02 and 5.3E-02 (central tendency and high-end) without PPE. (Table 4-51)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:

- Acute inhalation MOEs 229 and 0.2 (central tendency and high-end). (Table 4-49)
- Chronic inhalation MOEs 1043 and 1.0 (central tendency and high-end). (Table 4-50)

- Cancer (liver tumors):
  - Inhalation: 4.0E-06 and 5.4E-03 (central tendency and high-end). (Table 4-51)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for chronic inhalation at the high-end. EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using PCE for wipe cleaning. EPA separately calculated risk estimates for ONUs and workers based on monitoring data. Due to the large variety in shop types that may use PCE as a wipe cleaning solvent, it is unclear how representative these data are of a "typical" shop. EPA does not have a model for estimating exposures from wipe cleaning; therefore, the assessment is based on the identified monitoring data. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Metal (e.g., stainless steel) and stone polishes

5.3.46 Commercial Use – Other uses – Inks and ink removal products (based on printing)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE in other uses – inks and ink removal products (based on printing):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate – workers:

- Neurotoxicity:
  - Acute inhalation MOEs 2.6 and 0.8 (central tendency and high-end) without PPE. (Table 4-58)

- Chronic inhalation MOEs 12 and 3.8 (central tendency and high-end) without PPE. (Table 4-59)
- Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
- Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)

- Cancer (liver tumors):
  - Inhalation: 3.5E-04 and 1.4E-03 (central tendency and high-end) without PPE. (Table 4-60)
  - Dermal: 9.8E-04 and 3.8E-04 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 2.6 (central tendency). (Table 4-58)
  - Chronic inhalation MOEs 12 (central tendency). (Table 4-59)
- Cancer (liver tumors):
  - Inhalation: 3.5E-04 (central tendency). (Table 4-60)

Risk Considerations: For workers, all pathways of occupational exposure for this condition of use indicate risk (central tendency and high-end) in the absence of respiratory and dermal PPE. Acute, chronic, and cancer inhalation risk estimates for ONUs indicate risk at the central tendency. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Inks and ink removal products (based on printing)

5.3.47 Commercial Use – Other uses – Inks and ink removal products (based on photocopying)

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – inks and ink removal products (based on photocopying):

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate – workers:

- Neurotoxicity:
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks in the absence of respiratory PPE, dermal acute and chronic non-cancer (high-end and central tendency), and dermal cancer (high-end) risk estimates indicate risk in the absence of dermal PPE. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. Risk estimates for ONUs for acute and chronic inhalation do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Inks and ink removal products (based on photocopying)

5.3.48 Commercial Use – Other uses – Welding

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – welding:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation and dermal exposures.

- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Neurotoxicity resulting from acute and chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Neurotoxicity:
  - Acute inhalation MOEs 3.5 and 0.6 (central tendency and high-end) without PPE. (Table 4-31) (monitoring)
  - Chronic inhalation MOEs 16 and 2.9 (central tendency and high-end) without PPE. (Table 4-32) (monitoring)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-74)
  - Chronic dermal MOEs 5.1 and 1.7 (central tendency and high-end) without PPE. (Table 4-75)
- Cancer (liver tumors):
  - Inhalation: 2.6E-04 and 1.8E-03 (central tendency and high-end) without PPE. (Table 4-33) (monitoring)
  - Dermal: 9.6E-04 and 3.7E-03 (central tendency and high-end) without PPE. (Table 4-76)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 50 and 6.8 (central tendency and high-end). (Table 4-31) (modeling)
  - Chronic inhalation MOEs 260 and 31 (central tendency and high-end). (Table 4-32) (modeling)
- Cancer (liver tumors):
  - Inhalation: 2.0E-05 and 1.4E-04 (central tendency and high-end). (Table 4-33) (modeling)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While EPA does not assume routine use of PPE with this exposure scenario, risk was still present to workers with APF 50 for acute and chronic inhalation. The estimates based on monitoring data only include values for workers as monitoring data for ONUs were not identified. To account for lack of monitoring data for ONUs, EPA considered risk estimates from exposure modeling when determining ONU risk. The near-field/far-field exposure modeling incorporates variability in the model input parameters and distinguishes between workers and ONUs. Model results are generally higher than monitoring data; however, the monitoring data includes data from three sources that had concentrations of PCE in the aerosol formulation below the median value predicted by the model. EPA has a high level of confidence in the assessed exposure for this condition of use. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Welding

5.3.49 Commercial Use – Other uses – Photographic film

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – photographic film:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic inhalation, and acute and chronic dermal exposures.
- Cancer resulting from chronic inhalation and chronic dermal exposures.

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate – workers:

- Neurotoxicity:
  - Acute inhalation MOEs 0.8 and 8.9E-02 (central tendency and high-end) without PPE. (Table 4-58)
  - Chronic inhalation MOEs 3.6 and 0.4 (central tendency and high-end) without PPE. (Table 4-59)
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Inhalation: 1.1E-03 and 1.3E-02 (central tendency and high-end) without PPE. (Table 4-60)
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk estimate – ONUs:

- Neurotoxicity:
  - Acute inhalation MOEs 0.8 (central tendency). (Table 4-58)
  - Chronic inhalation MOEs 3.6 (central tendency). (Table 4-59)
- Cancer (liver tumors):
  - Inhalation: 1.1E-03 (central tendency). (Table 4-60)

Risk Considerations: For workers, all pathways of occupational exposure for this condition of use indicate risk (central tendency and high-end) in the absence of respiratory and dermal PPE. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. Risk estimates for ONUs for acute and chronic non-cancer and cancer inhalation exposures indicate risk at the central tendency EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Photographic Film

5.3.50 Commercial Use – Other uses – Mold cleaning, release and protectant products

Section 6(b)(4)(A) unreasonable risk determination for commercial use of PCE for other uses – mold cleaning, release and protectant products:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).
- Does not present unreasonable risk to the environment (aquatic organisms).

Unreasonable risk driver – workers:

- Neurotoxicity resulting from acute and chronic dermal exposures
- Cancer resulting from chronic dermal exposures

Driver benchmarks – workers:

- Neurotoxicity: Acute non-cancer benchmark MOE = 10.
- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.

Risk estimate – workers:

- Neurotoxicity:
  - Acute dermal MOEs 2.4 and 0.8 (central tendency and high-end) without PPE. (Table 4-77)
  - Chronic dermal MOEs 5.0 and 1.7 (central tendency and high-end) without PPE. (Table 4-78)
- Cancer (liver tumors):
  - Dermal: 9.8E-04 and 3.8E-03 (central tendency and high-end) without PPE. (Table 4-79)

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks, dermal acute non-cancer (high-end), dermal chronic non-cancer (high-end and central tendency), and dermal cancer risk estimates (high-end) indicate risk. EPA does not assume routine use of respiratory or dermal PPE with this exposure scenario. Risk estimates for ONUs for acute

and chronic inhalation exposures do not indicate risk at the central tendency. Data for this condition of use are area samples, not worker breathing zone samples. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk. No environmental risks were identified for this COU.

Life Cycle Stage	Category	Subcategory
Commercial Use	Other uses	Mold cleaning, release and protectant products

5.3.51 Consumer Use – Cleaning and furniture care products – Cleaners and degreasers (other)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – cleaners and degreasers (other):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 0.2 (moderate intensity user). (Table 4-86)
  - Acute dermal MOE 0.6 (moderate intensity user). (Table 4-87)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 0.8 (moderate intensity user). (Table 4-86)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Cleaners and degreasers (other)

5.3.52 Consumer Use – Cleaning and furniture care products – Dry cleaning solvent

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – dry cleaning solvent:

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute dermal exposures.

Driver benchmarks – consumers:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Half-body acute dermal MOE 8.6 (Table 4-109, 1 day after dry cleaning, 2nd and 3rd generation).
- Full-body acute dermal MOE 2.9, 3.7, and 4.9 (Table 4-109, 1, 2, and 3 days after dry cleaning, 2nd and 3rd generation).

Risk Considerations: Consumer exposure to perchloroethylene due to off-gassing from recently dry cleaned articles was evaluated for two scenarios, direct dermal contact with clothing to consumers and inhalation exposure to bystanders from article storage in a home closet. Modeling was used to estimate dermal and inhalation exposures. Measurements of PCE concentrations in indoor air from storage of recently dry cleaned articles are in good agreement with modeling results. No direct measurements were found for consumer dermal exposure to PCE from dry cleaned fabrics. Dermal exposure due to direct skin contact with recently dry cleaned fabrics during article wear was assessed for consumer users, for older and more modern dry cleaning technologies (2nd-5th generation). Risk estimates for consumer users from articles dry cleaned with 2nd and 3rd generation machines indicate risk for half-body dermal exposure to dry cleaned clothing (1 day after dry cleaning) and for full-body dermal exposure (1, 2, and 3 days after dry cleaning). EPA did not find risk to bystanders.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Dry cleaning solvent

5.3.53 Consumer Use – Cleaning and furniture care products – Automotive care products (Brake cleaner)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – automotive care products (brake cleaner):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 0.2 (moderate intensity user). (Table 4-88)
  - Acute dermal MOE 0.6 (moderate intensity user). (Table 4-89)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 0.8 (moderate intensity user). (Table 4-88)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Automotive care products (Brake cleaner)

5.3.54 Consumer Use – Cleaning and furniture care products – Automotive care products (Parts cleaner)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – automotive care products (parts cleaner):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 0.6 (moderate intensity user). (Table 4-90)
  - Acute dermal MOE 1.3E-02 (moderate intensity user). (Table 4-91)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 3.3 (moderate intensity user). (Table 4-90)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Automotive care products (Parts cleaner)

5.3.55 Consumer Use – Cleaning and furniture care products – Aerosol cleaner (Vandalism Mark & Stain Remover, Mold Cleaner, Weld Splatter Protectant)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – aerosol cleaner (vandalism mark & stain remover, mold cleaner, weld splatter protectant):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 0.3 (moderate intensity user). (Table 4-92)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 1.6 (moderate intensity user). (Table 4-92)

Risk Considerations: Risk estimates for consumer users and bystanders indicate risk from acute inhalation exposures. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Aerosol cleaner (Vandalism Mark & Stain Remover, Mold Cleaner, Weld Splatter Protectant)

5.3.56 Consumer Use – Cleaning and furniture care products – Non-aerosol cleaner (e.g., marble and stone polish)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in cleaning and furniture care products – non-aerosol cleaner (e.g., marble and stone polish):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 6.8E-02 (moderate intensity user). (Table 4-93)
  - Acute dermal MOE 5.4E-02 (moderate intensity user). (Table 4-94)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 0.4 (moderate intensity user). (Table 4-93)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Non-aerosol cleaner (e.g., marble and stone polish)

5.3.57 Consumer Use – Lubricants and greases – Lubricants and greases (cutting fluid)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in lubricants and greases – lubricants and greases (cutting fluid):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 1.3 (moderate intensity user). (Table 4-95)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 6.7 (moderate intensity user). (Table 4-95)

Risk Considerations: Risk estimates for consumer users and bystanders at the medium intensity use scenarios of acute inhalation exposures indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to

users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Lubricants and greases (cutting fluid)

5.3.58 Consumer Use – Lubricants and greases – Lubricants and greases (Lubricants and Penetrating Oils)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in lubricants and greases – lubricants and greases (lubricants and penetrating oils):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 1.4 (moderate intensity user). (Table 4-96)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 7.3 (moderate intensity user). (Table 4-96)

Risk Considerations: Risk estimates for consumer users and bystanders at the medium intensity use scenarios of acute inhalation exposures indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Cleaning and furniture care products	Lubricants and greases (lubricants and penetrating oils)

5.3.59 Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in adhesives and sealant chemicals – adhesives for arts and crafts (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant):

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 2.3 (moderate intensity user). (Table 4-97)

Risk Considerations: Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation exposures indicate risk. EPA did not find risk to bystanders. Consumer risk determinations reflect the effects associated with acute exposures. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Adhesive and sealant chemicals	Adhesives for arts and crafts (includes industrial adhesive, arts and crafts adhesive, gun ammunition sealant)

5.3.60 Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (Livestock Grooming Adhesive)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in adhesives and sealant chemicals – adhesives for arts and crafts (livestock grooming adhesive):

- Does not present an unreasonable risk of injury to health (consumers and bystanders).

Benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 12 (moderate intensity user). (Table 4-98)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 64(moderate intensity user). (Table 4-98)

Risk Considerations: Risk estimates for consumer users and bystanders at the medium intensity use scenarios of acute inhalation exposures do not indicate risk. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Adhesive and sealant chemicals	Adhesives for arts and crafts (Livestock grooming adhesive)

5.3.61 Consumer Use – Adhesives and sealant chemicals – Adhesives for arts and crafts (Column Adhesive, Caulk and Sealant)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in adhesives and sealant chemicals – adhesives for arts and crafts (column adhesive, caulk and sealant):

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders)**.**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 2.3 (moderate intensity user). (Table 4-99)

Risk Considerations: Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation exposures indicate risk. Consumer risk determinations reflect the effects associated with acute exposures. Acute inhalation exposure for bystanders was not evaluated, as the consumer area of use was assumed to be similar conditions as outside the home. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE.

Life Cycle Stage	Category	Subcategory
Consumer use	Adhesive and sealant chemicals	Light Repair Adhesives - Adhesives for arts and crafts (Column Adhesive, Caulk and Sealant)

5.3.62 Consumer Use – Paints and coatings – Solvent-based paints and coatings (Outdoor water shield (liquid))

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in paints and coatings – solvent-based paints and coatings (outdoor water shield (liquid)):

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 1.1 (moderate intensity user). (Table 4-100)
  - Acute dermal MOE 2.5E-02 (moderate intensity user) (Table 4-101)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 3.3 (moderate intensity user). (Table 4-100)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Paints and coatings	Solvent-based paints and coatings (Outdoor water shield (liquid))

5.3.63 Consumer Use – Paints and coatings – Solvent-based paints and coatings (Coatings and primers (aerosol))

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in paints and coatings – solvent-based paints and coatings (coatings and primers (aerosol)):

- Does not present an unreasonable risk of injury to health (consumers and bystanders).

Unreasonable risk driver – consumers and bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 62 (moderate intensity user). (Table 4-102)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 2143 (moderate intensity user). (Table 4-102)

Risk Considerations: Risk estimates for consumer users and bystanders at the medium intensity use scenarios of acute inhalation exposures do not indicate risk. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Paints and coatings	Solvent-based paints and coatings (Coatings and primers (aerosol))

5.3.64 Consumer Use – Paints and coatings – Solvent-based paints and coatings (Rust Primer and Sealant (liquid))

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in paints and coatings – solvent-based paints and coatings (rust primer and sealant (liquid)):

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from dermal exposures.

Driver benchmarks – consumers:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Acute dermal MOE 1.8E-02 (moderate intensity user) (Table 4-104)

Risk Considerations: Risk estimates for consumer users at the medium intensity use scenarios of dermal exposures indicate risk. EPA did not find risk to bystanders. Consumer risk determinations reflect the effects associated with dermal exposures. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Paints and coatings	Solvent-based paints and coatings (Rust Primer and Sealant (liquid))

5.3.65 Consumer Use – Paints and coatings – Solvent-based paints and coatings (Metallic Overglaze)

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in paints and coatings – solvent-based paints and coatings (metallic overglaze):

- Does not present an unreasonable risk of injury to health (consumers and bystanders).

Benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 337 (moderate intensity user). (Table 4-105)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 1674 (moderate intensity user). (Table 4-105)

Risk Considerations: Risk estimates for consumer users and bystanders at the medium intensity use scenarios of acute inhalation exposures do not indicate risk. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Paints and coatings	Solvent-based paints and coatings (Metallic Overglaze)

5.3.66 Consumer Use – Other Uses – Metal (e.g., stainless steel) and stone polishes

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in other uses – metal (e.g., stainless steel) and stone polishes:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity:
  - Acute inhalation MOE 0.2 (moderate intensity user). (Table 4-106)
  - Acute dermal MOE 0.1 (moderate intensity user) (Table 4-107)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 0.8 (moderate intensity user). (Table 4-106)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation and dermal exposures indicate risk. For bystanders, the risk estimates for the medium intensity use scenario of acute inhalation indicate risk. Because bystanders are not expected to be dermally exposed to PCE, dermal risks to bystanders were not evaluated. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Other Uses	Metal (e.g., stainless steel) and stone polishes

5.3.67 Consumer Use – Other Uses – Inks and ink removal products; welding; mold cleaning, release and protectant products

Section 6(b)(4)(A) unreasonable risk determination for consumer use of PCE in other uses – inks and ink removal products; welding; mold cleaning, release and protectant products:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Neurotoxicity resulting from acute inhalation.

Unreasonable risk driver – bystanders:

- Neurotoxicity resulting from acute inhalation.

Driver benchmarks – consumers and bystanders:

- Neurotoxicity: Benchmark MOE = 10.

Risk estimate – consumers:

- Neurotoxicity: Acute inhalation MOE 0.3 (moderate intensity user). (Table 4-92)

Risk estimate – bystanders:

- Neurotoxicity: Acute inhalation MOE 1.6 (moderate intensity user). (Table 4-92)

Risk Considerations: Risk estimates for consumer users and bystanders indicate risk from acute inhalation exposures. Consumer and bystander risk determinations reflect the effects associated with acute exposures. Dermal exposures were not quantified for this scenario, as consumer dermal exposure with impeded evaporation is not expected, and bystanders are not expected to be dermally exposed to PCE. For the consumer exposure scenario for bystanders, inhalation exposures were estimated using the same model (CEM 2.1) used to estimate exposure to users. CEM 2.1 is a two-zone model that allows for the estimation of air concentrations a user and bystander(s) would be exposed to following an exposure event.

Life Cycle Stage	Category	Subcategory
Consumer use	Other Uses	• Inks and ink removal products • Welding • Mold cleaning, release and protectant products

5.3.68 Disposal

Section 6(b)(4)(A) unreasonable risk determination for the disposal of PCE:

- **Presents an unreasonable risk of injury to health (workers).**
- **Presents an unreasonable risk to the environment (aquatic organisms).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers and aquatic organisms:

- Neurotoxicity resulting from chronic dermal exposures.
- Cancer resulting from chronic dermal exposures.
- Growth effects to aquatic invertebrates from chronic exposure.
- Algae mortality from exposure.

Driver benchmarks – workers and aquatic organisms:

- Neurotoxicity: Chronic non-cancer benchmark MOE = 100.
- Cancer (liver tumors): Benchmark = $1x10^{-4}$.
- Mortality: Algae RQ ≥ 1.

Risk estimate - workers:

- Neurotoxicity:
  - Chronic dermal MOEs 154 and 51 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-69)
- Cancer (liver tumors):
  - Dermal: 3.2E-05 and 1.2E-04 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-70)

Risk estimate for facilities with exceedances – aquatic organisms: (Table 4-110)

- Algae mortality from exposure: (some facilities had exceedances for multiple scenarios)
  - RQ = 6.4 (algae, 172 days of exceedance, indirect release).
  - RQ = 80 (algae, 20 days of exceedance, indirect release).
  - RQ = 25 (algae, 235 days of exceedance, indirect release).
  - RQ = 311 (algae, 20 days of exceedance, indirect release).
  - RQ = 2.2 (algae, 90 days of exceedance, indirect release).

Risk Considerations: For workers, while non-cancer and cancer risk estimates for inhalation exposures do not indicate risks with assumed respiratory protection (APF 25), dermal chronic non-cancer and dermal cancer risk estimates (high-end) indicate risk even with assumed dermal protection (PF 20). Risk estimates for ONUs for acute and chronic inhalation exposures do not indicate risk at the central tendency. EPA did not separately calculate risk estimates for ONUs and workers. There is uncertainty in the ONU risk estimate since the data did not distinguish between worker and ONU inhalation exposure estimates. ONU inhalation exposures are expected to be lower than inhalation exposures for workers directly handling the chemical substance; however, the relative exposure of ONUs to workers in these cases cannot be quantified. To account for this uncertainty, EPA considered the central tendency estimate when determining ONU risk.

Environmental releases for this condition of use indicate chronic risk to aquatic organisms and risk to algae. Of the 13 facilities assessed for the waste handling, disposal, treatment, and recycling of PCE, three facilities had releases indicating risk to aquatic organisms (RQs ≥ 1 and 20 days of exceedance for algae). RQ values ranged from 2.2 (90 days of exceedance, indirect discharge) to 311 (20 days of exceedance, indirect discharge). Industrial wastewater or liquid wastes may be treated on-site and then released to surface water (direct discharge) or pre-treated and released to POTW (indirect discharge). EPA estimated 80% removal of PCE from indirect discharging facilities and 0% removal for direct releases to surface water. Exceedances occurred using indirect release scenarios. An exceedance from indirect release indicates that risk can exist even with waste water treatment if the rate of PCE release to

surface water is high. Four of the 13 facilities assessed as for the waste handling, disposal, treatment, and recycling of PCE did not have NPDES permits. EPA identified risk to algae from indirect release of PCE to surface water from one of the facilities without a NPDES permit. Lack of a NPDES permit increases the uncertainty in the surface water release estimate for a facility. Based on the surface water PCE concentration and COC confidence levels, the overall confidence in the risk estimate to aquatic organisms from exposure to PCE is medium.

Life Cycle Stage	Category	Subcategory
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

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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		
Toxics Substances Control Act (TSCA) – Section 6(b)	EPA is directed to identify and begin risk evaluations on 10 chemical substances drawn from the 2014 update of the TSCA Work Plan for Chemical Assessments.	PCE is on the initial list of chemicals to be evaluated for unreasonable risk under TSCA (81 FR 91927, December 19, 2016).
Toxics Substances Control Act (TSCA) – Section 8(a)	The TSCA Section 8(a) Chemical Data Reporting (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 United States.	PCE manufacturing (including importing), processing, and use information is reported under the Chemical Data Reporting (CDR) rule (40 CFR 711).
Toxics Substances Control Act (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 United States.	PCE was on the initial TSCA Inventory and therefore was not subject to EPA's new chemicals review process (60 FR 16309, March 29, 1995).
Toxics Substances Control Act (TSCA) – Section 8(e)	Manufacturers (including imports), 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.	Eleven risk reports received for PCE (1978-2010) (US EPA, ChemView. Accessed April 13, 2017).
Toxics Substances Control Act (TSCA) – Section 4	Provides EPA with authority to issue rules and orders requiring manufacturers (including importers) and processors to test chemical substances and mixtures.	Nine chemical data submissions from test rules received for PCE (1978-1980) (US EPA, ChemView. Accessed April 13, 2017).
Emergency Planning and Community Right-to-Know Act	Requires annual reporting from facilities in specific industry sectors that employ 10 or more full time	PCE is a listed substance subject to reporting requirements under 40 CFR 372.65 effective as of January 1, 1987.

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
(EPCRA) – Section 313	equivalent employees and that manufacture, process or otherwise use a TRI-listed chemical in quantities above threshold levels.	
Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) - Sections 3 and 6	FIFRA governs the sale, distribution and use of pesticides. Section 3 of FIFRA generally requires that pesticide products be registered by EPA prior to distribution or sale. Pesticides may only be registered if, among other things, they do not cause "unreasonable adverse effects on the environment." Section 6 of FIFRA provides EPA with the authority to cancel pesticide registrations if either (1) the pesticide, labeling or other material does not comply with FIFRA; or (2) when used in accordance with widespread and commonly recognized practice, the pesticide generally causes unreasonable adverse effects on the environment.	EPA removed PCE and other chemical substances from its list of pesticide product inert ingredients used in pesticide products (63 FR 34384, June 24, 1998).
Clean Air Act (CAA) – Section 112(b)	Defines the original list of 189 hazardous air pollutants (HAP). 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 deleting a substance. Since 1990 EPA has removed two pollutants from the original list leaving 187 at present.	Lists PCE as a Hazardous Air Pollutant (42 U.S. Code § 7412), and is considered an "urban air toxic" (CAA Section 112(k)).
Clean Air Act (CAA) – Section 112(d)	Section 112(d) states that the EPA must establish national emission standards for HAP (NESHAP) for each category or subcategory of 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 to be achievable by each	There are a number of source-specific CAA, Section 112, NESHAPs for PCE, including: Dry cleaners (73 FR 39871, July 11, 2008) Organic liquids distribution (non-gasoline) (69 FR 5038, February 3, 2004)

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	particular source category. Different criteria for maximum achievable control technology (MACT) apply for new and existing sources. Less stringent standards, known as generally available control technology (GACT) standards, are allowed at the Administrator's discretion for area sources.	Off-site waste and recovery operations (64 FR 38950, July 20, 1999) Rubber Tire Manufacturing (67 FR 45588, July 9, 2002) Wood furniture manufacturing (60 FR 62930, December 7, 1995) Synthetic organic chemical manufacturing (59 FR 19402, April 22,1994) Chemical Manufacturing Area Source Categories (74 FR 56008, October 29, 2009) Publicly Owned Treatment Works (64 FR 57572, October 26, 1999) Site Remediation includes PCE (68 FR 58172, October 8, 2003)
Clean Air Act (CAA) – Section 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, as necessary, taking into account developments in practices, processes and control technologies."	EPA has promulgated a number of RTR NESHAP (e.g., the RTR NESHAP for PCE Dry Cleaning (71 FR 42724; July 27, 2006) and 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
Clean Air Act (CAA) – Section 183(e)	Section 183(e) requires EPA to list the categories of consumer and commercial products that account for at least 80 percent of all VOC emissions in areas that violate the National Ambient Air Quality Standards (NAAQS) for ozone and to issue standards for these categories that require "best available controls." In lieu of regulations, EPA may issue control techniques guidelines if the guidelines are determined to be substantially as effective as regulations.	PCE is listed under the National Volatile Organic Compound Emission Standards for Aerosol Coatings (40 CFR part 59, subpart E). PCE has a reactivity factor of 0.04g O3/g VOC.
Clean Air Act (CAA) – Section 612	Under Section 612 of the Clean Air Act (CAA), EPA's Significant New Alternatives Policy (SNAP) program	Under the SNAP program, EPA listed PCE as an acceptable substitute in cleaning solvent for metal cleaning,

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	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.	electronics cleaning and precision cleaning (59 FR 13044, March 18, 1994). PCE is cited as an alternative to methyl chloroform and CFC-113 for metals, electronics and precision cleaning. PCE was also noted to have no ozone depletion potential and cited as a VOC-exempt solvent and acceptable ozone-depleting substance substitute (72 FR 30142, May 30, 2007).
Clean Water Act (CWA) – Section 301(b), 304(b), 306, and 307(b)	Requires establishment of Effluent Limitations Guidelines and Standards for conventional, toxic, and non-conventional 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.	PCE is designated as a toxic pollutant under section 307(a)(1) of CWA and as such is subject to effluent limitations. Also under section 304, PCE is included in the list of total toxic organics (TTO) (40 CFR 413.02(i)).
Clean Water Act (CWA) 304(a)	Section 304(a)(1) of the Clean Water Act (CWA) requires EPA to develop and publish, and from time to time revise, recommended criteria for the protection of water quality that accurately reflect the latest scientific knowledge. Water quality criteria developed under section 304(a) are based solely on data and scientific judgments on the relationship between pollutant concentrations and environmental and human health effects.	
Clean Water Act (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 Code of Federal Regulations at 40 CFR 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	

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	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 (Section 402(a)(1)(B)).	
Safe Drinking Water Act (SDWA) – Section 1412	Requires EPA to publish a 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 judgment 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 contaminant level (MCL) or a required treatment technique. Public water systems are required to comply with NPDWRs	PCE is subject to National Primary Drinking Water Regulations (NPDWR) under SDWA with a MCLG of zero and an enforceable maximum contaminant level (MCL) of 0.005 mg/L (40 CFR 141.61). On January 11, 2017, EPA announced a review of the eight existing NPDWRs (82 FR 3518). PCE is one of the eight NPDWRs. EPA requested comment on the eight NPDWRs identified as candidates for revision.
Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) – Section 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	PCE is a hazardous substance under CERCLA. Releases of PCE in excess of 100 pounds must be reported (40 CFR 302.4).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	knowledge of a release of a hazardous substance above the reportable quantity threshold.	
Resource Conservation and Recovery Act (RCRA) – Section 3001	Directs EPA to develop and promulgate criteria for governing hazardous waste identification, classification, generation, management and disposal.	RCRA Subtitle C, Section 3001 identifies PCE as a characteristic and listed hazardous waste. RCRA Hazardous Waste Code: D039 (Toxicity); F001, F002; U210. 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 (78 FR 46447, July 31, 2013).
Superfund Amendments and Reauthorization Act (SARA) –	Requires the Agency to revise the hazardous ranking system and update the National Priorities List of hazardous waste sites, increases state and citizen involvement in the superfund program and provides new enforcement authorities and settlement tools.	PCE is listed on SARA, an amendment to CERCLA and the CERCLA Priority List of Hazardous Substances. This list includes substances most commonly found at facilities on the CERCLA National Priorities List (NPL) that have been deemed to pose the greatest threat to public health.
Other Federal Regulations		
Federal Hazardous Substance Act (FHSA)	Allows the Consumer Product Safety Commission (CPSC) to (1) require precautionary labeling on the immediate container of hazardous household products or (2) to ban certain products that are so dangerous or the nature of the hazard is such that required labeling is not adequate to protect consumers.	Under the Federal Hazardous Substance Act, section 1500.83(a)(31), visual novelty devices containing PCE are regulated by CPSC.
Federal Food, Drug, and Cosmetic Act (FFDCA)	Provides the U.S. FDA (Food and Drug Administration) with authority to oversee the safety of food, drugs and cosmetics.	The FDA regulates PCE in bottled water. The maximum permissible level of PCE in bottled water is 0.005 mg/L (21 CFR 165.110).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
Occupational Safety and Health Act (OSH 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. Under the Act, the Occupational Safety and Health Administration can issue occupational safety and health standards including such provisions as Permissible Exposure Limits (PELs), exposure monitoring, engineering and administrative control measures and respiratory protection.	In 1970, OSHA issued occupational safety and health standards for PCE that included a Permissible Exposure Limit (PEL) of 100 ppm 8 hr. TWA, with a ceiling level of 200 ppm for 5 minutes in any 3 hr. period with a maximum peak of 300 ppm (29 CFR 1910.1000).
Atomic Energy Act Department of Energy (DOE)	The Atomic Energy Act authorizes DOE to regulate the health and safety of its contractor employees	10 CFR 851.23, Worker Safety and Health Program, requires the use of the 2005 ACGIH® TLV®s if they are more protective than the OSHA PEL. The 2005 TLV® for PCE is 25 ppm (8hr Time Weighted Average) and 100 ppm Short Term Exposure Limit(STEL).

A.2 State Laws and Regulations

Table_Apx A-2. State Laws and Regulations

State Actions	Description of Action
State actions	
State Permissible Exposure Limits	California has a workplace PEL of 25 ppm (California, OEHHA, 1988)
State Right-to-Know Acts	Massachusetts (454 CMR 21.00), New Jersey (42 N.J.R 1709(a)), Pennsylvania (Chapter 323, Hazardous Substance List), Rhode Island (RI Gen. Laws Sec. 28-21-1et seq).
Volatile Organic Compound (VOC) Regulations for Consumer Products	Many states regulate PCE 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),

State Actions	Description of Action
	Delaware (Adm. Code Title 7, 1141), District of Columbia (Rules 20-720, 20-721, 20-735, 20-736, 20737), 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.
Other	There are several state level NESHAPs for dry cleaning and restrictions or phase outs of PCE (e.g. California, Maine, Massachusetts). Numerous states list PCE on a list of chemical substances of high concern to children (e.g. Oregon, Vermont, Washington). Under the California Proposition 65 list (California OEHHA), PCE is known to the state of California to cause cancer.

A.3 International Laws and Regulations

Table_Apx A-3. Regulatory Actions by Other Governments and Tribes

Country/Organization	Requirements and Restrictions
Canada	PCE is on the Canadian List of Toxic Substances (CEPA 1999 Schedule 1). The use and sale of PCE in the dry cleaning industry is regulated under *Use in Dry Cleaning and Reporting Requirements Regulations (Canada Gazette*, Part II on March 12, 2003. PCE is also regulated for use and sale for solvent degreasing under Solvent Degreasing Regulations (SOR/2003-283) (Canada Gazette, Part II on August 13, 2003). The purpose of the regulation is to reduce releases of PCE into the environment from solvent degreasing facilities using more than 1,000 kilograms of PCE per year. The regulation includes a market intervention by establishing tradable allowances for the use of PCE in solvent degreasing operations that exceed the 1,000 kilograms threshold per year.
European Union	PCE was evaluated under the 2013 Community Rolling Action Plan (CoRAP). The conclusion was no additional regulatory action was required (European Chemicals Agency (ECHA) database. Accessed April, 18 2017).
Australia	In 2011, a preliminary assessment of PCE was conducted (National Industrial Chemicals Notification and Assessment Scheme, NICNAS, 2016, Tetrachloroethylene. Accessed April, 18 2017).
Japan	PCE 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) • Act on Confirmation, etc. of Release Amounts of Specific Chemical Substances in the Environment and Promotion of Improvements to the Management Thereof

Country/Organization	Requirements and Restrictions
	• Industrial Safety and Health Act (ISHA) • Air Pollution Control Law • Water Pollution Control Law • Soil Contamination Countermeasures Act • Law for the Control of Household Products Containing Harmful Substances (National Institute of Technology and Evaluation (NITE) Chemical Risk Information Platform (CHIRP). Accessed April 18, 2017)
Australia, Austria, Belgium, Canada, Denmark, European Union, Finland, France, Germany, Hungary, Ireland, Israel, Japan, Latvia, New Zealand, People's Republic of China, Poland, Singapore, South Korea, Spain, Sweden, Switzerland, United Kingdom	Occupational exposure limits for PCE (GESTIS International limit values for chemical agents (Occupational exposure limits, OELs) database. Accessed April 18, 2017).
Basel Convention	Halogenated organic solvents (Y41) are listed as a category of waste under the Basel Convention – Annex I. Although the United States 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.

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Appendix B LIST OF SUPPLEMENTAL DOCUMENTS

1. Draft Risk Evaluation for Perchloroethylene (U.S. EPA 2020a)

2. Draft Charge to the Panel for Perchloroethylene

3. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Extraction Tables for Environmental Fate and Transport Studies

4. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Fate and Transport Studies

5. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Physical Chemical Properties

6. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data Common Sources

7. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure

8. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation for Consumer and Environmental Exposure

9. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Extraction for Consumer and Environmental Exposure

10. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Ecological Hazard Studies

11. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Extraction Tables for Environmental Hazard Studies

12. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies

13. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Epidemiological Studies

14. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Extraction for Human Health Hazard Studies

15. Draft Risk Evaluation for Perchloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Animal Studies

16. Draft Risk Evaluation for Perchloroethylene Assessment of Occupational Exposure and Environmental Releases for Perchloroethylene

17. Draft Risk Evaluation for Perchloroethylene Occupational Risk Calculations

18. Draft Risk Evaluation for Perchloroethylene Consumer Inhalation Risk Calculations

19. Draft Risk Evaluation for Perchloroethylene Consumer Dermal Risk Calculations

20. Draft Risk Evaluation for Perchloroethylene Supplemental Information on Consumer Exposure

21. Draft Risk Evaluation for Perchloroethylene Supplemental Information on E-Fast Surface Water Modeling Outputs

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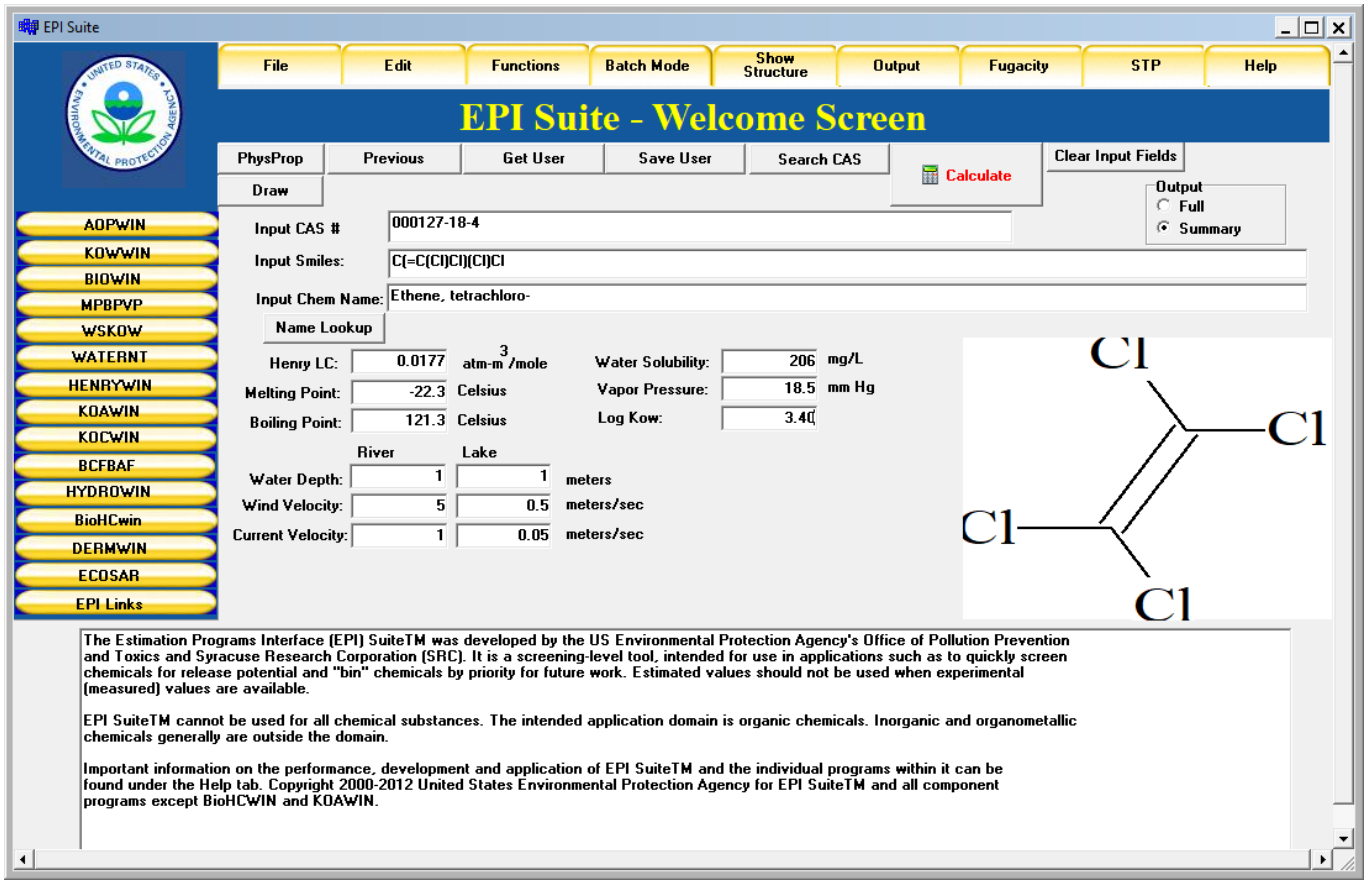
Appendix C FATE AND TRANSPORT

EPI Suite™ Model Inputs

To set up EPI Suite™ for estimating fate properties of PCE, PCE 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).



Figure_Apx C-1. Screen capture of EPISuite™ parameters used to calculate fate and physical chemical properties for PCE.

Appendix D ENVIRONMENTAL EXPOSURES

EPA presents the industrial sectors for each condition of use category below. In cases where the NPDES 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 (U.S. EPA 2014b) to estimate potential occurrence of PCE shown in Table_Apx D-1.

EPA also conducted a geospatial analysis at the watershed level (HUC-8 and HUC-12) to compare the measured and predicted surface water concentrations and investigate if the facility releases may be associated with the observed concentrations in surface water. Below in Table_Apx D-2, Table_Apx D-3 and Table_Apx D-4 EPA has broken out the occurrence of PCE by facility, monitoring sites and location by State.

Table_Apx D-1 provides the industrial sectors for each condition of use.

Table_Apx D-1. Industry Sector Modeled for Facilities without Site-Specific Flow Data in E-FAST 2014

Condition of Use	Industry Sector (SIC Code Option)
OES: Manufacturing	Organic Chemicals Manufacture
OES: Import/Repackaging	POTW (Industrial)
OES: Processing as a Reactant	Organic Chemicals Manufacture
OES: Incorporation into Formulation	n/a
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, Cold Cleaning, and Metalworking Fluids)	Primary Metal Forming Manufacture
OES: Aerosol Degreasing/Lubricants	n/a
OES: Dry Cleaning (commercial only)	n/a
OES: Dry Cleaning (industrial only)	n/a
OES: Adhesives, Paints, and Coatings	n/a
OES: Chemical Maskant	Metal Finishing
OES: Industrial Processing Aid	POTW (Industrial)
OES: Wipe Cleaning and Metal/Stone Polishes	n/a
OES: Other Spot Cleaning/Spot Removers (Including Carpet Cleaning)	n/a
OES: Other Industrial Uses	POTW (Industrial)
OES: Other Commercial Uses	POTW (Industrial)
OES: Waste Handling, Disposal, Treatment, and Recycling	POTW (Industrial)

n/a = Not applicable because a NPDES or surrogate NPDES was available in E-FAST 2014 (U.S. EPA 2014b) to obtain a site-specific stream flow for all facilities within the OES.

Table_Apx D-2 and Table_Apx D-3 show the occurrence of PCE release via facilities and monitoring sites for HUC 8 and HUC 12 respectively.

Table_Apx D-2. Occurrence of PCE Releases (Facilities) and Monitoring Sites By HUC-8.

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
Co-located PCE Releases (Facilities) and Monitoring Sites (n = 4 HUCs)							
04040001	Little Calumet-Galien	440799.0	1783.8	IL,IN,MI	1	2	5
04050006	Lower Grand	1293837.6	5236.0	MI	1	1	4
07040001	Rush-Vermillion	711813.5	2880.6	MN,WI	1	1	1
11030012	Little Arkansas	910452.3	3684.5	KS	1	5	14
PCE Releases (Facilities) Only (n = 66 HUCs)							
10190003	Middle South Platte-Cherry Creek	1838438.0	7439.9	CO	5	0	0
02030105	Raritan	707463.2	2863.0	NJ	4	0	0
08080206	Lower Calcasieu	812177.5	3286.8	LA	4	0	0
12040104	Buffalo-San Jacinto	756769.3	3062.5	TX	4	0	0
02060003	Gunpowder-Patapsco	907202.4	3671.3	MD,PA	3	0	0
07120004	Des Plaines	931517.4	3769.7	IL,WI	3	0	0
08070204	Lake Maurepas	456253.8	1846.4	LA	3	0	0
02040201	Crosswicks-Neshaminy	347995.5	1408.3	NJ,PA	2	0	0
04120104	Niagara	871679.6	3527.6	CN,NY	2	0	0
05030201	Little Muskingum-Middle Island	1161545.0	4700.6	OH,WV	2	0	0
07090002	Middle Rock	1172085.4	4743.3	IL,WI	2	0	0
07120005	Upper Illinois	644077.9	2606.5	IL	2	0	0
08090301	East Central Louisiana Coastal	1728228.3	6993.9	LA	2	0	0
12020003	Lower Neches	709968.8	2873.1	TX	2	0	0
12040204	West Galveston Bay	776232.4	3141.3	TX	2	0	0
18070106	San Gabriel	579966.3	2347.0	CA	2	0	0
01090001	Charles	955681.2	3867.5	MA	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	1	0	0
02060002	Chester-Sassafras	833436.9	3372.8	DE,MD,PA	1	0	0
03050107	Tyger	517390.6	2093.8	SC	1	0	0
03050111	Lake Marion	351158.0	1421.1	SC	1	0	0
03050204	South Fork Edisto	555149.8	2246.6	SC	1	0	0
03090206	Florida Southeast Coast	2352752.2	9521.3	FL	1	0	0
03160103	Buttahatchee	553396.1	2239.5	AL,MS	1	0	0
03160112	Upper Black Warrior	797270.7	3226.4	AL	1	0	0

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
03160113	Lower Black Warrior	929969.4	3763.5	AL	1	0	0
04060101	Pere Marquette-White	1333169.6	5395.1	MI	1	0	0
04080201	Tittabawassee	926364.9	3748.9	MI	1	0	0
04110003	Ashtabula-Chagrin	401605.3	1625.2	OH,PA	1	0	0
04120103	Buffalo-Eighteenmile	457151.3	1850.0	NY	1	0	0
04120200	Lake Erie	6483450.8	26237.6	CN,MI,NY,OH,PA	1	0	0
04130001	Oak Orchard-Twelvemile	685684.0	2774.9	CN,NY	1	0	0
04150403	Winooski River	680464.2	2753.7	VT	1	0	0
05020003	Upper Monongahela	296728.7	1200.8	PA,WV	1	0	0
05030101	Upper Ohio	1271402.1	5145.2	OH,PA,WV	1	0	0
05040006	Licking	499187.6	2020.1	OH	1	0	0
05050008	Lower Kanawha	591554.2	2393.9	WV	1	0	0
05080001	Upper Great Miami, Indiana, Ohio	1607903.9	6507.0	IN,OH	1	0	0
05080002	Lower Great Miami, Indiana, Ohio	883871.2	3576.9	IN,OH	1	0	0
05120201	Upper White	1740657.8	7044.2	IN	1	0	0
05140101	Silver-Little Kentucky	807385.6	3267.4	IN,KY	1	0	0
07120003	Chicago	419754.7	1698.7	IL,IN	1	0	0
07120006	Upper Fox	988245.7	3999.3	IL,WI	1	0	0
07140106	Big Muddy	1526746.1	6178.5	IL	1	0	0
08070201	Bayou Sara-Thompson	444709.9	1799.7	LA,MS	1	0	0
10190004	Clear	365027.3	1477.2	CO	1	0	0
11030017	Upper Walnut River	620982.8	2513.0	KS	1	0	0
11110104	Robert S. Kerr Reservoir	1128010.3	4564.9	AR,OK	1	0	0
11130303	Middle Washita	1605161.6	6495.9	OK	1	0	0
12030102	Lower West Fork Trinity	969001.7	3921.4	TX	1	0	0
12040201	Sabine Lake	636218.6	2574.7	LA,TX	1	0	0
12070104	Lower Brazos	1051241.4	4254.2	TX	1	0	0
12110201	North Corpus Christi Bay	111266.8	450.3	TX	1	0	0
12110202	South Corpus Christi Bay	322454.2	1304.9	TX	1	0	0
16020204	Jordan	520846.5	2107.8	UT	1	0	0
17020010	Upper Columbia-Entiat	958508.9	3878.9	WA	1	0	0
17050114	Lower Boise	850233.1	3440.8	ID	1	0	0
17110012	Lake Washington	388533.5	1572.3	WA	1	0	0
18050002	San Pablo Bay	784983.8	3176.7	CA	1	0	0
18070102	Santa Clara	1040515.7	4210.8	CA	1	0	0
18070203	Santa Ana	1084241.9	4387.8	CA	1	0	0

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
PCE Monitoring Sites Only (n = 47 HUCs)							
02020004	Mohawk	1632666.9	6607.2	NY	0	1	1
02040105	Middle Delaware-Musconetcong	869995.3	3520.8	NJ,PA	0	1	3
02050205	Pine	627641.5	2540.0	PA	0	1	2
02050206	Lower West Branch Susquehanna	1158170.9	4687.0	PA	0	1	3
02050301	Lower Susquehanna-Penns	926808.1	3750.7	PA	0	1	6
02070004	Conococheague-Opequon	1457399.0	5897.9	MD,PA,VA,WV	0	2	6
04010201	St. Louis	1882043.1	7616.4	MN,WI	0	1	4
04010302	Bad-Montreal	832709.3	3369.9	MI,WI	0	1	4
04030101	Manitowoc-Sheboygan	1043247.9	4221.9	WI	0	1	4
04030204	Lower Fox	414795.8	1678.6	WI	0	1	3
04040002	Pike-Root	267751.0	1083.5	IL,WI	0	1	4
04050001	St. Joseph	3016829.4	12208.7	IN,MI	0	1	4
04050003	Kalamazoo	1300194.9	5261.7	MI	0	1	1
04080206	Saginaw	160773.8	650.6	MI	0	1	4
04090003	Clinton	510065.3	2064.2	MI	0	1	4
04090004	Detroit	567874.0	2298.1	CN,MI	0	1	4
04100009	Lower Maumee	689823.7	2791.6	OH	0	9	17
04100012	Huron-Vermilion	488453.3	1976.7	OH	0	1	3
04110001	Black-Rocky	572567.0	2317.1	OH	0	1	1
04110002	Cuyahoga	519309.5	2101.6	OH	0	1	3
04130003	Lower Genesee	682891.3	2763.6	NY	0	1	4
04140101	Irondequoit-Ninemile	445757.0	1803.9	NY	0	1	3
04140203	Oswego	93064.4	376.6	NY	0	1	4
06030003	Upper Elk	821468.2	3324.4	AL,TN	0	4	8
07090004	Sugar	486750.9	1969.8	IL,WI	0	1	3
07140102	Meramec	1375977.1	5568.4	MO	0	4	7
08040302	Castor	612659.1	2479.3	LA	0	2	3
10300102	Lower Missouri-Moreau	2176536.7	8808.1	MO	0	1	1
11140207	Lower Red-Lake Iatt	912489.8	3692.7	LA	0	3	3
11140209	Black Lake Bayou	579878.2	2346.7	LA	0	1	2
12100303	Lower San Antonio	950344.1	3845.9	TX	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
14030005	Upper Colorado-Kane Springs	1455869.5	5891.7	CO,UT	0	5	9
14060008	Lower Green	1195181.0	4836.7	UT	0	1	2

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
15010008	Upper Virgin	1397207.4	5654.3	UT	0	2	2
15060106	Lower Salt	666211.2	2696.1	AZ	0	5	12
15070102	Aqua Fria	1758350.5	7115.8	AZ	0	7	11
17090001	Middle Fork Willamette	874861.9	3540.4	OR	0	1	1
17090002	Coast Fork Willamette	426542.2	1726.2	OR	0	2	2
17090003	Upper Willamette	1198500.4	4850.2	OR	0	3	5
17090004	Mckenzie	857010.6	3468.2	OR	0	4	5
21010005	Eastern Puerto Rico	914478.3	3700.8	PR	0	1	2

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14618 **Table_Apx D-3. Occurrence of PCE Releases (Facilities) and Monitoring Sites By HUC-12.**

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
Co-located PCE Releases (Facilities) and Monitoring Sites (n = 1 HUC)							
040400010509	Willow Creek-Burns Ditch	13501.8	54.6	IN	1	1	1
PCE Releases (Facilities) Only (n =81 HUCs)							
010900010402	Outlet Saugus River	17633.5	71.4	MA	1	0	0
020301030802	Peckman River-Passaic River	22354.8	90.5	NJ	1	0	0
020301040204	Morses Creek-Arthur Kill	18931.5	76.6	NJ,NY	1	0	0
020301050306	Devils Brook	9890.5	40.0	NJ	1	0	0
020301050312	Lower Millstone River	31839.8	128.8	NJ	1	0	0
020301050504	Green Brook	32590.3	131.9	NJ	1	0	0
020301050505	Lawrence Brook	29837.9	120.8	NJ	1	0	0
020402010202	West Branch Neshaminy Creek	15964.6	64.6	PA	1	0	0
020402010404	Van Sciver Lake-Delaware River	16914.3	68.5	NJ,PA	1	0	0
020600020202	Little Elk Creek	26942.3	109.0	MD,PA	1	0	0
020600030902	Dead Run-Gywnns Falls	31450.3	127.3	MD	3	0	0
030501070305	Lower South Tyger River	29288.0	118.5	SC	1	0	0
030501110109	Lake Marion-Santee River	165146.0	668.3	SC	1	0	0
030502040108	Lower Shaw Creek	32220.3	130.4	SC	1	0	0
030902061003	Lake Worth Inlet-Boynton Inlet Frontal	39017.9	157.9	FL	1	0	0
031601030202	Cannon Mill Creek-Beaver Creek	28263.4	114.4	AL	1	0	0
031601120101	Headwaters Valley Creek	34201.6	138.4	AL	1	0	0
031601130204	Goose Pond-Black Warrior River	25818.5	104.5	AL	1	0	0
040500060712	Lloyd Bayou-Grand River	31929.6	129.2	MI	1	0	0
040601010904	White Lake-White River	39040.6	158.0	MI	1	0	0
040802010604	Prairie Creek-Tittabawassee River	25251.7	102.2	MI	1	0	0
041100030504	Doan Brook-Frontal Lake Erie	28193.7	114.1	OH	1	0	0
041201030401	Smoke Creek	21267.2	86.1	NY	1	0	0
041201040604	City of North Tonawanda-Niagara River	8541.4	34.6	NY	1	0	0
041201040605	Niagara Falls-Niagara River	21666.5	87.7	CN,NY	1	0	0
041202000300	Lake Erie	6359988.3	25738.0	CN,MI,NY,OH,PA	1	0	0
041300010703	Headwaters Eighteenmile Creek	15270.7	61.8	NY	1	0	0
041504030101	Headwaters Stevens Branch	22103.3	89.5	VT	1	0	0
050200030307	Cobun Creek-Monongahela River	21730.5	87.9	WV	1	0	0
050301011103	Carpenter Run-Ohio River	23323.8	94.4	OH,PA,WV	1	0	0
050302011004	Haynes Run-Ohio River	19386.4	78.5	OH,WV	2	0	0
050400060409	Beaver Run-South Fork Licking River	19150.9	77.5	OH	1	0	0

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
050500080304	Scary Creek-Kanawha River	20472.1	82.8	WV	1	0	0
050800012005	Poplar Creek-Great Miami River	34854.0	141.1	OH	1	0	0
050800020105	Town of Oakwood-Great Miami River	16944.9	68.6	OH	1	0	0
051202011205	Dollar Hide Creek-White River	30882.8	125.0	IN	1	0	0
051401010903	Mill Creek Cutoff	20966.7	84.8	KY	1	0	0
070400010206	Town of Pine Bend	31880.6	129.0	MN	1	0	0
070900021402	Delavan Lake	22265.1	90.1	WI	1	0	0
070900021502	City of Beloit-Rock River	30612.6	123.9	IL,WI	1	0	0
071200030407	Grand Calumet River-Little Calumet River	17191.8	69.6	IL,IN	1	0	0
071200040905	Des Plaines River	23822.3	96.4	IL	3	0	0
071200050106	Walley Run-Aux Sable Creek	12878.4	52.1	IL	1	0	0
071200050705	Bills Run-Illinois River	33003.8	133.6	IL	1	0	0
071200061206	Jelkes Creek-Fox River	25551.9	103.4	IL	1	0	0
071401060407	Ewing Creek	14114.5	57.1	IL	1	0	0
080702010402	Devils Swamp-Bayou Baton Rouge	17328.4	70.1	LA	1	0	0
080702040101	Bayou Francois	16194.6	65.5	LA	1	0	0
080702040103	Grand Goudine Bayou-New River	17644.3	71.4	LA	1	0	0
080702040302	Hope Canal-Pipeline Canal	18663.6	75.5	LA	1	0	0
080802060301	Maple Fork-Bayou d'Inde	22308.4	90.3	LA	2	0	0
080802060302	Bayou Verdine-Calcasieu River	24546.0	99.3	LA	1	0	0
080802060303	Prien Lake-Calcasieu River	29606.9	119.8	LA	1	0	0
080903010307	Town of Westwego-Main Canal	39569.2	160.1	LA	2	0	0
101900030304	Cherry Creek-South Platte River	35554.2	143.9	CO	5	0	0
101900040404	Outlet Clear Creek	19355.3	78.3	CO	1	0	0
110300120204	Headwaters Dry Turkey Creek	30940.1	125.2	KS	1	0	0
110300170403	Constant Creek-Walnut River	28347.5	114.7	KS	1	0	0
111101040611	Massard Creek	10720.0	43.4	AR	1	0	0
111303030708	Outlet Caddo Creek	26104.7	105.6	OK	1	0	0
120200030406	Union Canal-Neches River	26733.6	108.2	TX	1	0	0
120200030407	Grays Bayou-Neches River	39760.5	160.9	TX	1	0	0
120301020206	Brogden Branch-Town Creek	14887.3	60.3	TX	1	0	0
120401040703	Vince Bayou-Buffalo Bayou	38130.8	154.3	TX	3	0	0
120401040706	Goose Creek-Frontal Galveston Bay	37289.7	150.9	TX	1	0	0
120402010300	Salt Bayou	212334.8	859.3	TX	1	0	0
120402040100	Clear Creek-Frontal Galveston Bay	190566.3	771.2	TX	1	0	0

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
120402040400	Mustang Bayou	183973.7	744.5	TX	1	0	0
120701040505	Outlet Barzos River	35803.4	144.9	TX	1	0	0
121102010001	Rincon Bayou	28406.5	115.0	TX	1	0	0
121102020107	Tule Lake	12284.3	49.7	TX	1	0	0
160202040304	City Creek	11166.6	45.2	UT	1	0	0
170200100307	Rainey Spring-Columbia River	21142.9	85.6	WA	1	0	0
170501140403	Crane Creek-Boise River	18624.7	75.4	ID	1	0	0
171100120301	Bear Creek	30140.7	122.0	WA	1	0	0
180500020801	San Pablo Bay Estuaries	85721.1	346.9	CA	1	0	0
180701020507	Gorman Creek	23547.6	95.3	CA	1	0	0
180701060102	Lower Dominguez Channel	36125.6	146.2	CA	1	0	0
180701060701	Long Beach Harbor	33394.5	135.1	CA	1	0	0
180701060703	San Pedro Bay	40623.1	164.4	CA	1	0	0
180702031003	Greenville Banning Channel-Santa Ana River	22359.3	90.5	CA	1	0	0
PCE Monitoring Sites Only (n = 67 HUCs)							
020200040908	Lower Canajoharie Creek	13216.2	53.5	NY	0	1	1
020401050911	Buck Creek-Delaware River	15442.9	62.5	NJ,PA	0	1	3
020502050607	Furnace Run-Pine Creek	27631.1	111.8	PA	0	1	2
020502061103	Beaver Run-Chillisquaque Creek	26019.5	105.3	PA	0	1	3
020503010603	Lower West Branch Mahantango Creek	13445.1	54.4	PA	0	1	6
020700040702	Dennis Creek-Back Creek	32533.8	131.7	PA	0	1	4
020700041009	Sharmans Branch-Antietam Creek	36619.8	148.2	MD	0	1	2
040102011503	City of Cloquet-Saint Louis River	36671.5	148.4	MN	0	1	4
040103020702	Camerons Creek-Bad River	13498.0	54.6	WI	0	1	4
040301010605	Manitowoc River	11648.4	47.1	WI	0	1	4
040302040405	City of Green Bay-Fox River	19046.2	77.1	WI	0	1	3
040400010603	Calumet River-Frontal Lake Michigan	34563.8	139.9	IL,IN	0	1	4
040400020101	Wind Point-Frontal Lake Michigan	16148.3	65.3	WI	0	1	4
040500012210	City of Niles-Saint Joseph River	8758.5	35.4	MI	0	1	4
040500030911	Peach Orchid Creek-Kalamazoo River	15046.6	60.9	MI	0	1	1
040500060708	Jubb Bayou-Grand River	11389.8	46.1	MI	0	1	4
040802060201	Crow Island-Saginaw River	33918.2	137.3	MI	0	1	4
040900030402	Cranberry Marsh Drain-Clinton River	21236.7	85.9	MI	0	1	4
040900040406	Ashcroft Sherwood Drain-River Rouge	12735.6	51.5	MI	0	1	4

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
041000090509	Lower Beaver Creek	10727.3	43.4	OH	0	1	2
041000090510	Lick Creek-Maumee River	14952.3	60.5	OH	0	1	2
041000090603	Haskins Road Ditch-Maumee River	10054.5	40.7	OH	0	1	1
041000090804	Heilman Ditch-Swan Creek	23569.6	95.4	OH	0	1	2
041000090903	Crooked Creek-Maumee River	12075.0	48.9	OH	0	2	5
041000090904	Delaware Creek-Maumee River	10576.9	42.8	OH	0	3	5
041000120204	Town of Vermilion-Vermilion River	17985.5	72.8	OH	0	1	3
041100010203	Rocky River	16199.9	65.6	OH	0	1	1
041100020602	Village of Independence-Cuyahoga River	10848.3	43.9	OH	0	1	3
041300030704	Genesee River	14336.9	58.0	NY	0	1	4
041401010703	Allen Creek	20188.5	81.7	NY	0	1	3
041402030204	Oswego River	11026.9	44.6	NY	0	1	4
060300030201	Bradley Creek	30268.8	122.5	TN	0	4	8
070400010102	Lock and Dam Number Three-Mississippi River	40106.3	162.3	MN,WI	0	1	1
070900040201	Badger Mill Creek	21661.8	87.7	WI	0	1	3
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
080403020401	Caney Creek Reservoir	26803.0	108.5	LA	0	2	3
103001020709	Black Branch-Perche Creek	12012.4	48.6	MO	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
111402070401	Sibley Lake	24862.2	100.6	LA	0	3	3
111402090404	Grand Bayou	34707.7	140.5	LA	0	1	2
121003030306	Salt Creek-Ecleto Creek	18817.5	76.2	TX	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
140300050205	Outlet Courthouse Wash	18177.4	73.6	UT	0	1	1
140300050307	Negro Bill Canyon-Colorado River	19473.5	78.8	UT	0	1	2
140300051001	Little Canyon-Colorado River	32843.3	132.9	UT	0	2	4
140300051002	Bull Canyon-Colorado River	32166.0	130.2	UT	0	1	2
140600080708	Upheaval Canyon-Green River	20259.5	82.0	UT	0	1	2
150100080109	Lower North Fork Virgin River	34874.9	141.1	UT	0	2	2
150601060202	Upper Indian Bend Wash	27058.2	109.5	AZ	0	1	3

HUC8	Name	Acres	Square km	States	No. of Facilities	No. of Monitoring Sites	No. of Monitoring Samples in HUC
150601060306	City of Phoenix-Salt River	87618.1	354.6	AZ	0	2	4
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
170900011003	Mill Race-Middle Fork Willamette River	12666.2	51.3	OR	0	1	1
170900020405	Papenfus Creek-Coast Fork Willamette River	17460.5	70.7	OR	0	2	2
170900030601	Sring Creek-Willamette River	29305.8	118.6	OR	0	3	5
170900040705	Camp Creek	16999.1	68.8	OR	0	1	1
170900040706	Walterville Canal-McKenzie River	33735.2	136.5	OR	0	3	4
210100050503	Cienaga de las Cucharillas Drainage Watershed	6557.0	26.5	PR	0	1	2

14619

14620

Table_Apx D-4 provides a list of states/territories with facilities that have releases of PCE and/or monitoring sites for the year of 2016

Table_Apx D-4. States with Monitoring Sites or Facilities in 2016

State Name	PCE Facility[a]	PCE Monitoring Site	PCE Facility or Monitoring Site
Alabama	X		X
Arizona		X	X
Arkansas	X		X
California	X		X
Colorado	X		X
Florida	X		X
Idaho	X		X
Illinois	X		X
Indiana	X	X	X
Kansas	X	X	X
Kentucky	X		X
Louisiana	X	X	X
Maryland	X	X	X
Massachusetts	X		X
Michigan	X	X	X
Minnesota	X	X	X
Missouri		X	X
New Jersey	X	X	X
New Mexico		X	X
New York	X	X	X
Ohio	X	X	X
Oklahoma	X		X
Oregon		X	X
Pennsylvania	X	X	X
Puerto Rico		X	X
South Carolina	X		X
Tennessee		X	X
Texas	X	X	X
Utah	X	X	X
Vermont	X		X
Washington	X		X
West Virginia	X		X
Wisconsin	X	X	X
Total	***27***	***19***	***33***

a. PCE Facility is based on the location of the facility mapped. For indirect releasers, the receiving facility was mapped if known.

Appendix E BENCHMARK DOSE ANALYSIS

The following is a summary of the cancer dose response modeling from Appendix D of U.S. EPA (2012e).

E.1 Model Selection Details for Tumor Sites from JISA (1993)

Table_Apx E-1. Model predictions for hepatocellular tumors in male mice (JISA, 1993)[a], using several dose metrics and multistage cancer model

Model stages	Goodness of fit			BMD_{10}	$BMDL_{10}$	Conclusion
	p-value[b]	Largest standardized residual(s)	AIC			
Total liver oxidative metabolism (mg/kg$^{0.75}$-day)						
One	**0.24**	**1.1, low-dose −1.2, mid-dose**	**239.7**	**2.9**	**2.1**	All three fits were adequate by conventional criteria.[b] There was no statistical improvement in adding higher-order coefficients (using likelihood ratio test); one-stage fit was selected.
Two	0.16	−0.7, control 1.1, low-dose	240.8	6.4	2.2	
Three	0.18	−0.7, control 1.0, low-dose	240.6	6.5	2.2	
TCA AUC in liver (mg-hr/L-day)						
One	**0.25**	**1.0, low-dose −1.2, mid-dose**	**239.7**	**97.1**	**68.8**	All three fits were adequate by conventional criteria.[b] There was no statistical improvement in adding higher-order coefficients (using likelihood ratio test); one-stage fit was selected.
Two	0.17	−0.7, control 1.1, low-dose	240.8	209.9	72.8	
Three	0.19	−0.7, control 1.0, low-dose	240.6	213.9	73.8	
Administered PCE concentration (ppm)						
One	**0.27**	**1.2, low-dose −1.0, mid-dose**	**239.5**	**3.9**	**2.7**	All three fits were adequate by conventional criteria.[b] There was no statistical improvement in adding higher-order coefficients (using likelihood ratio test); one-stage fit was selected.
Two	0.16	−0.8, control 1.1, low-dose	240.9	9.0	2.8	
Three	0.17	−0.8, control 1.1, low-dose	240.8	8.2	2.9	

[a] Incidence data and human equivalent continuous exposure estimates provided in Table 3-6.

[b] Goodness-of-fit *p*-values <0.05 for a preferred model, or <0.10 when considering many models, fail to meet conventional goodness-of-fit criteria. In addition, visual fit and residuals (within ±2 units) are considered. Best-fit model is highlighted in bold; output for best-fit models provided in following pages.

AIC = Akaike's Information Criteria, BMD = benchmark dose, BMDL = lower bound benchmark dose.

14641 ## E.1.1 Modeling Output for Male Mice, Hepatocellular Tumors (JISA, 1993)

14642

14643 ### E.1.1.1 With total oxidative metabolism in liver as dose metric

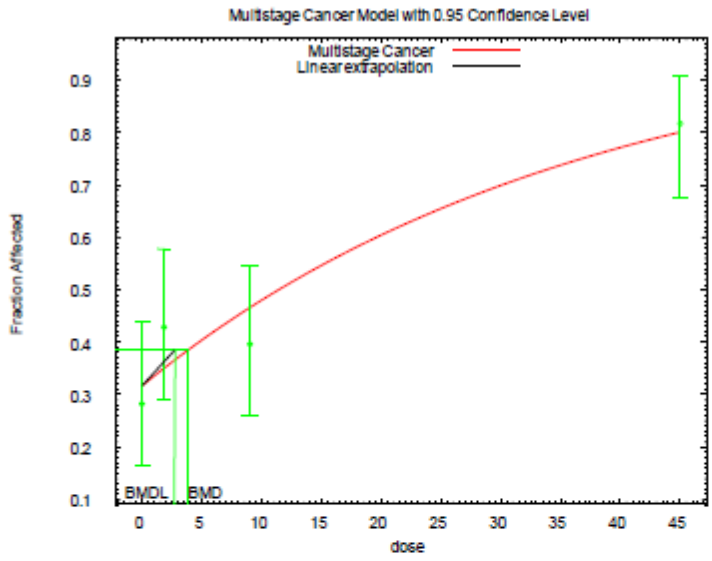

Figure D-1 One-degree multistage model fit to hepatocellular tumors in male mice (JISA, 1993), with BMD and BMDL at 10% extra risk, using total oxidative metabolism in liver (mg/kg$^{0.75}$-day).

```
=================================================================
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
=================================================================

  The form of the probability function is:

  P[response] = background + (1-background)*[1-EXP(
               -beta1*dose^1)]

  The parameter betas are restricted to be positive


  Dependent variable = Response
  Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 2
Total number of specified parameters = 0
Degree of polynomial = 1
```

14644

Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008

Default Initial Parameter Values
Background = 0.285739
Beta(1) = 0.0395068

Asymptotic Correlation Matrix of Parameter Estimates

	Background	Beta(1)
Background	1	-0.53
Beta(1)	-0.53	1

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval Lower Conf. Limit	Upper Conf. Limit
Background	0.301268	*	*	*
Beta(1)	0.0361674	*	*	*

* - Indicates that this value is not calculated.

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	P-value
Full model	-116.442	4			
Fitted model	-117.844	2	2.80477	2	0.246
Reduced model	-132.99	1	33.0977	3	<.0001
AIC:	239.688				

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Size	Scaled Residual
0.0000	0.3013	13.858	13.000	46	-0.276
2.2500	0.3559	17.438	21.000	49	1.063
8.3000	0.4825	23.158	19.000	48	-1.201
33.6000	0.7927	38.844	40.000	49	0.408

Chi^2 = 2.81 d.f. = 2 P-value = 0.2448

Benchmark Dose Computation

Specified effect = 0.1
Risk Type = Extra risk
Confidence level = 0.95
BMD = 2.91314
BMDL = 2.06187
BMDU = 4.49484

Taken together, (2.06187, 4.49484) is a 90 % two-sided confidence interval for the BMD

Multistage Cancer Slope Factor = 0.0484996

14645

E.1.1.2 With TCA AUC in liver as dose metric

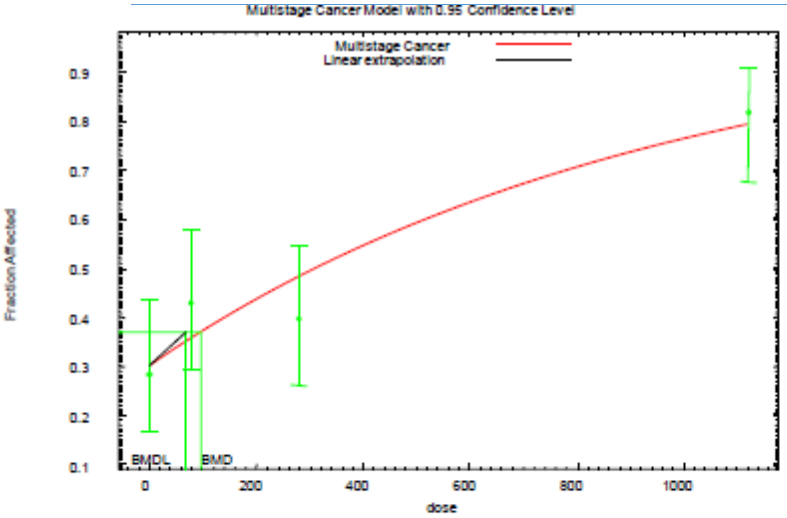

Figure D-2. One-degree multistage model fit to hepatocellular tumors in male mice (JISA, 1993), with BMD and BMDL at 10% extra risk, using TCA AUC as dose metric (mg-hr/L-d).

```
=================================================================
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
=================================================================

  The form of the probability function is:

  P[response] = background + (1-background)*[1-EXP(
                -beta1*dose^1)]

  The parameter betas are restricted to be positive


  Dependent variable = Response
  Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 2
Total number of specified parameters = 0
Degree of polynomial = 1


Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008


                Default Initial Parameter Values
                   Background =     0.283935
                      Beta(1) =    0.00118591


          Asymptotic Correlation Matrix of Parameter Estimates
```

	Background	Beta(1)
Background	1	-0.53
Beta(1)	-0.53	1

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval Lower Conf. Limit	Upper Conf. Limit
Background	0.299803	*	*	*
Beta(1)	0.0010848	*	*	*

* - Indicates that this value is not calculated.

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	P-value
Full model	-116.442	4			
Fitted model	-117.833	2	2.78303	2	0.2487
Reduced model	-132.99	1	33.0977	3	<.0001
AIC:	239.666				

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Size	Scaled Residual
0.0000	0.2998	13.791	13.000	46	-0.255
78.4900	0.3570	17.491	21.000	49	1.046
279.7000	0.4831	23.186	19.000	48	-1.209
1121.1000	0.7925	38.832	40.000	49	0.411

Chi^2 = 2.79 d.f. = 2 P-value = 0.2477

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 97.1242

BMDL = 68.7915

BMDU = 149.76

Taken together, (68.7915, 149.76) is a 90 % two-sided confidence interval for the BMD

Multistage Cancer Slope Factor = 0.00145367

14648

14649

14650

E.1.1.3 With administered PCE concentration (ppm) as dose metric

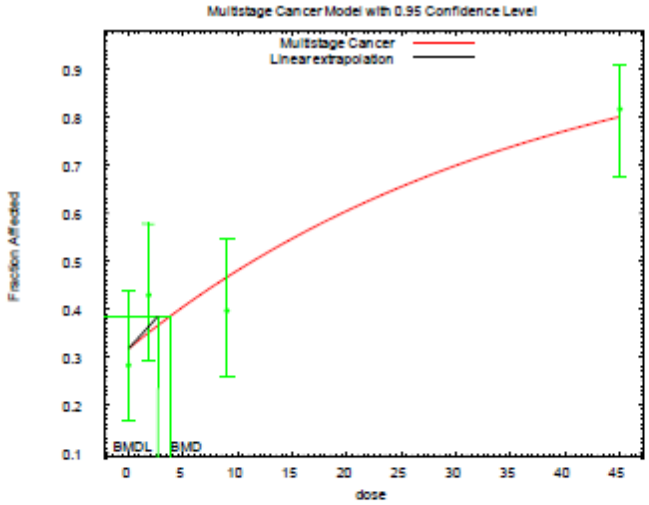

Figure D-3. One-degree multistage model fit to hepatocellular tumors in male mice (JISA, 1993), with BMD and BMDL at 10% extra risk, using administered tetrachloroethylene concentration (ppm).

```
=================================================================
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
=================================================================

   The form of the probability function is:

   P[response] = background + (1-background)*[1-EXP(-beta1*dose^1)]

   The parameter betas are restricted to be positive


   Dependent variable = Response
   Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 2
Total number of specified parameters = 0
Degree of polynomial = 1


Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008


                 Default Initial Parameter Values
                    Background =     0.307193
                       Beta(1) =    0.0290723
```

14651

```
          Asymptotic Correlation Matrix of Parameter Estimates

            Background      Beta(1)

Background           1        -0.48

   Beta(1)       -0.48             1


                          Parameter Estimates

                                                          95.0% Wald Confidence
Interval
       Variable         Estimate        Std. Err.     Lower Conf. Limit   Upper Conf.
Limit
     Background         0.316506             *                 *                 *
        Beta(1)        0.0278229             *                 *                 *

* - Indicates that this value is not calculated.


                      Analysis of Deviance Table

      Model      Log(likelihood)  # Param's  Deviance  Test d.f.   P-value
     Full model        -116.442          4
   Fitted model        -117.738          2       2.59226      2         0.2736
  Reduced model         -132.99          1       33.0977      3         <.0001

          AIC:         239.476


                           Goodness of Fit
                                                                  Scaled
    Dose     Est._Prob.    Expected    Observed     Size        Residual
 ----------------------------------------------------------------------
  0.0000      0.3165       14.559     13.000          46        -0.494
  1.8000      0.3493       17.116     21.000          49         1.164
  9.0000      0.4655       22.344     19.000          48        -0.968
 45.0000      0.8001       39.206     40.000          49         0.284

 Chi^2 = 2.62      d.f. = 2        P-value = 0.2704


   Benchmark Dose Computation

Specified effect =           0.1

Risk Type        =      Extra risk

Confidence level =          0.95

             BMD =       3.85613

            BMDL =       2.70709

            BMDU =       5.98909

Taken together, (2.70709, 5.98909) is a 90      % two-sided confidence
interval for the BMD

Multistage Cancer Slope Factor =       0.03694
```

Table_Apx E-2. Model predictions for hepatocellular tumors in female mice (JISA, 1993)[a], using several dose metrics and multistage cancer model

Model stage	Goodness of fit			BMD_{10}	$BMDL_{10}$	Comments	Conclusions
	p-value[b]	Largest standardized residual(s)	AIC				
Total liver oxidative metabolism (mg/kg$^{0.75}$-day)							
One-stage	0.14	-1.4, mid-dose	154.9	3.7	2.8	Adequate fit	Selected two-degree multistage, based on likelihood ratio test.
Two-stage	**0.82**	**-0.18, low-dose**	**152.8**	**8.4**	**4.0**	Adequate fit	
Three-stage	0.82	-0.18, low-dose	152.8	8.4	3.9	Adequate fit	
TCA AUC in liver (mg-hr/L-day)							
One-stage	0.13	-1.4, mid-dose	155.1	129	98	Adequate fit	Selected two-

Two-stage	**0.82**	**-0.18, low-dose**	**152.9**	**292**	**141**	Adequate fit	degree multistage, based on likelihood ratio test.
Three-stage	0.82	-0.18, low-dose	152.9	292	139	Adequate fit	
Administered PCE concentration (ppm)							
One-stage	**0.36**	**−1.1, mid-dose**	**153.0**	**5.0**	**3.8**	Adequate fit	Selected one-degree multistage; no statistical improvement in adding higher order parameters.
Two-, three-stage	0.83	−0.1, low-dose	152.8	9.7	4.3	Identical fits resulted from both models	

[a] Incidence data provided in Table 5-13, and dose metrics provided in Table 3-6; both are included in following output.

[b] Values <0.05 for a preferred model, or <0.10 when considering a suite of models, fail to meet conventional goodness-of-fit criteria. In addition, visual fit and residuals (within ±2 units) are considered. Best-fit model is highlighted in bold; output for best-fit models provided in following pages.

E.1.2 Modeling Output for Female Mice, Hepatocellular Tumors (JISA, 1993)

E.1.2.1 With total oxidative metabolism in liver as dose metric

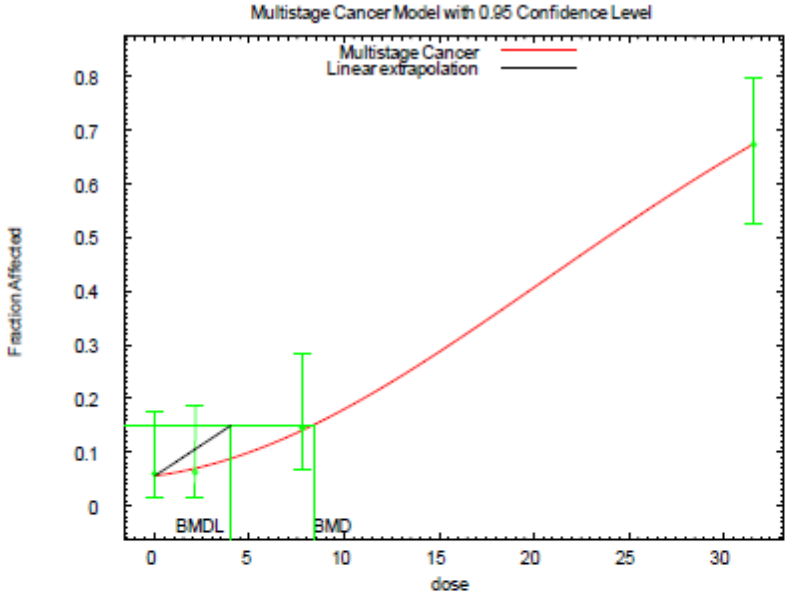

Figure D-4. Two-degree multistage model fit to hepatocellular tumors in female mice (JISA, 1993), with BMD and BMDL at 10% extra risk.

```
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
        Input Data File: C:\Usepa\BMDS21\msc_JISA1993_MF_HepAC_oxmet_Perc3_MultiCanc2_0.1.(d)
```

```
  The form of the probability function is:

  P[response] = background + (1-background)*[1-EXP(
               -beta1*dose^1-beta2*dose^2)]

  The parameter betas are restricted to be positive


  Dependent variable = Response
  Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 3
Total number of specified parameters = 0
Degree of polynomial = 2


Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008
```

Default Initial Parameter Values
Background = 0.0554081
Beta(1) = 0.00569729
Beta(2) = 0.000883583

Asymptotic Correlation Matrix of Parameter Estimates

	Background	Beta(1)	Beta(2)
Background	1	-0.69	0.59
Beta(1)	-0.69	1	-0.97
Beta(2)	0.59	-0.97	1

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval Lower Conf. Limit	Upper Conf. Limit
Background	0.0566119	*	*	*
Beta(1)	0.00500318	*	*	*
Beta(2)	0.000907152	*	*	*

* - Indicates that this value is not calculated.

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	P-value
Full model	-73.398	4			
Fitted model	-73.4233	3	0.050713	1	0.8218
Reduced model	-106.26	1	65.7232	3	<.0001

AIC: 152.847

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Size	Scaled Residual
0.0000	0.0566	2.831	3.000	50	0.104
2.1300	0.0704	3.311	3.000	47	-0.177
7.8000	0.1414	6.789	7.000	48	0.087
31.6000	0.6744	33.048	33.000	49	-0.015

Chi^2 = 0.05 d.f. = 1 P-value = 0.8230

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 8.36661

BMDL = 4.02336

BMDU = 11.6726

Taken together, (4.02336, 11.6726) is a 90 % two-sided confidence interval for the BMD

Multistage Cancer Slope Factor = 0.0248549

14667

E.1.2.2 With TCA AUC in liver as dose metric

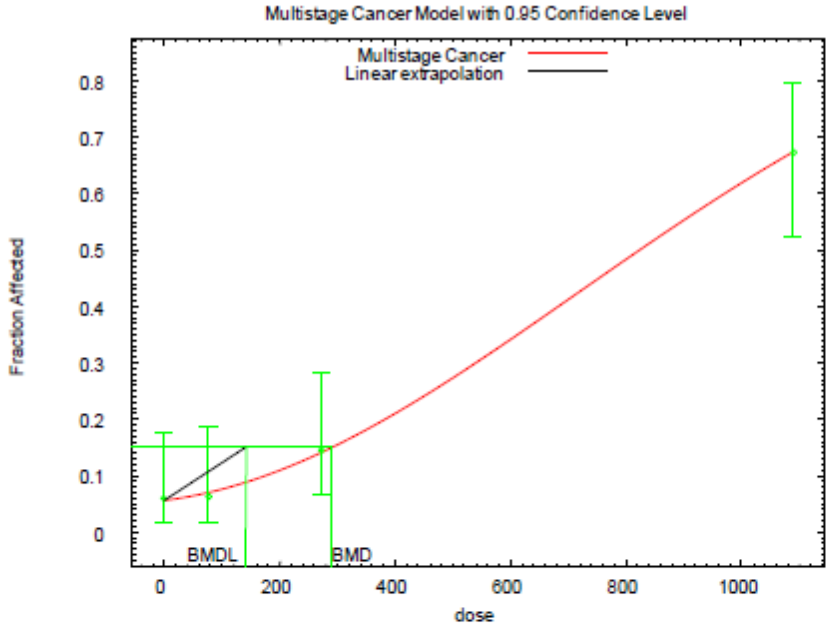

Figure D-5. Two-degree multistage model fit to hepatocellular tumors in female mice (JISA, 1993), with BMD and BMDL at 10% extra risk.

```
=============================================================================
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
        Input Data File: C:\Usepa\BMDS21\msc_JISA1993_MF_HepAC_tcaAUC_Perc3_MultiCanc2_0.1.(d)
=============================================================================

   The form of the probability function is:

   P[response] = background + (1-background)*[1-EXP(
                -beta1*dose^1-beta2*dose^2)]

   The parameter betas are restricted to be positive


   Dependent variable = Response
   Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 3
Total number of specified parameters = 0
Degree of polynomial = 2


Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008


                 Default Initial Parameter Values
                    Background =     0.0553149
                       Beta(1) =   0.000156854
```

Beta(2) = 7.50947e-007

Asymptotic Correlation Matrix of Parameter Estimates

	Background	Beta(1)	Beta(2)
Background	1	-0.69	0.6
Beta(1)	-0.69	1	-0.97
Beta(2)	0.6	-0.97	1

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval Lower Conf. Limit	Upper Conf. Limit
Background	0.0565811	*	*	*
Beta(1)	0.000135812	*	*	*
Beta(2)	7.71737e-007	*	*	*

* - Indicates that this value is not calculated.

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	P-value
Full model	-73.398	4			
Fitted model	-73.4249	3	0.0538645	1	0.8165
Reduced model	-106.26	1	65.7232	3	<.0001
AIC:	152.85				

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Size	Scaled Residual
0.0000	0.0566	2.829	3.000	50	0.105
76.9500	0.0706	3.320	3.000	47	-0.182
271.8000	0.1412	6.776	7.000	48	0.093
1089.6000	0.6745	33.051	33.000	49	-0.016

Chi^2 = 0.05 d.f. = 1 P-value = 0.8177

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 291.833

BMDL = 141.409

BMDU = 402.749

Taken together, (141.409, 402.749) is a 90 % two-sided confidence interval for the BMD

Multistage Cancer Slope Factor = 0.000707168

14670

E.1.2.3 With administered PCE concentration (ppm) as dose metric

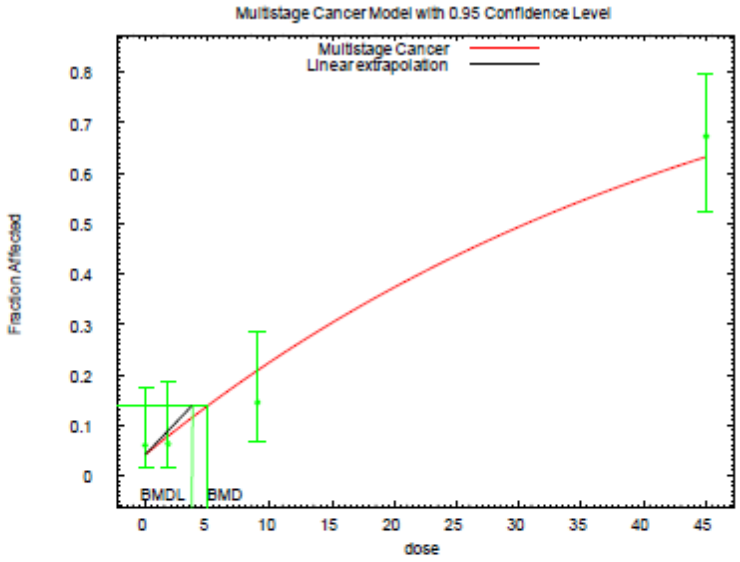

Figure D-6. One-degree multistage model fit to hepatocellular tumors in female mice (JISA, 1993), with BMD and BMDL at 10% extra risk.

```
================================================================
        Multistage Cancer Model. (Version: 1.7;  Date: 05/16/2008)
================================================================

   The form of the probability function is:

   P[response] = background + (1-background)*[1-EXP(-beta1*dose^1)]

   The parameter betas are restricted to be positive


   Dependent variable = Response
   Independent variable = Dose

Total number of observations = 4
Total number of records with missing values = 0
Total number of parameters in model = 2
Total number of specified parameters = 0
Degree of polynomial = 1


Maximum number of iterations = 250
Relative Function Convergence has been set to: 1e-008
Parameter Convergence has been set to: 1e-008


                Default Initial Parameter Values
                   Background =     0.0124442
```

Beta(1) = 0.0242761

Asymptotic Correlation Matrix of Parameter Estimates

	Background	Beta(1)
Background	1	-0.47
Beta(1)	-0.47	1

Parameter Estimates

Variable	Estimate	Std. Err.	95.0% Wald Confidence Interval Lower Conf. Limit	Upper Conf. Limit
Background	0.0427836	*	*	*
Beta(1)	0.0212108	*	*	*

* - Indicates that this value is not calculated.

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	P-value
Full model	-73.398	4			
Fitted model	-74.4575	2	2.11904	2	0.3466
Reduced model	-106.26	1	65.7232	3	<.0001
AIC:	152.915				

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Size	Scaled Residual
0.0000	0.0428	2.139	3.000	50	0.602
1.8000	0.0786	3.696	3.000	47	-0.377
9.0000	0.2091	10.038	7.000	48	-1.078
45.0000	0.6315	30.942	33.000	49	0.610

Chi^2 = 2.04 d.f. = 2 P-value = 0.3609

Benchmark Dose Computation

Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 4.96731

BMDL = 3.75394

BMDU = 6.8242

Taken together, (3.75394, 6.8242) is a 90 % two-sided confidence interval for the BMD

Multistage Cancer Slope Factor = 0.0266387

14673

14674

Appendix F Cancer Study Summaries

F.1 Epidemiological Data

This section is a synthesis of the findings from the older epidemiological literature, as presented in the 2012 IRIS Assessment (U.S. EPA 2012c) combined with the results of the newer studies described above. Epidemiological studies provide suggestive evidence for an association between PCE exposure and tumor development in humans. Tumor types in humans with varying degrees of supporting evidence for an association with PCE exposure include NHL, MM, and bladder, esophagus, lung, liver, cervical, and breast cancer according to (U.S. EPA 2012c) and references cited therein, as well as the newer studies (Purdue et al. 2017; Mattei et al. 2014; Silver et al. 2014; Vizcaya et al. 2013; Vlaanderen et al. 2013; Gallagher et al. 2011; Lipworth et al. 2011).

F.1.1 Bladder

(U.S. EPA 2012c) concluded that, with respect to bladder cancer, the pattern of results from the studies available at that time was consistent with an elevated risk for PCE of a relatively modest magnitude (i.e., a 10−40% increased risk). The effect estimates from five of the six studies with relatively high-quality exposure assessment methodologies ranged from 1.44 to 4.03 (U.S. EPA 2012c). An exposure-response gradient was observed in a large case-control study using a semiquantitative cumulative exposure assessment, with adjusted ORs of 0.8 (95% CI = 0.6-1.2), 1.3 (95% CI = 0.9-1.7), and 1.8 (95% CI = 1.2-2.7) for medium, high, and substantial exposure, respectively, compared to low exposure. A similar exposure-response pattern was not observed in a different study that examined exposure duration, in contrast with the previously described data based on varied exposure concentration. Relative risk estimates between bladder cancer risk and ever having a job title of dry cleaner or laundry worker in four large cohort studies ranged from 1.01 to 1.44. As expected, the results from the smaller studies are more variable and less precise, reflecting their reduced statistical power. Confounding by smoking is an unlikely explanation for the findings, given the included adjustment for smoking in several case-control studies (U.S. EPA 2012c),.

More recent studies provide little support for an association between bladder cancer and PCE exposure. The SMR was 0.84 (95% CI = 0.49-1.35) based on 17 observed deaths from bladder and other urinary cancers and 20.2 expected in the subset (n=5,830, sex and race combined) of a cohort of aircraft manufacturing workers judged based on detailed exposure assessment to have had routine or intermittent exposure to PCE while employed for at least 1 year between 1960 and 1996 at the Lockheed Martin aircraft manufacturing facilities in Burbank, California and followed for mortality experience through 2008 (Lipworth et al. 2011). Similarly, a cohort of workers employed 91 days or more at a microelectronics and business machine facility in New York state between 1969 and 2001 and followed through 2009 showed no association between cumulative PCE exposure score estimated from detailed exposure assessment and deaths from malignant neoplasms of the bladder and other urinary organs (HR = 0.89, 95% CI = 0.37-2.13) relative to internal referents (Silver et al. 2014). A large case-control study of incident bladder cancer cases extracted from the NOCCA cohort, which relied on a standardized job exposure matrix to estimate cumulative occupational exposure to PCE (and other agents), reported HRs of 1.00 (95% CI = 0.92-1.09, 747 cases/3,560 controls), 1.12 (95% CI = 1.02-1.23, 660 cases/2,783 controls), and 0.94 (95% CI = 0.73-1.22, 159 cases/702 controls) in low, medium, and high PCE exposure groups, respectively; the p-level for dose-response trend was 0.10 (Hadkhale et al. 2017). These results show a slight significant increase in risk of bladder cancer in the medium PCE exposure category, but no increase in the high-exposure group and no significant dose-related trend, suggesting a cause other than PCE exposure for the slight association observed in the medium-exposure group.

Results from other newer studies were not informative due to small numbers of bladder cancer cases with exposure to PCE (Bove et al. 2014a, b; Christensen et al. 2013).

F.1.2 NHL

(U.S. EPA 2012c) concluded that results from studies of NHL available at that time indicated an elevated risk for PCE. The results from five cohort studies that used a relatively high-quality exposure assessment methodology generally reported relative risks between 1.7 and 3.8 (U.S. EPA 2012c). There is some evidence of exposure-response gradients, with higher NHL risks observed in the highest exposure categories, in studies with PCE-specific exposure measures based on intensity, duration, or cumulative exposure. Effect estimates in studies with broader exposure assessments showed a more variable pattern. Confounding by life-style factors is an unlikely explanation for the observed results because common behaviors, such as smoking and alcohol use, are not strong risk factors for NHL (U.S. EPA 2012c).

Newer studies provide some support for an association between NHL and PCE exposure. In the cohort of aircraft manufacturing workers initially studied by (Boice et al. 1999) and updated by (Lipworth et al. 2011), there was a marginally significant increase in risk of death due to NHL among workers with routine or intermittent exposure to PCE (SMR = 1.43, 95% CI = 1.00-1.98) based on 36 observed cases and 25.1 expected. An internal analysis based on duration of exposure (<1, 1-4, ≥5 years) to PCE, however, did not support an association with NHL; relative risks were 1.26 (95% CI = 0.65-2.45, 11 observed), 1.00 (95% CI = 0.05-2.00, 10 observed), and 1.02 (95% CI = 0.53-1.99, 12 observed) in the low- to high-duration exposure groups compared with unexposed factory workers (P_{trend} >0.2). In the New York state cohort studied by (Silver et al. 2014), there was a nonsignificant increase in NHL risk (HR = 1.25, 95% CI = 0.90-1.73) associated with cumulative exposure to PCE relative to internal referents that is noteworthy because hourly male workers from the cohort as a whole showed a significant increase in mortality due to NHL (SMR = 1.49, 95% CI = 1.15-1.89, 65 observed) and all of the other chemical exposures assessed (trichloroethylene, methylene chloride, chlorinated hydrocarbons, and other hydrocarbons) showed nonsignificant decreases in NHL risk with increasing cumulative exposure in the internal analysis. A large case-control study of incident NHL cases extracted from the NOCCA cohort found no association with cumulative PCE exposure in men, women, or both sexes combined when analyzed by tertiles, but did find a significant or near significant risk increase in men (but not women) with high (90^{th} percentile) PCE exposure (HR = 1.54, 95% CI = 0.99-2.42 based on 25 cases using a cumulative exposure metric; HR = 1.74, 95% CI = 1.15-2.64 based on 30 cases using a metric of average intensity × prevalence) (Vlaanderen et al. 2013). A study of Marine and Navy personnel exposed to contaminated drinking water at Camp Lejeune, North Carolina between 1975 and 1985 found no association between NHL deaths (1979-2008) and exposure to PCE, as estimated by water system modeling and housing records, but is preliminary because fewer than 6% of the cohort had died by the end of the study (Bove et al. 2014a, b). Results from other newer studies were not informative, primarily due to small numbers of NHL cases with exposure to PCE (Bulka et al. 2016; Christensen et al. 2013; Morales-Suárez-Varela et al. 2013; Ruckart et al. 2013).

F.1.3 MM

(U.S. EPA 2012c) concluded that results from studies of MM available at that time indicated an elevated risk for PCE, although this was based on a smaller set of studies than available for NHL. The larger cohort studies that used a relatively nonspecific exposure measure (broad occupational title of launderers and dry cleaners, based on census data) did not report an increased risk of MM, with effect estimates ranging from 0.99 to 1.07. Some uncertainty in these estimates arises from these studies' broader exposure assessment methodology. (U.S. EPA 2012c) cited a set of results from cohort and case-control

studies as providing evidence of an association between PCE exposure and MM. The strongest evidence of association was from a case-control study that reported a nonsignificant increase in risk of MM among those ever exposed to PCE (OR = 1.5, 95% CI = 0.8-2.9) based on 16 cases, with a significantly increasing trend for risk with cumulative PCE exposure (P_{trend} = 0.02) and a significant increase in risk in the highest exposure quartile (OR = 3.3, 95% CI = 1.2-9.5) based on 10 cases. A second case-control study had too few MM cases with PCE exposure (n=3) to perform a meaningful analysis (U.S. EPA 2012c).

Among the newer studies, the large case-control study by (Vlaanderen et al. 2013) derived from the NOCCA cohort found no association of MM with cumulative PCE exposure in men, women, or both sexes combined when analyzed by tertiles; slight nonsignificant risk increases were seen in women with high (90th percentile) PCE exposure (HR = 1.14, 95% CI = 0.84-1.54 based on 52 cases using a cumulative exposure metric; HR = 1.28, 95% CI = 0.92-1.78 based on 44 cases using a metric of average intensity × prevalence). Results in men were based on smaller numbers of cases and were less stable, with high exposure based on the cumulative metric giving a HR of 1.22 (95% CI = 0.65-2.30, 12 cases) and high exposure based on average intensity × prevalence giving a HR of 0.85 (95% CI = 0.42-1.72, 9 cases). The newer cohort studies provided no support for an association between MM and PCE exposure. (Lipworth et al. 2011) reported an SMR of 1.07 (95% CI = 0.58-1.79) for MM in aircraft manufacturing workers with routine or intermittent exposure to PCE based on 14 observed and 13.2 expected cases, and no relation to duration of exposure among observed cases (RR = 0.87, 1.14, and 0.34 in low-, medium-, and high-exposure duration groups). Studies by (Silver et al. 2014), (Bove et al. 2014a), and (Bove et al. 2014b) were also negative for an association between PCE exposure and MM.

F.1.4 Esophagus

(U.S. EPA 2012c) concluded there was limited suggestive evidence for an association between esophageal cancer and PCE exposure, based on studies available at that time. The SIR in a large cohort study (n=95 cases) using broad exposure categories was 1.18 (95% CI = 0.96-1.46). The point estimates of the association in seven of eight smaller studies, four studies with specific exposure assessments, and four other studies with less precise assessments were between 1.16 and 2.44 (U.S. EPA 2012c). Two small case-control studies with relatively high-quality exposure assessment approaches reported ORs of 0.76 (95% CI = 0.34-1.69) based on 8 exposed cases and 6.4 (95% CI = 0.6-68.9) based on 2 exposed cases, respectively. Some uncertainties in these estimates arise from the lack of job title information for 25% of the cases and 19% of the controls in one study and the small number of exposed cases in the other study. One study examining exposure-response suggested a positive relationship, with SMRs of 2.16 (95% CI = 0.85-4.54, 5 cases) and 4.78 (95% CI = 2.68-7.91, 11 cases) for durations of <5 years and ≥5 years, respectively (U.S. EPA 2012c). In contrast, one study did not did not find a trend with exposure duration, but included only 0-3 cases per duration category, and another study found similar risks in subjects with little to no exposure (RR = 2.1, 95% CI = 0.9-4.4, 7 cases) and medium to high exposure (RR = 2.2, 95% CI = 1.2-3.5, 16 cases). None of the cohort studies can exclude possible confounding from alcohol and smoking—risk factors for squamous cell carcinoma of the esophagus, however based on smoking rates in blue-collar workers, the 2-fold estimated increase in relative risk reported in another set of studies (RR = 2.44, 95% CI = 1.40-3.97, RR = 2.2, 95% CI = 1.5-3.3) were higher than levels which could reasonably be attributed solely to smoking.

Findings in newer studies were generally unsupportive of an association between esophageal cancer and PCE exposure. In an update of the (Boice et al. 1999) study, (Lipworth et al. 2011) reported an SMR of 1.13 (95% CI = 0.72-1.68) for esophageal cancer among aircraft manufacturing workers with routine or intermittent exposure to PCE (24 cases versus 21.3 expected). In the internal analysis from this study

based on duration of exposure, relative risk for esophageal cancer was significantly increased in workers with less than 1 year of exposure (RR = 2.30, 95% CI = 1.14-4.66, 11 cases), but decreased with increasing exposure duration (in the high-duration group with exposure of 5 years or more, RR = 0.66, 95% CI = 0.22-1.96, 4 cases). Similarly, (Bove et al. 2014a) and (Bove et al. 2014b) reported decreasing HRs of 1.27 (95% CI = 0.57-2.81, 11 cases), 0.55 (95% CI = 0.20-1.55, 5 cases), and 0.41 (95% CI = 0.13-1.26, 4 cases) for esophageal cancer in low, medium, and high cumulative PCE exposure groups, respectively, in the Camp Lejeune cohort exposed by drinking water. The only other newer study that evaluated this endpoint was not informative due to lack of observed cases with PCE exposure (Christensen et al. 2013).

F.1.5 Kidney

(U.S. EPA 2012c) acknowledged mixed results in studies of kidney cancer available at that time, concluding that overall the evidence was suggestive but limited. One primary study supporting an association between PCE exposure and kidney cancer, a large international case-control study (245 exposed cases from Australia, Denmark, Germany, Sweden, and the United States), reported a relative risk of 1.4 (95% CI = 1.1-1.7) for any exposure to dry cleaning solvents. This study was able to adjust for smoking history, body mass index, and other risk factors for kidney cancer. Results from the large cohort studies, using a more general exposure classification based on national census occupation data, presented more variable results, with relative risks of 0.94, 1.11, and 1.15 (U.S. EPA 2012c). The results from the smaller studies using a relatively specific exposure assessment approach to refine classification of potential PCE exposure in dry cleaning settings were mixed, with some studies reporting little or no evidence of an association and other studies reporting elevated risks (U.S. EPA 2012c). An increasing trend in relative risk with increasing exposure surrogate was not observed in any of the larger occupational exposure studies with three or more exposure categories but some indication of higher risk with higher exposure (or duration) was observed in other studies (U.S. EPA 2012c).

Mixed results were obtained in newer studies as well. A case-control study of kidney cancer cases from Detroit, Michigan and Chicago, Illinois using detailed exposure assessment methodology found no significant association with probability of exposure to PCE, or with PCE exposure duration, average weekly exposure or cumulative exposure for those with ≥50% probability of exposure, but did observe a significant increase in kidney cancer risk for those in the highest tertile of cumulative hours exposed when the analysis was restricted to those with high-intensity exposure to PCE (OR = 3.1, 95% CI = 1.3-7.4, 14 cases/8 controls, P_{trend} = 0.03) (Purdue et al. 2017). This relationship was also seen in additional analyses that incorporated 5-year (OR = 3.5, 95% CI = 1.3-10.0, P_{trend} = 0.03) or 15-year (OR = 6.2, 95% CI = 1.8-21.3, P_{trend} = 0.003) exposure lag periods, included only jobs assigned an exposure probability with high confidence (OR = 5.1, 95% CI = 1.5-7.2, P_{trend} = 0.12), or excluded participants with ≥50% probability of exposure to trichloroethylene (OR = 3.0, 95% CI = 0.99-9.0, 17 cases/ 14 controls, P_{trend} = 0.08), a potential confounder. Results in other newer studies were negative. The large case-control study by (Vlaanderen et al. 2013) derived from the NOCCA cohort found no association of kidney cancer with cumulative PCE exposure in men, women, or both sexes combined when analyzed by tertiles or when the analysis was restricted to those with high (90^{th} percentile) exposure (HR = 0.81, 95% CI = 0.65-1.01 based on 88 cases using a cumulative exposure metric; HR = 1.01, 95% CI = 0.82-1.25 based on 103 cases using a metric of average intensity × prevalence). In cohort studies, (Lipworth et al. 2011) found no association between kidney cancer mortality and routine or intermittent exposure to PCE in aircraft manufacturing workers (SMR = 0.80, 95% CI = 0.43-1.37, 13 cases versus 16.3 expected) and no relation to exposure duration among the observed cases, and (Silver et al. 2014) found no association between kidney cancer and cumulative PCE exposure among electronics workers (HR = 0.15, 95% CI = 0.01-4.04). (Bove et al. 2014a) and (Bove et al. 2014b)

reported nonsignificant elevations in HR for kidney cancer that were, however, unrelated to cumulative PCE exposure in the Camp Lejeune cohort (HR = 1.40, 95% CI = 0.54-3.58, 8 cases; 1.82, 95% CI = 0.75-4.42, 11 cases; and 1.59, 95% CI = 0.66-3.86, 11 cases in low, medium, and high groups, respectively). The only other newer study that evaluated this endpoint was not informative due to few observed cases with PCE exposure (Christensen et al. 2013).

A meta-analysis of five selected epidemiologic studies (Purdue et al. 2017; Silver et al. 2014; Vlaanderen et al. 2013; Dosemeci et al. 1999; Aschengrau et al. 1993) considered to be reliable and informative for the association of kidney cancer and exposure to PCE was performed as part of the current assessment. Applying a fixed-effects model to the five informative studies produced a meta-RR of 0.96 (95% CI = 0.85-1.07) for overall exposure to PCE, with no heterogeneity among studies (I^2=0.0%, p=0.72). Estimates of the association of kidney cancer with high exposure to PCE were available for two studies (Purdue et al. 2017; Vlaanderen et al. 2013). A fixed-effects model based on the association of kidney cancer with high exposure in those two studies and with any exposure in the remaining studies produced a meta-RR of 1.07 (95% CI = 0.89-1.28) with moderate heterogeneity (I^2=45.9%, p=0.12). These results are consistent with no association or weak positive association between the occurrence of kidney cancer and exposure to PCE, but should be interpreted with caution due to the small number of informative studies.

F.1.6 Lung

(U.S. EPA 2012c) concluded there was limited suggestive evidence for an association between lung cancer risk and PCE exposure. The results from seven large cohort studies of dry cleaners available at that time were consistent with an elevated lung cancer risk of 10−40%. Similar results were observed in four of the five occupational studies that were identified as having a relatively strong exposure assessment methodology, with slightly higher relative risks identified for laundry workers compared with dry cleaning workers in a separate comparison. These studies were unable to control for potential confounding from cigarette smoking, however, and the magnitude of the association in these studies is consistent with that expected assuming the prevalence of smoking among dry cleaners and laundry workers was slightly higher (e.g., 10% higher) than among the general population. Features of the selection of study participants and study analysis in the available case-control studies reduce the potential for confounding by smoking. Two case-control studies were limited to either nonsmokers or ex-smokers and both of these studies indicate an approximate 2-fold increased risk with a history of work in the dry cleaning industry (OR = 1.8, 95% CI = 1.1-3.0; OR = 1.83, 95% CI = 0.98-3.40 among women). The other case-control studies adjusted for smoking history, and the results for these (somewhat smaller studies) are similar to the previously cited estimates. Among the studies that evaluated exposure-response gradients, the evidence for a trend in risk estimates was mixed (U.S. EPA 2012c).

Newer case-control studies of lung cancer support a relationship with PCE exposure. A study of lung cancer cases from Montreal that included adjustment for smoking (Comprehensive Smoking Index) reported ORs of 2.5 (95% CI = 1.2-5.6, 23 cases) for “any” exposure to PCE and 2.4 (95% CI = 0.8-7.7, 10 cases) for “substantial” exposure (Vizcaya et al. 2013). A larger study from France that also included adjustment for smoking (Comprehensive Smoking Index) reported ORs of 1.26 (95% CI = 0.87-1.82, 107 cases) in men and 2.74 (95% CI = 1.23-6.09, 26 cases) in women ever exposed to PCE (Mattei et al. 2014). In additional analyses by cumulative PCE exposure (split into high and low groups based on median cumulative exposure), ORs for men were 1.14 (95% CI = 0.67-1.94, 45 cases) in the low-dose group and 1.36 (95% CI = 0.84-2.22, 62 cases) in the high-dose group, while ORs for women were 3.80 (95% CI = 1.41-10.24, 21 cases) in the low-dose group and 1.43 (95% CI = 0.37-5.50, 5 cases) in the

high-dose group. Further analyses stratified by overlapping exposure to multiple solvents suggested that the observed increase in lung cancer risk was due to PCE, and not co-exposure to other chlorinated solvents (trichloroethylene, methylene chloride, chloroform, carbon tetrachloride). Newer cohort studies that investigated lung cancer risk were negative. (Lipworth et al. 2011) found no association between lung cancer mortality and routine or intermittent exposure to PCE in aircraft manufacturing workers (SMR = 0.94, 95% CI = 0.81-1.07, 206 cases versus 220.3 expected) and no relation to exposure duration among the observed cases. (Bove et al. 2014a) and (Bove et al. 2014b) reported nonsignificant elevations in HR for lung cancer that were, however, unrelated to cumulative PCE exposure in the Camp Lejeune drinking water cohort (HR = 1.33, 95% CI = 0.93-1.90, 56 cases; 1.27, 95% CI = 0.88-1.83, 55 cases; and 1.08, 95% CI = 0.75-1.57, 51 cases in low, medium, and high groups, respectively).

F.1.7 Liver

(U.S. EPA 2012c) cited results available at that time showing a mixed pattern of results for liver cancer, concluding that there was suggestive but limited evidence of an association. One case-control study with a large number of exposed liver cancer cases and a relatively high-quality exposure assessment methodology reported an OR estimate of 0.76 (95% CI = 0.38-1.72) for liver cancer and dry cleaning. Cohort studies of Nordic subjects with broad exposure assessment approaches reported SIRs of 1.02 (95% CI = 0.84-1.24), 1.22 (95% CI = 1.03-1.45), and 1.23 (95% CI = 1.02-1.49) for liver and biliary tract cancer and work as a dry cleaner or laundry worker. Three other studies with strong exposure assessment approaches specific to PCE, but whose risk estimates are based on fewer observed liver cancer cases or deaths, reported risk estimates of 1.21-2.05 for the association between liver cancer and PCE. However, dry cleaning workers did not have a higher liver cancer risk estimate than laundry workers. Exposure response was not observed, and the SIR for PCE-exposed subjects with the longest employment duration was lower than that for subjects with shorter employment duration. Potential confounding may be an alternative explanation, as no study adjusted for known and suspected risk factors for liver cancer (U.S. EPA 2012c). Nine other cohort and case-control studies with fewer observed events and/or a broad exposure assessment methodology carried less weight in the analysis and reported a mixed pattern of results (U.S. EPA 2012c). One of these reported a risk estimate of 2.57 (95% CI = 1.21-5.46) for the association between liver cancer and residence in a village with groundwater contamination, but subjects were from a region with a high prevalence of hepatitis C infection, and hepatitis C status may confound the observed association.

Among the newer studies, the large case-control study by (Vlaanderen et al. 2013) derived from the NOCCA cohort reported slight nonsignificant increases in liver cancer risk in the second (HR = 1.18, 95% CI = 0.97-1.44, 121 cases) and third (HR = 1.13, 95% CI = 0.92-1.38, 114 cases) tertiles, respectively, of cumulative PCE exposure (both sexes combined), and in those with high (90^{th} percentile) PCE exposure (HR = 1.11, 95% CI = 0.79-1.57 based on 40 cases using a cumulative exposure metric; HR = 1.26, 95% CI = 0.88-1.80 based on 38 cases using a metric of average intensity × prevalence). (Lipworth et al. 2011) found no association between liver cancer mortality and routine or intermittent exposure to PCE in aircraft manufacturing workers (SMR = 0.93, 95% CI = 0.56-1.45, 19 cases versus 20.5 expected). There was no significant relationship with exposure duration among the observed cases (P_{trend} >0.20) in this study, but relative risk was highest in workers with the longest (≥5 years) duration of exposure (RR = 1.29, 95% CI = 0.60-2.78, 10 cases). (Silver et al. 2014) found no association between liver cancer and cumulative PCE exposure among electronics workers (HR = 0.79, 95% CI = 0.27-2.30). (Bove et al. 2014a) and (Bove et al. 2014b) reported decreasing HRs of 1.17 (95% CI = 0.55-2.49, 12 cases), 0.96 (95% CI = 0.43-2.14, 10 cases), and 0.82 (95% CI = 0.36-1.89, 9 cases) for liver cancer in low, medium, and high cumulative PCE exposure groups, respectively, in the Camp Lejeune cohort exposed by drinking water. The only other newer study that evaluated this endpoint was

not informative because there was only a single observed case with PCE exposure (Christensen et al. 2013).

F.1.8 Cervix

(U.S. EPA 2012c) included cervical cancer among the tumor types with limited suggestive evidence for an association with PCE exposure. The results from two large cohort studies with a broad exposure assessment were consistent with an elevated cervical cancer risk of 20-30%: SIR = 1.20 (95% CI = 1.08-1.34) and SIR = 1.34 (95% CI = 1.12-1.60). Results from four smaller cohort and case-control studies with a relatively high-quality exposure assessment methodology presented a pattern of more variable results, with relative risks of 0.98 (95% CI = 0.65-1.47), 1.19 (95% CI = 0.64-1.93), 2.10 (95% CI = 0.68-4.90), and 3.20 (95% CI = 0.39-11.6). A fourth study with higher quality exposure assessment specific to PCE did not observe any cervical cancer deaths among women, but less than one death was expected. Only a single study reported an increasing exposure response gradient with employment duration. Dry cleaning workers did not have higher cervical cancer risks compared with laundry workers. None of the cohort studies of cervical cancer considered socioeconomic or lifestyle factors such as smoking or exposure to the human papilloma virus (HPV), a known risk factor for cervical cancer that is correlated with socioeconomic status. A case-control study included controls similar in socioeconomic status as cases, and the OR estimate in that study for dry cleaners did not support an association with PCE (U.S. EPA 2012c). The only newer study that evaluated this endpoint ((Lipworth et al. 2011), update of (Boice et al. 1999)) was not informative because there was only a single observed case with PCE exposure.

F.1.9 Breast

Breast cancer was among the endpoints considered by (U.S. EPA 2012c) to have suggestive but limited evidence of an association with PCE exposure based on studies available at that time. Results from the large studies of breast cancer risk in women in relation to PCE exposure were mixed. The largest study, based on 1,757 breast cancer cases in female dry cleaners and laundry workers, reported a statistically significant deficit in the risk of breast cancer incidence compared to the populations of Nordic countries. Findings in the other four studies were based on fewer events or exposed cases; two of four studies with a nonspecific exposure assessment methodology provided evidence for association between breast cancer in females and PCE exposure, but no association to PCE was observed in two other large cohort studies with a relatively high-quality exposure assessment methodology (U.S. EPA 2012c). Small studies also observed mixed findings. Although cohort studies were unable to control for potential confounding from reproductive history or menopausal status, observations in case-control studies controlled for these potential confounders in statistical analyses and provided support for an association between female breast cancer and PCE compared to controls. Three studies examined exposure-response relationships (U.S. EPA 2012c), and two of these studies with semiquantitative or quantitative exposure assessment approaches reported that risk estimates in females were monotonically increased in higher exposure groups. A third study examining exposure duration observed an inverse relation, but exposure duration is more uncertain than use of a semiquantitative surrogate given increased potential for bias associated with exposure misclassification.

Few data on breast cancer were found in newer studies. (Gallagher et al. 2011) conducted a case-control study that included an updated exposure assessment and reanalysis of breast cancer data previously evaluated by (Aschengrau et al. 2003), (Aschengrau et al. 1998), and (Paulu et al. 1999). They found no increase in breast cancer risk for women “ever” exposed to PCE versus unexposed, but modest nonsignificant risk increases in women with high cumulative exposure defined as 90th percentile (ORs mostly 1.3-1.5 depending on latency) or as a higher cut point identified by curve smoothing analysis (ORs 1.3-1.4 with 0-7-year latency and 1.6-2.0 with 9-15-year latency). In the (Lipworth et al. 2011)

update of the (Boice et al. 1999) cohort of aircraft manufacturing workers, there was also a nonsignificant increase in breast cancer risk (SMR = 1.52, 95% CI = 0.78-2.65) based on only 12 cases (versus 7.9 expected), but no significant trend based on exposure duration (P_{trend} >0.20) in an analysis limited by the small number of cases per exposure duration category. The only other newer study that evaluated this endpoint was not informative due to few observed cases with PCE exposure (Bove et al. 2014a, b).

Because of the limitation in statistical power, none of the older (U.S. EPA 2012c) or newer (Ruckart et al. 2015) studies reporting on male breast cancer was adequate to examine PCE exposure.

F.1.10 Other

No other cancers were identified by (U.S. EPA 2012c) as having potential associations with PCE exposure. Among the newer studies, case-control studies by (Barul et al. 2017), (Carton et al. 2017) and (Christensen et al. 2013) presented results suggesting potential associations between PCE exposure and prostate cancer in men and pharyngeal/laryngeal cancers in both sexes. However, these findings were based on small numbers of cases (≤10) and so are highly uncertain. Other studies did not report supporting results. (Lipworth et al. 2011) found no increase in risk of death due to cancers of the buccal cavity and pharynx (SMR = 0.77, 95% CI = 0.41-1.32, 13 observed and 16.8 expected), larynx (SMR = 0.90, 95% CI = 0.36-1.84, 7 observed and 7.8 expected), or prostate (SMR = 0.92, 95% CI = 0.72-1.16, 71 observed and 77.1 expected) in their cohort of aircraft manufacturing workers exposed to PCE. No significant relationship between cumulative exposure to PCE and risk of prostate or oral cancers was evident in the Camp Lejeune cohort (Bove et al. 2014a, b).

F.1.11 Detailed Summary Epidemiologic Evidence on Cancer Published after the 2012 IRIS Toxicological Assessment on PCE

Lipworth et al. (2011) conducted a follow-up analysis of the aircraft manufacturing worker cohort originally evaluated by (Boice et al. 1999) and described in (U.S. EPA 2012c). The cohort consisted of 77,943 employees who had worked for at least 1 year at a Lockheed Martin manufacturing facility in California on or after January 1, 1960. The cohort included both exposed factory workers (n=45,318) and unexposed non-factory workers (n=32,625). Subjects were identified using employee work history records, personnel files, and retirement records. Deaths through December 31, 2008 (n=34,298) were determined using the California Death Statistical Master File (CDSMF), National Death Index (NDI), and Social Security Administration Death Master File (SSADMF), as well as company pension records and a commercial service specializing in death record location. Workers for whom no death records were identified were traced using Social Security Administration Service to Epidemiologic Researchers and LexisNexis records to confirm that they were alive; these methods confirmed the identification of 42,309 living workers. The vital status of the remaining 1,336 workers (1.7% of cohort) was not determined. For deaths after 1978, underlying cause of death was available in the NDI; the CDSMF provided cause of death for subjects who died in California, and death certificates were obtained for the remaining subjects (and for a small number of subjects whose records in NDI were incomplete).

Exposures were determined based on historical job descriptions, chemical usage patterns, environmental assessment reports, industrial hygiene records, interviews with long-term workers, and walk-throughs of aircraft manufacturing facilities; details of the exposure assessment were published by (Marano et al. 2000). Approximately 12.9% of factory workers (n=5,830) had some exposure to PCE. According to (Marano et al. 2000), many PCE-exposed workers also had exposure to chromate (76%), trichloroethylene (39%), mixed solvents (56%), and/or asbestos (5%). Relative exposure to each worker was assigned based on length of time in specific jobs with potential for exposure to each substance. (Marano et al. 2000) indicated that exposures were categorized as either routine or intermittent, and that

approximately 55% of the PCE-exposed workers were classified as having intermittent exposure. Thus, there may have been a wide range of cumulative exposure levels in the group exposed to PCE, which could bias the analysis toward the null. No information was available to the researchers regarding smoking, alcohol consumption, or other lifestyle factors.

For standard mortality ratio (SMR) calculations, expected numbers of deaths were obtained using age, race, calendar year, and sex-specific rates from California (for white workers) or the U.S. general population (for non-white workers, to better match the racial composition of the worker population) (Lipworth et al. 2011). For internal analyses examining the influence of exposure duration, the comparison group consisted of factory workers without exposure to solvents or chromates (n=9,520). The model included date of birth, date of hire, date of termination, sex, and race. There was no explicit consideration of latency.

There were 2,641 deaths among the workers exposed to PCE (Lipworth et al. 2011). SMRs for all causes of death and all malignant neoplasms were reduced slightly (0.93 and 0.96, respectively), consistent with a healthy worker effect. A marginally significant increase in the SMR for NHL (SMR = 1.43; 95% confidence interval [CI] = 1.00-1.98; n=36 cases) was observed. Nonsignificant increases in SMRs for cancers of the breast (SMR = 1.52, 95% CI = 0.78-2.65, n=12 cases), connective and other soft tissues (SMR = 1.58; 95% CI = 0.58-3.43; n=6 cases), ovary and other female genital (SMR = 1.28, 95% CI = 0.26-3.74; n=3 cases), and testes and other male genital (SMR = 2.18, 95% CI = 0.45-6.37; n=3 cases) were based on small numbers of cases. Other sites, including bladder, kidney, liver, lung, esophagus, and cervix and MM had SMRs below or close to 1.0 (SMR ≤1.13).

Analyses based on duration of exposure (<1, 1-4, ≥5 years) to PCE did not support an association between PCE and NHL or any other tumor type examined, including MM and cancers of the breast, kidney, liver, lung, or esophagus (Lipworth et al. 2011). For NHL, relative risks were 1.26 (95% CI = 0.65-2.45, 11 observed), 1.00 (95% CI = 0.05 2.00, 10 observed), and 1.02 (95% CI = 0.53-1.99, 12 observed) in the low- to high-duration exposure groups compared with unexposed factory workers (Ptrend >0.2). Interpretation of the duration of exposure analysis was limited for most other tumor types (all of those listed above, except lung) by small numbers of observed tumors (≤4) in one or more of the duration groups.

In another cohort study, (Silver et al. 2014) evaluated the association between PCE exposure and cancer mortality in a cohort of 34,494 microelectronics workers in New York state. The workers were engaged in business machine production and manufacture of circuit boards and substrates between 1906 and 2001. Machine production involved exposure to dust, solvents, and metals, while circuit board production involved exposure to chlorinated solvents and other industrial chemicals. Facility records indicated that use of trichloroethylene in circuit board production began in the mid-1960s, and that use of PCE increased in 1974 when substrate manufacturing began.

Members of the cohort included all direct employees who had worked at least 91 days between January 1, 1969 and December 31, 2001 and were U.S. citizens (Silver et al. 2014). The Social Security Administration, NDI, and Internal Revenue Service were used to determine vital status of cohort members through December 31, 2009. Cause of death was determined from the NDI for deaths after 1979 and from death certificates for earlier deaths and coded according to the International Classification of Diseases (ICD) revision in effect at the time of death.
Higher percentages of hourly than salaried workers were ever potentially exposed to a compound considered in the study; however, even among hourly workers, the prevalence of PCE exposure was low

(Silver et al. 2014). Among male hourly workers, 15.1% were exposed to PCE, compared with 60.5% exposed to "other hydrocarbons." Chemical exposure was estimated using work histories from electronic personnel databases, chemical use and exposure information from the company, industrial hygiene monitoring results/documents, and company environmental impact assessments, as well as interviews of former employees and results from an Agency for Toxic Substances and Disease Registry (ATSDR) study of volatile organic compound (VOC) use at the plant from 1969 to 1980. An exposure database linking chemical use with department and year was developed and used to assign each subject to an exposed/unexposed category for PCE, trichloroethylene, methylene chloride, and chlorinated hydrocarbons as a class. Cumulative exposure duration was modified by a parameter categorizing the extent of chemical use in a department and another that categorized the extent of exposure by job function.

SMRs were calculated for all cohort members, but these analyses were not chemical-specific (Silver et al. 2014). Internal analyses by chemical exposure were performed using conditional logistic regression based on full risk sets (equivalent to Cox proportional hazards analysis). In these analyses, chemical exposure of cases was compared with those of "controls": workers who began at an age younger than the cases and survived longer (these could include cases from other risk sets). Age was controlled using risk set selection, and models were adjusted for sex and pay code (as it is potentially associated with exposure, smoking, and other potential confounders). Smoking, alcohol consumption, and other lifestyle factors were not explicitly considered. The authors did not control for other chemical exposures or evaluate correlations among them. Hazard ratios (HRs) at 5 modified exposure years were reported, along with the regression coefficient, with a 10-year lag time incorporated for all outcomes apart from leukemia (for which a 2-year lag was used).

SMRs for all cause and all cancer mortality were significantly decreased in the cohort relative to U.S. general population rates, showing the expected healthy worker effect (Silver et al. 2014). Also among the cohort as a whole, the SMR for NHL was significantly increased in hourly male workers (SMR = 1.49, 95% CI = 1.15-1.89, 65 observed). In the analyses for specific chemical exposures, PCE showed a small nonsignificant increase in HR for NHL (HR = 1.25, 95% CI = 0.90-1.73), while the other exposures examined (trichloroethylene, methylene chloride, chlorinated hydrocarbons, and other hydrocarbons) showed nonsignificant decreases. PCE showed no association (HR ≤1.0) with other cancers, including bladder, kidney, liver, brain, or MM. The study was limited by the young age of the cohort (only 17% had died at the end of follow-up), as well as by the low prevalence of PCE exposure and failure to control for co-exposures.

(Gallagher et al. 2011) performed a case-control study that included a reanalysis of breast cancer data previously evaluated by (Aschengrau et al. 1998), (Aschengrau et al. 2003), and (Paulu et al. 1999) and described in (U.S. EPA 2012c), updating the exposure assessment of the Cape Cod population exposed to PCE leaching from the vinyl lining of drinking water distribution pipes. Briefly, while earlier assessments used the Webler and Brown model to estimate residential PCE exposures based on the configuration, size, age, and water flow rate in contaminated pipe serving each residence, (Gallagher et al. 2011) employed the EPANET software to provide more robust modeling of water flow throughout the entire distribution system. Participant selection was identical to earlier assessments, except that subjects from the earlier analyses were excluded if information needed for EPANET modeling was missing. Eligible persons consisted of permanent female residents of eight affected towns in Cape Cod. Incident breast cancer cases between 1983 and 1993 were identified using the state cancer registry; controls of comparable age and vital status were identified through random digit dialing (for controls up to 64 years of age), Medicare records (65 years of age and older), or death certificates (deceased

controls). Of 1,192 cases and 7,869 controls initially identified, 87 cases and 1,125 controls could not be located; 31 cases and 4,404 controls were not eligible based on residential criteria; 8 cases and 34 controls lacked exposure information; and 136 cases and 338 controls declined to participate (or their physicians declined consent). Finally, 666 eligible controls identified by random digit dialing were excluded because the target number of controls had already been reached. Of the 930 cases and 1,302 controls included in previous analyses, 19 lacked information needed for EPANET exposure modeling and were excluded, leaving 920 cases and 1,293 controls for the reanalysis.

From each subject, detailed residential history, history of occupational exposure to PCE, risk factors for breast cancer, and other demographic information was obtained via interview (Gallagher et al. 2011). Using the EPANET software to model water flow in the distribution system and leaching components from the Webler-Brown model, the study authors estimated relative delivered dose (RDD) to each residence. The RDD is a relative dose estimate intended to approximate the amount of PCE delivered to each residence. Odds ratios (ORs) were evaluated using multiple logistic regression controlling for the following variables: age at diagnosis or index year, vital status at interview, family history of breast cancer, personal history of prior breast cancer, age at first live birth or stillbirth, occupational PCE exposure, and study of origin (first study or second expanded study). Use of bottled water was considered by stratifying the results. Other potential confounders, including education, hormone use, and parity were considered, but did not modify effect estimates by at least 10% and were excluded from the final model. ORs were calculated with and without latency periods of 5-19 years, based on ever/never exposed, cumulative RDD, peak RDD, and duration of exposure to PCE. The impact of PCE leaching rate was evaluated by sensitivity analysis, and smoothing analysis was used to refine the cut points for high exposure.

The updated exposure assessment using the EPANET software categorized larger percentages of cases and controls as exposed (48.8% and 50.1%, respectively) compared to the earlier method (20.5% and 16.7%, respectively), which had assumed that residences not in close proximity to a source pipe were not exposed (Gallagher et al. 2011). Because most of the participants whose status shifted from non-exposed to exposed were exposed at low levels, the EPANET method yielded a downward shift in RDD distribution percentiles compared to the earlier method; for example, 75th and 90th percentile RDD estimates (unitless) with no latency period were 7.1 and 19.5, compared with 15.5 and 41.8 (respectively) using the earlier method.

Using the updated exposure estimates, no increases in the adjusted ORs for breast cancer were observed for women “ever” versus never exposed, regardless of latency period considered (adjusted OR = 1.0 for all latencies) (Gallagher et al. 2011). Compared to unexposed subjects, modest nonsignificant increases in the adjusted ORs were observed for cumulative RDDs above the 90th percentile (adjusted ORs mostly 1.3-1.5 depending on latency) and for peak RDD above the 90th percentile (adjusted ORs 0.9-1.5), but not the lower exposure levels. Analysis for duration of exposure showed a nonsignificant increase in breast cancer risk in women with more than 10 years of exposure when a 13-year latency period was included (adjusted OR = 1.8, 95% CI = 0.7-4.4); none of the women had more than 10 years of exposure when longer latency periods were considered. No associations were found between shorter durations of exposure and breast cancer, regardless of latency period. When the cut points for higher cumulative exposure were redefined based on smoothing analysis (RDD >35), adjusted ORs (none significant) were 1.3-1.4 with 0-7-year latency and 1.6-2.0 with 9-15-year latency. Results were reported to be similar for peak exposure, but data were not shown. Finally, slightly higher risks were seen for exposed women who did not drink bottled water regularly (adjusted ORs = 1.1 1.3 across latency periods) when compared with those who did (adjusted ORs = 0.6-0.8). As in the previous studies

conducted on these data, this study suggests a modest association between high drinking water exposure to PCE and breast cancer risk in women.

(Ruckart et al. 2013) conducted a case-control study of childhood hemopoietic cancers (leukemia and NHL) in children exposed prenatally and in early childhood to contaminated drinking water at the Marine Corps Base at Camp Lejeune, North Carolina. Contaminated water at the camp, which opened in the 1940s, was discovered in the early 1980s in wells of the Camp's Hadnot Point and Tarawa Terrace distribution systems. The Tarawa Terrace system was primarily contaminated with PCE (up to 215 µg/L) from a nearby dry cleaner, while Hadnot Point was primarily contaminated with trichloroethylene (up to 1,400 µg/L), with lesser amounts of vinyl chloride, 1,2-dichlorethylene, PCE, and benzene. These authors did not detail other contaminants in the Tarawa Terrace system; however, (Ruckart et al. 2015) estimated that low levels (≤20 µg/L) of dichloroethylene, trichloroethylene, and vinyl chloride were present along with PCE.

The study population consisted of children born alive between 1968, when North Carolina began computerizing birth certificates, and 1985, when the contaminated wells were closed, and whose mothers had lived at Camp Lejeune during pregnancy (Ruckart et al. 2013). A total of 12,493 children whose mothers lived on base when they delivered were identified by birth certificates, and an additional 4,000 children whose mothers had moved off base prior to delivery were identified via media campaigns and referrals from enrolled subjects. Telephone interviews of parents were conducted by ATSDR to obtain information on childhood (before age 20) leukemia and NHL and residential histories. Of 12,498 subjects whose parents were contacted, 76% agreed to participate, including 10,044 identified by birth certificates and 2,554 identified by referral.

Exposures to contaminated water were estimated by ATSDR via base-wide models of groundwater fate and transport and drinking water distribution systems, which yielded monthly average concentration estimates at each residence (Ruckart et al. 2013). Base housing records and parental interview information were combined with the concentrations to estimate average exposure to each subject across pregnancy and the first year of life. The study authors did not isolate subjects by water distribution system, so the study population included those using the Hadnot Point system with exposure primarily to trichloroethylene. Exposures were estimated for each trimester, for the whole gestation period, and for the first year of life.

A total of 14 childhood hematopoietic cancers were reported by parents (Ruckart et al. 2013). Of these, 13 cases were confirmed via vital and medical records, including 11 leukemias and 2 NHL. The parents of 651 potential control subjects were contacted; 103 refused or could not be contacted, so 548 were interviewed. Subsequently, 14 control children were excluded because their parents reported in the interview that the mother had not resided on the base during pregnancy; 6 were excluded because the parents were interviewed about the wrong child; and two lacked residential history during pregnancy, leaving 526 controls. ORs were estimated using unconditional logistic regression. Potential confounders considered in the analysis were not reported, and adjusted results were only reported if the difference from the crude estimates was more than 20%.

The median estimated average PCE exposure of subjects was 44 µg/L (Ruckart et al. 2013). Using the average first trimester exposure estimate, the unadjusted OR for exposed versus unexposed was 1.6 (95% CI = 0.5-4.8) based on 7 cases (total for childhood leukemia and NHL combined), and the unadjusted ORs for exposure above and below the median, compared with unexposed, were similar and also imprecise (OR = 1.4, 95% CI 0.3-5.6 for exposure ≥44 µg/L based on 3 cases; OR=1.8, 95% CI =

0.5-6.6 for exposure >0 and <44 µg/L based on 4 cases). Other metrics for first trimester exposure (maximum, unexposed including exposure <1 µg/L) yielded comparable effect estimates (data not reported), while no association with childhood leukemia and NHL was seen using cumulative exposure to PCE through pregnancy or the first year of life (data not reported). These data are highly uncertain due to the small number of observed cases exposed to PCE.

(Ruckart et al. 2015) assessed male breast cancer risk in a case-control study of U.S. Marine Corps personnel stationed at Camp Lejeune. Cases and controls were identified using the Veteran's Affairs Central Cancer Registry (VACCR). The study population was defined as male Marines diagnosed or treated for cancers between January 1, 1995 (when the VACCR began) and May 5, 2013 at a medical facility run by the Veterans Administration (VA). Those who were not old enough to have been at Camp Lejeune during the time of water contamination (e.g., at least 17 years old by December 31, 1985) were excluded. A total of 78 incident cases of male breast cancer were identified. Controls were diagnosed with cancers not known to be related to solvent exposure, including non-melanoma skin cancer, bone cancer, and pleural or peritoneal mesothelioma. To achieve the targeted 5 controls per case, the study authors included all 32 bone cancer cases, all 76 mesothelioma cases, and a random sample of 292 skin cancers from among the 555 identified in VACCR, yielding a total of 400 controls.

All information was obtained from databases; no subject interviews were conducted (Ruckart et al. 2015). Military personnel records were used to determine whether and when subjects had been stationed at Camp Lejeune before 1986, as well as their marital status at each time period stationed there; these records were missing for 7 cases and 27 controls. The VACCR and VA patient treatment files were examined for information on tumor histological confirmation, date of birth, age at diagnosis, race, and medical conditions (e.g., diabetes, obesity, gynecomastia, and Klinefelter syndrome) potentially related to male breast cancer development. Finally, information on service in Vietnam (with potential exposure to dioxin via Agent Orange) and military occupational specialties with potential exposure to solvents and electromagnetic fields was obtained from military personnel records.

The same historical reconstruction method used by (Ruckart et al. 2013) was used to estimate monthly average exposure concentrations at each residence (Ruckart et al. 2015). The residential histories of cases and controls were developed from base housing records, military personnel records, and unit-specific housing records. Exposure began with the earliest time each subject was stationed at Lejeune and ended either when his tour ended or on December 31, 1985. Cumulative and average exposures were estimated for each subject; exposure-response analysis was performed by categorizing exposures above and below the median. The study authors employed exact logistic and conditional regression methods to estimate associations, but since results were similar, only the exact logistic method results were presented. Results were adjusted for age at diagnosis, race, and service in Vietnam; other potential covariates (case/control status, ethnicity, rank, diabetes, or gynecomastia) did not alter risk estimates by at least 10%. Finally, proportional hazards analysis, adjusted for race and service in Vietnam, was used to assess whether PCE exposure resulted in earlier age at breast cancer diagnosis. While latency was not explicitly included in the assessment, the authors noted that an implicit latency of at least 10 years was considered, because exposures ended in 1985, and cases were diagnosed after 1995 (when the VACCR commenced operation).

The final analysis included 71 cases and 373 controls, but only 4 cases exposed to PCE (Ruckart et al. 2015). For cumulative PCE exposure, the adjusted ORs for low (>0 and <36 µg/L-months) and high (≥36 µg/L-months) exposure were 1.05 (95% CI = 0.14-5.14) and 1.20 (95% CI = 0.16-5.89), respectively. For monthly average exposure, the adjusted ORs for low (>0 and <2 µg/L) and high (≥2

µg/L) exposure were 0.91 (95% CI = 0.13-4.21) and 1.47 (95% CI = 0.18-7.91). In the evaluation for reduced age at diagnosis, the adjusted HRs were 1.19 (95% CI = 0.2-7.07) for low and 2.08 (95% CI = 0.31 14.00) for high cumulative exposures. All of these results are highly uncertain, as they are based on only 2 cases per exposure group.

A retrospective cohort study of military personnel at Camp Lejeune was conducted by (Bove et al. 2014a) and (Bove et al. 2014b). A primary focus of the study was standardized mortality analysis of personnel stationed at Camp Lejeune (with exposure to drinking water contaminated with PCE, trichloroethylene, and other solvents) and analyses comparing personnel at Camp Lejeune with those stationed at Camp Pendleton (without exposure to contaminated water); these analyses are not discussed here, because they do not provide hazard identification information specific to PCE. The study authors also conducted an internal analysis of Camp Lejeune with chemical-specific effect estimates, as described here.

The study population was defined as all Marine and Navy personnel who were stationed for active duty at Camp Lejeune between April 1975 and December 1985 (Bove et al. 2014a, b). A total of 154,932 subjects were identified using personnel files that included date of birth, sex, race/ethnicity, marital status, rank, active duty start date, total months of service, and military occupation. Vital status was determined using Social Security Administration data and a commercial tracing service, and deaths and causes (underlying and contributing) were identified using the NDI. Subjects whose vital status could not be determined contributed person-years until the last date known to be alive.

Exposure assessment employed the same historical reconstruction methods used by (Ruckart et al. 2015) and (Ruckart et al. 2013). Residential histories were determined using base housing records together with rank, gender, marital status, and dates of service. For each subject, monthly average exposure concentrations at each residence were combined with duration at each residence to estimate cumulative exposure. Exposure estimates for PCE exhibited correlations (0.44-0.53) with other contaminants; the authors noted that the Tarawa Terrace system, with the highest PCE levels (up to 158 µg/L, with mean monthly average estimate of 75.7 µg/L), had low levels of other contaminants (e.g., mean estimated monthly averages of 3.1 µg/L trichlorethylene and 5.6 µg/L vinyl chloride). The other contaminated system at the Camp, Hadnot Point, was primarily contaminated with trichloroethylene (mean monthly average estimate of 358.7 µg/L; means for PCE, vinyl chloride, and benzene were 15.7, 24.0, and 5.4 µg/L, respectively).

The study authors analyzed the association between cancer mortality and PCE exposure as HRs using Cox extended regression models with age as the time variable and cumulative exposure as a time-varying variable (Bove et al. 2014a, b). Lag periods of 0, 10, 15, and 20 years were considered in assessments of cumulative exposures. Confounders were incorporated into the model if they altered the effect estimate by 10% or more; these included sex, race, rank, and education. Because the data sources used for the study lacked information on smoking, the HR for smoking-related diseases (stomach cancer, cardiovascular disease, chronic obstructive pulmonary disease [COPD]) were subtracted from the HR for the disease of interest to assess potential confounding by smoking. The validity of this method to control for confounding by smoking is uncertain. No information on alcohol consumption or non-service-related occupational exposures was available in the data sources used in the study.

The analysis based on cumulative exposure to PCE showed no significant exposure-related increase in cancer risk for any tumor type, including bladder, kidney, liver, esophagus, breast, brain, lung, MM, NHL, Hodgkin's disease, and leukemia (Bove et al. 2014a, b). Nonsignificant Increases in kidney cancer

risk were observed for all cumulative exposure levels of PCE, but risk did not increase with estimated exposure: HRs were 1.40 (95% CI = 0.54-3.58, 8 cases), 1.82 (95% CI = 0.75-4.42, 11 cases), and 1.59 (95% CI = 0.66-3.86, 11 cases) for low (>1 to 155 µg/L-month), medium (>155-380 µg/L-month), and high (>380 8,585 µg/L-month) exposures, respectively. The authors reported that similar results were observed when exposure was quantified as average exposure or duration of exposure (data not shown). Findings from this study should be considered preliminary, as fewer than 6% of the cohort had died by the end of the study, with 97% remaining under the age of 55 years.

(Christensen et al. 2013) performed a case-control study to examine the relationship between occupational solvent exposure and multiple cancer types in residents of Montreal, Canada. Among 4,576 eligible Canadian males aged 35-70 years diagnosed with any of 11 different types of cancer (bladder, NHL, liver, pancreas, kidney, esophagus, stomach, colon, rectum, prostate, melanoma) between 1979 and 1985 in the 18 largest hospitals in Montreal, 3,730 (82%) were successfully interviewed (proportion by proxy varied with tumor type from low of 11.6% for melanoma to high of 60.4% for liver cancer). Population controls, stratified by sex and age to the distribution of cases, were randomly sampled from electoral lists; 533 (72%) of 740 eligible controls were interviewed (12.6% by proxy). Interviews were conducted to obtain information on lifestyle factors and job history (company, products, nature of work site, subject's main and secondary tasks, use of protective equipment, etc.), which was translated into potential exposures to chlorinated solvents (PCE and 5 other individual chemicals, chlorinated alkanes, chlorinated alkenes) by a team of chemists and industrial hygienists, blinded to a subject's case or control status. Exposures were graded with respect to confidence that the exposure had occurred (possible, probable, definite), frequency of exposure in a normal work week (<5%, 5-30%, >30% of the time), and intensity of exposure (low, medium, or high). Exposures that were probable or definite, with frequency and intensity of medium or high and duration of 5 or more years were considered to be "substantial" for the analysis.

The authors did not discuss the extent of overlap of exposures (Christensen et al. 2013), but review of the occupations with highest prevalence of exposure for each material analyzed showed considerable overlap in occupations that is likely to have extended to exposures as well. Analyses were performed using both population and cancer controls, as well as a pooled control group with cancer controls given equal weight to population controls. Cancer controls for a given tumor type were cancer cases with other tumors that were: (1) not lung cancer, (2) not from adjacent sites in the body to the site in question, and (3) selected so that no more than 20% were from any one cancer site. All models were adjusted for age, ethnicity (French Canadian or other), socioeconomic status, and respondent (proxy or self). Models for some cancer types (not NHL) were also adjusted for smoking and consumption of alcohol, coffee, and/or tea. Models were not adjusted for co-exposures to other solvents. Most cases and controls were current or former smokers.

Numbers of cases and population controls with "substantial" or even "any" exposure to PCE were low for all tumor types, 4 or lower in most cases (Christensen et al. 2013), which limits the conclusions that can be drawn based on reported ORs for most endpoints in this study, whether above or below 1.0. However, a significant increase was found for risk of prostate cancer with "substantial" exposure to PCE relative to both population controls (OR = 6.0, 95% CI = 1.2-30 based on 9/449 cases and 2/533 controls) and cancer controls (OR = 4.3, 95% CI = 1.4-13 based on 9/1,550 controls). None of the other chemicals evaluated showed a significant association with prostate cancer, and neither did chlorinated alkenes or alkanes collectively. Confidence in the suggested association between PCE exposure and prostate cancer is low due to small numbers of cases and controls.

(Vizcaya et al. 2013) published separate and pooled analyses of lung cancer from two population-based case-control studies performed in Montreal, Quebec. Analyses of non-pulmonary cancer types in one of the case-control studies (referred to as Study I) were published by (Christensen et al. 2013); details of the case and control selection, participation rates, and exposure assessment for Study I are discussed in that study description. Study II was conducted using nearly identical procedures but from 1995 to 2001 (Study I was 1980-1986). A total of 851 male lung cancer cases and 533 male controls (79% and 70% of eligible subjects, respectively) were identified in Study I, while 735 male and 430 female lung cancer cases and 898 male and 570 female controls (86% and 70% of eligible subjects, respectively) were identified in Study II. Next-of-kin proxies responded for about one-third of cases and one-tenth of controls. ORs were calculated using unconditional logistic regressions adjusted for age, income, ethnicity, educational attainment, questionnaire respondent (self versus proxy), tobacco smoking (Comprehensive Smoking Index), exposure to occupational lung carcinogens (never, ever, or substantial occupational exposure to any of the 8 known or probable International Agency for Research on Cancer (IARC) lung carcinogens: asbestos, crystalline silica, chromium VI, arsenic compounds, diesel exhaust emissions, soot, wood dust, or benzo[a]pyrene), and in the pooled analysis, study (I versus II). The authors noted that sample sizes were limited and there was overlapping exposure to multiple solvents, and thus it was not possible to evaluate risks to subjects exposed to only one solvent.

Prevalence of exposure to any chlorinated solvent was 14.4% in male and 9.6% in female population controls across both studies (Vizcaya et al. 2013). Because there were fewer women included and their exposure prevalence was lower, the study had little power to detect an effect in women and results were presented for men only. The lifetime prevalence of PCE exposure in controls was very low (0.9% across both studies). ORs for lung cancer with PCE exposure were 4.3 (95% CI = 1.1-16) based on 11/667 cases and 4/403 controls with “any” exposure and 5.7 (95% CI = 0.9-36) based on 6/667 cases and 2/403 controls with “substantial” exposure in Study I, 2.3 (95% CI = 0.8-6.2) based on 12/646 cases and 9/822 controls with “any” exposure and 1.6 (95% CI = 0.3-8.3) based on 4/646 cases and 4/822 controls with “substantial” exposure in Study II, and 2.5 (95% CI = 1.2 5.6) based on 23/1,313 cases and 13/1,225 controls with “any” exposure and 2.4 (95% CI = 0.8-7.7) based on 10/1,313 cases and 6/1,225 controls with “substantial” exposure in the pooled analysis. Similar results were observed when the analysis was restricted to subjects who completed the questionnaires themselves (no proxy respondents). Among the other chemicals evaluated, only carbon tetrachloride showed a significant association with lung cancer, with results comparable to those for PCE among those with “substantial” exposure. There was no association with lung cancer for chlorinated alkenes or alkanes collectively. These findings suggest an association between exposure to PCE and lung cancer, but are limited by the low numbers of cases and controls with PCE exposure.

(Mattei et al. 2014) performed a large, multicenter population-based case-control study of lung cancer and solvent exposure in France. Cases were recruited from health care providers associated with French cancer registries. A total of 4,865 eligible cases (ages 18-75 years) of incident, histologically-confirmed lung cancer were identified between 2001 and 2007; of these, 3,357 living subjects were located and healthy enough to be interviewed, and 2,926 (87%) were willing to participate. Controls were selected by incidence density sampling and frequency-matched by age and gender. Investigators were able to contact 4,411 (94%) of 4,673 eligible controls and 3,555 (81%) agreed to participate. Analyses were based on 2,274 male and 622 female cases, and 2,780 male and 760 female controls. Exposure assessment employed standardized questionnaires administered by trained interviewers for collection of data regarding smoking history, sociodemographic characteristics, and lifetime occupational history (company, tasks, specific exposures). The only chlorinated solvent specifically listed in the questionnaire was trichloroethylene, although subjects could self-report other known exposures, such as

PCE. A short-form questionnaire without the detailed job information was used for proxy interviews (5% of men and 3% of women). Job histories were mapped to a job-exposure matrix to classify solvent exposures by probability, intensity, frequency, and duration. Cumulative exposure indices were calculated as the product of probability, frequency, intensity, and duration for each job, and then categorized using deciles of the distribution in the control subjects. Lag times of 0, 5, and 10 years were analyzed. Covariates considered in the analyses included age at interview, location, smoking history (Comprehensive Smoking Index), number of jobs held, occupational exposure to asbestos, and in some cases, socioeconomic status.

Among controls, prevalence of lifetime exposure to chlorinated solvents was 8.5% for men and 2.1% for women (Mattei et al. 2014). The individual solvent with the highest prevalence of exposure was trichloroethylene (7.6% of male and 1.1% of female controls). Only 0.3% of male and 0.9% of female controls had any exposure to PCE, and almost all of these were exposed to other solvents as well. Men were exposed to PCE primarily as printers, while women were exposed primarily as launderers and dry cleaners. Trichloroethylene was the only individual solvent with a significant number of study subjects that were not exposed to any other chlorinated solvents. In order to elucidate effects of other solvents (such as PCE) individually, despite the multiple overlapping chemical exposures, the researchers performed stratified analysis of mutually exclusive multiple solvent exposures (e.g., trichloroethylene alone, versus trichloroethylene plus PCE, versus trichloroethylene plus PCE and methylene chloride, etc.).

After adjustment for covariates, including socioeconomic status, the OR for PCE comparing ever exposed to never exposed was 1.26 for men (95% CI = 0.87-1.82) based on 107 lung cancer cases and 94 controls with PCE exposure and 2.74 for women (95% CI = 1.23-6.09) based on 26 cases and 13 controls (Mattei et al. 2014). In analyses by cumulative PCE exposure (split into high and low groups based on median cumulative exposure), ORs for men were 1.14 in the low-dose group (95% CI = 0.67-1.94, 45 cases and 47 controls) and 1.36 in the high-dose group (95% CI = 0.84-2.22, 62 cases and 47 controls), while ORs for women were 3.80 in the low-dose group (95% CI = 1.41-10.24, 21 cases and 7 controls) and 1.43 in the high-dose group (95% CI = 0.37-5.50, 5 cases and 6 controls). In analyses stratified by overlapping exposure to multiple solvents, ORs were elevated for women exposed to PCE with trichloroethylene (2.39, 95% CI = 0.47 12.18, 6 cases and 3 controls) and with both trichloroethylene and methylene chloride (4.57, 95% CI = 1.14-18.34, 12 cases and 3 controls), but not those exposed to trichloroethylene alone (1.16, 95% CI = 0.64-2.11, 49 cases and 32 controls) or with methylene chloride (0.73, 95% CI = 0.29-1.87, 12 cases and 17 controls) or methylene chloride and chloroform and carbon tetrachloride (1.12, 95% CI = 0.31-4.08, 6 cases and 7 controls). In men, ORs were also higher in the PCE groups (OR = 1.28-1.32) than the others (OR = 0.79-0.95), although the difference was less pronounced than in women. These findings suggest an association between lung cancer and PCE exposure, but are limited by low prevalence of PCE exposure among study subjects.

(Ruder et al. 2013) conducted a population-based case-control study focused on the association between exposure to chlorinated aliphatic solvents, including PCE, and risk of glioma. Eligible participants were residents of non-metropolitan counties in the states of Iowa, Michigan, Minnesota, and Wisconsin who were diagnosed with glioma between 1995 and 1997 (cases) or were residents of the counties on January 1, 1995 (controls). Histologically-confirmed primary intracranial glioma cases were identified from neurosurgery offices and other participating health care facilities. A pool of candidate controls was established prior to case enrollment based on the age and sex distribution of glioma cases from an earlier time period, using state driver license records (ages 18-64 years) or Medicare data tapes (ages 65 80 years). Persons diagnosed with cancers other than glioma (20.6% of controls) were eligible to

participate. Participants included 798 cases (91.5% of eligible cases) and 1,175 controls (70.4% of eligible controls). Interviews of cases (n=438), case next-of-kin (n=360), and controls (n=1,141) were performed to obtain occupational history. Standardized questionnaires were used to establish details (employer name, industry, job title, tasks, materials used, and employment frequency) of jobs held for at least 1 year between 16 years of age and 1992; the questionnaires asked explicit questions regarding exposures to solvents, thinners, glues, inks, varnishes, stains, and paint strippers. An industrial hygienist blinded to case status combined the job history information with the authors' exposure database (from published literature sources) to estimate probability, frequency, and intensity of exposure, as well as confidence in the probability and frequency of exposure. Cumulative exposures were estimated as the product of employment duration, employment frequency, exposure frequency, and exposure intensity. Analyses were adjusted for sex, age, and education. Sensitivity analyses were performed excluding cases with job history based on proxy questionnaires (to improve validity of the exposure estimates) or limiting the exposed group to those with high probability (>0.5) of exposure. Types of gliomas observed in cases included glioblastoma multiforme (equivalent to stage 4 glioma) (58%), astrocytoma (22%), oligodendroglioma (11%), and other (8%). A subset of participants agreed to provide blood samples for GST genotyping; these data were used to analyze the influence of GST on the association between glioma risk and chlorinated solvent exposure.

ORs for PCE exposure and glioma risk were <1.0 in all analyses, including: when all subjects were considered together (OR = 0.75, 95% CI = 0.62-0.91, 299 cases and 500 controls); when stratified by sex; when analyzed as "any" versus no exposure; when analyzed by cumulative exposure; when cases with proxy exposure data were excluded; and when exposed subjects were limited to those with high probability of exposure (Ruder et al. 2013). GST genotype did not influence the relationship between solvent exposure and glioma risk. Results were similarly negative for any chlorinated solvent and for the other solvents considered individually. In this study, the large proportion of case questionnaires completed by proxy (next of kin) is problematic, although excluding proxy interviews did not affect results. Potential memory impairment (induced by glioma) among cases who did complete the questionnaires may have affected exposure estimates in cases relative to controls. In addition, controls were older than cases, and thus had greater chance of higher exposure from working during earlier eras, and cases had slightly more education than controls, and therefore lower probability of solvent-related employment. These limitations would tend to bias the risk estimates toward the null.

(Neta et al. 2012) evaluated associations between solvent exposure and risk of glioma and meningioma in a hospital-based study. Cases were patients at one of four hospitals (referral centers for brain cancers in Massachusetts, Pennsylvania, and Arizona) who had received a histologically-confirmed diagnosis of primary glioma or other neuroepitheliomatous neoplasm or meningioma within the previous 8 weeks. A total of 484 cases of glioma (92% of eligible cases) and 197 cases of meningioma (94% of eligible cases) agreed to participate. Controls were patients at the same hospitals who were receiving treatment for non-cancer conditions. Controls were frequency matched on sex, age at interview, race/ethnicity, hospital, and residential proximity to the hospital. A total of 797 controls (86% of eligible subjects) agreed to participate. Trained interviewers administered questionnaires to patients (or a proxy if the patient was too ill or deceased) to document jobs in which the patients worked for at least 6 months after the age of 16 years; details included employer, dates of employment, job title, full or part time work status, type of business, tasks, and materials and equipment used. Proxy interviews were conducted for 16% (n=78) of glioma cases, 8% (n=15) of meningioma cases and 3% (n=23) of controls. When respondents indicated employment in jobs with chemical exposures, more detailed industry- or job-specific questions were asked to obtain information on frequency and duration of solvent-related tasks as well as other information pertaining to exposure (e.g., potential for dermal exposure, sensory

descriptions) or mitigation of exposure (engineering controls, personal protective equipment). Results were reviewed by expert industrial hygienists who identified incomplete or inconsistent answers; investigators followed up with supplementary subject phone interviews to resolve these discrepancies. Using the finalized job histories and exposure data from occupational health literature, industrial hygienists assigned exposure levels for six solvents including PCE. Analyses were adjusted for age at diagnosis, sex, race/ethnicity, hospital site, residential zone/proximity to hospital, and estimated cumulative occupational exposure to potential confounders: lead, magnetic fields, herbicides, and insecticides. Analyses by any/no exposure to a given solvent were also adjusted for exposure to other solvents. The investigators determined that adjustment for education and smoking did not result in changes to the effect estimates, so these covariates were not included in the final models. ORs comparing high to low exposure were also calculated (in addition to any/none) to control for potential unidentified differences between exposed and unexposed subjects. Finally, a lag time of 10 years was analyzed by excluding exposures in the 10 years prior to diagnosis.

The OR for glioma was 0.7 (95% CI = 0.5-0.9, 136 cases and 255 controls) for study subjects with "possible" exposure to PCE and 0.7 (95% CI = 0.3-1.6, 9 cases and 20 controls) for those with "probable" exposure (Neta et al. 2012). Results were similar when stratified by sex and various measures of exposure (years exposed, cumulative exposure, average weekly exposure, highest exposure). For meningioma, the ORs for "possible" and "probable" exposure were 0.9 (95% CI = 0.6-1.3, 52 cases and 255 controls) and 0.5 (95% CI = 0.1-1.7, 3 cases and 20 controls), respectively, without adjustment for exposure to other solvents and 1.0 (95% CI = 0.5-2.2) and 0.3 (95% CI = 0.1-1.7), with the adjustment. Similarly, no clear associations were seen for the other solvents analyzed or for the solvents collectively. Because relatively few subjects had exposures characterized as high, the study had limited power to evaluate dose-response relationships (e.g., only 10 controls and 3 glioma cases were classified as having high cumulative PCE exposure). The researchers noted that the complexity of use of these solvents, which have been used interchangeably and at times together, makes evaluation of specific exposures difficult. Exposure misclassification and potential memory impairment (induced by glioma) among cases would tend to bias the risk estimates toward the null.

(Carton et al. 2017) investigated the relationship between occupational solvent exposure and head and neck cancer in a case-control study in France. The final study group included 296 women with squamous cell carcinoma of the oral cavity, oropharynx, hypopharynx, or larynx and 775 controls. Incident cases were women aged 18-75 years at diagnosis between 2001 and 2007 identified from cancer registries in 10 geographic areas in France and whose cancers were histologically confirmed. Controls were chosen at random from the same geographic areas with age and sex distribution comparable to cases and distribution of socioeconomic status similar to the general population. Participation rate was 82.5% for cases and 80.6% for controls. Subjects were interviewed in person using a standardized questionnaire for detailed occupation history, residential history, and lifetime alcohol and tobacco consumption. Job-exposure matrices developed for the French population by the French Institute of Health Surveillance were used to estimate probability, intensity, and frequency of exposure to PCE and other solvents for each job held at least 1 month. The products of duration, probability, intensity, and frequency of exposure for each job were summed to give cumulative exposure, and cumulative exposure was divided by total duration of employment to calculate the mean intensity of exposure.

Controls smoked significantly less and drank alcohol significantly less than cases and were of significantly higher socioeconomic status (Carton et al. 2017). Age and geographic distributions differed significantly as well. Analyses were performed by unconditional logistic regression and adjusted for

geographical area, age, smoking status (never smoker, former smoker, and current smoker), tobacco consumption in pack-years, and alcohol consumption in drink-years. Socioeconomic status, assessed by the last occupation held and by the longest held occupation, was included in preliminary models, but removed from the final models because it did not significantly affect results.

There was a significant association between "ever" exposed to PCE and head and neck cancer (OR = 2.97, 95% CI = 1.05-8.45), based on 10 cases and 13 controls (Carton et al. 2017). Of these, however, no cases and only 3 controls were exposed to PCE alone without other chlorinated solvents. The rest were exposed to PCE in combination with trichloroethylene (OR = 4.47, 95% CI = 1.27-15.8, 9 cases and 7 controls) or with trichloroethylene and methylene chloride (OR = 2.16, 95% CI = 0.19-24.1, 1 case and 3 controls). "Ever" exposed to trichloroethylene was also significantly associated with head and neck cancer (OR = 2.15, 95% CI = 1.21-3.81) based on many more subjects (38 cases and 60 controls). For "ever" exposed to trichloroethylene alone, the OR was 1.81 (95% CI = 0.81 4.04) based on 20 cases and 32 controls. The 10 cases "ever" exposed to PCE (with trichloroethylene and/or methylene chloride) included 1 oral cavity (OR = 0.98, 95% CI = 0.11-8.47), 5 oropharynx (OR = 3.43, 95% CI = 1.01-11.8), 0 hypopharynx, and 4 larynx (OR = 7.95, 95% CI = 1.92-32.9). The 38 trichloroethylene cases were split primarily between oral cavity (12 cases, OR = 2.12, 95% CI = 0.97-4.60), oropharynx (13 cases, OR = 1.66, 95% CI = 0.78-3.54), and larynx (10 cases, OR = 3.80, 95% CI = 1.55-9.32). There was no association between duration, mean intensity of exposure, or cumulative exposure index for PCE and head and neck cancer. There was a small significant relationship between mean intensity of trichloroethylene exposure and head and neck cancer (OR = 1.30, 95% CI = 1.01-1.66). These results suggest a relationship between trichloroethylene and head and neck cancer. The apparent relationship for "ever" exposed to PCE may reflect co-exposure to trichloroethylene.

A companion analysis of head and neck cancers in men was performed as part of the same study (Barul et al. 2017). Methods were the same as reported by (Carton et al. 2017). The analysis included a total of 1,857 cases and 2,780 controls. As for the women, cases smoked more than controls and had higher alcohol consumption. There was no relationship between "ever" exposed to PCE and head and neck cancer in men (OR = 1.04, 95% CI = 0.69-1.59, 70 cases/89 controls). Analysis based on cumulative PCE exposure, however, showed a nonsignificant increase in head and neck cancer risk in the high-exposure group (OR = 1.81, 95% CI = 0.68-4.82, 14 cases/11 controls) that was traced to a significant increase in laryngeal cancer in this group (OR = 3.86, 95% CI = 1.30-11.48, 8 cases). All subjects exposed to PCE were exposed to other chlorinated solvents as well, primarily trichloroethylene. In contrast to the results in women, however, there was no evidence in the men of an association between trichloroethylene exposure and laryngeal cancer or head and neck cancers more broadly.

(Talibov et al. 2014) studied occurrence of acute myeloid leukemia (AML) relative to occupational solvent exposure in a large population-based case-control study in four Nordic countries. The study population comprised a subset of the NOCCA (Nordic Occupational Cancer Study) cohort of 14.9 million individuals from Finland, Iceland, Norway, Denmark, and Sweden who participated in population censuses in 1960, 1970, 1980/1981, and/or 1990. For this study, all incident AML cases diagnosed from 1961 to 2005 were extracted from the NOCCA cohort (the researchers did not have access to individual records from Denmark, so those data were not included). Cases included in the study were at least 20 years of age at diagnosis and had occupational information from at least one census record (n=14,982). Five controls were randomly selected per case, matched for year of birth, sex, and country (n=74,505). Controls were alive and free from AML on the date of diagnosis of the case. Cases and controls could have a history of any cancer other than AML. Occupational exposures to solvents were estimated based on the NOCCA job exposure matrix (developed by national experts from

the Nordic countries), which characterizes proportion of exposed (P) and mean level of exposure for exposed persons (L) for 29 exposure agents in 300 specific occupations over 4 time periods from 1945 to 1994, but does not account for heterogeneity of exposure within an occupation (e.g., with tasks performed or workplace). Cumulative exposure for each subject was calculated by multiplying employment period (T) in years by P × L for each job held and summing the products over their working career (assumed to be ages 20-65 years), based on occupational codes in census records for each subject. The census records provide snapshots in time, but do not provide a complete picture of work history; for this study, it was assumed that when occupation changed from one census to the next that the change occurred in the middle of the time period between censuses. Exposures in the 10 years prior to diagnosis were not counted (alternative lag times of 0, 3, 5, 7, and 20 years were also used, but these data were not shown). Subjects were split into low (0-50th percentile), moderate (50-90th percentile), and high (>90th percentile) cumulative exposure groups in the analysis for each agent. Unexposed subjects served as the reference group, although these data were not shown. Conditional logistic regression was used to estimate HRs. Models included adjustment for exposure to other solvents and also ionizing radiation and formaldehyde. The models did not adjust for suspected lifestyle (e.g., smoking) or genetic risk factors because that information was not available for study subjects.

No significant association was found between PCE exposure and AML (Talibov et al. 2014). HRs in the low (>0-<12.1 ppm/year), medium (12.1-106 ppm/year), and high (>106 ppm/year) cumulative exposure groups were 1.07 (95% CI = 0.83-1.38, 89 cases/472 controls), 0.83 (95% CI = 0.61-1.12, 67 cases/381 controls), and 0.72 (95% CI = 0.39-1.34, 16 cases/96 controls), respectively, and the p-level for dose-response trend was 0.39. There were also no significant findings for other solvents in this study, including benzene, which has shown evidence of a positive association in other studies. A small nonsignificant elevation of AML risk was seen for high cumulative exposure to toluene (HR = 1.35, 95% CI = 0.74-2.46, 76 cases/400 controls). Although the study included a large number of subjects, the low prevalence of occupational exposure to solvents in general, and PCE in particular, limits confidence in these results.

A similar study was performed by (Vlaanderen et al. 2013) to investigate the association between solvent exposure and NHL, MM, and kidney and liver cancer in a subset of the NOCCA cohort. For this study, incident cases of NHL, MM, kidney and liver cancer were extracted from the cohort, which included all NOCCA subjects aged 30-64 years who participated in the 1960, 1970, 1980-1981, and/or 1990 census in Finland, Iceland, Norway, or Sweden and were still alive on January 1 of the year following the census. The study included 76,130 kidney cancer cases, 23,896 liver cancer cases, 69,254 NHL cases, and 35,534 MM cases. For each case, five controls were randomly selected from all cohort members alive and cancer free at the time of diagnosis of the case, matched for age, sex, and country. Occupational exposures to solvents were estimated based on the NOCCA job exposure matrix, as described above. Cumulative exposure was calculated by adding annual exposures, starting at age 20 years or start of working career, whichever occurred later, and ending at incidence date of case or at age 65 years, whichever occurred first. For this study, it was assumed that individuals continued in the same occupation reported in the census until the calendar year in which the census was updated, and that workers had worked in the job they reported in the first census since age of entry into the cohort (30 years). Conditional logistic regression was used to estimate HRs. For analysis, subjects were split into tertiles with approximately equal numbers of exposed controls based on cumulative exposure. Alternatively, high-exposure groups were defined based on 90th percentile of cumulative exposure or 90th percentile of average intensity × prevalence of exposure (calculated by dividing cumulative exposure by duration of exposure). Unexposed subjects served as the reference group in all analyses, although these data were not shown. Pearson correlation coefficients were calculated to describe the

association between potential confounding exposures between agents (solvents and ionizing radiation). The models did not adjust for lifestyle (e.g., smoking, alcohol intake) risk factors because that information was not available for study subjects. Model fit was not affected by lagging calculation of cumulative exposure by 0, 1, 5, 10, or 20 years, so unlagged results were presented.

In the analysis by tertiles of cumulative exposure, no significant associations were found between first, second, or third tertile of cumulative exposure to PCE and NHL, MM, or liver or kidney cancer in men, women, or both sexes combined (Vlaanderen et al. 2013). In the analysis of high-exposure groups, significant or near significant associations were found for NHL in men (HR = 1.54, 95% CI = 0.99-2.42 based on 25 cases using the cumulative exposure metric; HR = 1.74, 95% CI = 1.15-2.64 based on 30 cases using the average intensity × prevalence metric), but not in women (HR = 0.94, 95% CI = 0.74-1.20 based on 77 cases using the cumulative exposure metric; HR = 1.12, 95% CI = 0.88-1.42 based on 83 cases using the average intensity × prevalence metric). PCE findings for other tumors were limited to slight nonsignificant increases in HR for MM and liver cancer in men and/or women based on one or the other of the high-exposure metrics. Among the other agents analyzed, slight associations were noted between ionizing radiation and liver cancer and MM and between benzene and liver cancer. Although PCE exposure in this study was correlated with exposure to trichloroethylene and other chlorinated solvents (no tumor associations found for these agents), it was not correlated with exposure to ionizing radiation or benzene. These results suggest an association between exposure to PCE and NHL in men, and possibly to MM and liver cancer as well, although those data are much weaker. As in the previously described study, the low prevalence of occupational exposure to PCE is a limiting factor for this study.

In another case-control study based on the NOCCA cohort, (Hadkhale et al. 2017) studied the potential link between solvent exposure and bladder cancer. All incident cases of bladder cancer were extracted from the NOCCA cohort, and persons with a minimum age of 20 years at diagnosis and having occupation information from at least one census record before diagnosis were included in the study. Five controls were randomly selected for each case from among individuals alive and free from bladder cancer at the date of diagnosis of the case, matched by birth year and sex. Cases and controls could have a history of any cancer type other than bladder cancer. A total of 113,343 cases and 566,715 controls were included. Occupational exposures to solvents were estimated based on the NOCCA job exposure matrix, as described above. Exposure was assumed to start at the age of 20 years and end at the date of diagnosis or at 65 years, whichever occurred first. If there were different occupational codes in the census records for a given person, the individual was assumed to have changed occupations at the mid-point between two known census years. Cumulative exposure was estimated by summing annual exposure estimates for the entire employment period. In addition to organic solvents, other exposures assessed were ionizing radiation, asbestos, benzo[a]pyrene, diesel engine exhaust, and sulfur dioxide, all considered to be potential confounders. Subjects were split into low (0-50th percentile), moderate (50-90th percentile), and high (>90th percentile) cumulative exposure groups in the analysis for each agent, which was performed by conditional logistic regression. Unexposed subjects served as the reference group. Exposures in the 10 years prior to diagnosis were not counted (lag times of 0 or 20 years were also performed, but these results were not presented). Models were adjusted for exposure to other solvents and agents, but not nonoccupational risk factors (e.g., smoking, alcohol consumption) because that information was not available for study subjects.

HRs for bladder cancer in the low (>0<13.6 ppm/year), medium (13.6-87.55 ppm/year), and high (>87.5 ppm/year) cumulative PCE exposure groups were 1.00 (95% CI = 0.92-1.09, 747 cases/3,560 controls), 1.12 (95% CI = 1.02-1.23, 660 cases/2,783 controls), and 0.94 (95% CI = 0.73-1.22, 159 cases/702 controls), respectively, and the p-level for dose-response trend was 0.10 (Hadkhale et al. 2017). These

results show a slight significant increase in risk of bladder cancer in the medium PCE exposure category, but no increase in the high-exposure group and no significant dose-related trend, suggesting a cause other than PCE exposure for the slight association observed in the medium-exposure group. Bladder cancer risks were significantly elevated in the high-exposure groups for trichloroethylene, benzene, toluene, and ionizing radiation. Although the models included adjustment for co-exposure to other agents, the researchers noted the difficulty of disentangling the effects of PCE and trichloroethylene (structurally similar chemicals with overlapping uses) using the available data. There were approximately 5 times more cases with trichloroethylene exposure than PCE exposure.

(Morales-Suárez-Varela et al. 2013) studied the potential association between occupational solvent exposure and mycosis fungoides (MF, the most common form of cutaneous T-cell lymphoma, a heterogenous group of NHL). Cases were patients aged 35 to 69 years diagnosed with MF in 25 selected areas from six European countries between January 1, 1995, and June 30, 1997. Of 118 pathologically-confirmed cases, 100 agreed to be interviewed for this study (85% participation rate). Population controls were randomly selected from the same areas as cases, frequency matched by sex and age. The study was part of a larger study of seven cancers: MF, gall bladder, small intestine, bone, eye melanoma, thymus, and breast cancer. The controls served as a common pool of controls for all seven groups of cancer cases included in the larger study. In all, 4,629 eligible controls were identified and 3,156 were interviewed (participation rate = 68%). For the MF study, only controls in the strata defined by age and study area where at least one MF case was diagnosed were included (2,846 controls, including 1,957 men and 889 women). Due to illness, 4 case and 95 control interviews were conducted with surrogates. Interviews were performed using standardized questionnaires that included questions on lifestyle factors (smoking, alcohol consumption, etc.) and lifelong occupational history, including details regarding specific tasks performed, products used, etc. Occupational exposures to solvents were assessed for each job held over 6 months using a job exposure matrix developed by the French Institute of Health Surveillance, which provided semiquantitative indicators of exposure probability, frequency, and intensity for each solvent and occupation. A cumulative exposure score for each solvent was calculated for each study subject as the sum of the job-specific exposure scores over his or her lifetime job history. Subjects were split into high- and low-exposure groups based on median cumulative exposure in the analysis for each agent. Unexposed subjects served as the reference group. The analysis was conducted by unconditional logistic regression, with adjustments for age, sex, country, smoking habit, alcohol intake, body mass index, and level of education. No adjustment for co-exposure to other chemicals was noted. Alternative analyses were performed introducing lag times of 5, 10, or 15 years and excluding jobs with low probability of exposure, but these were not shown because they did not affect findings.

For PCE, the results suggested a significant elevation of MF risk in high-dose women (OR = 11.38, 95% CI = 1.04-124.85), but this finding is highly uncertain, as indicated by the extremely wide confidence interval, because it is based on only 2 cases (Morales-Suárez-Varela et al. 2013). There were no female cases with low-dose exposure to PCE. Among men, there were 2 cases with low-dose exposure (OR = 1.80, 95% CI = 0.22-14.80) and 2 with high-dose exposure (OR = 1.60, 95% CI = 0.30-13.60). The low prevalence of PCE exposure and small number of cases in this study limit interpretation of these findings.

(Purdue et al. 2017) conducted an analysis for associations between exposure to PCE and other chlorinated solvents and kidney cancer within the U.S. Kidney Cancer Study, a population-based case-control study conducted in Detroit, Michigan and Chicago, Illinois. Cases were histologically confirmed incident kidney cancer newly diagnosed in Detroit from February 2002 until July 2006 (white cases) or January 2007 (black cases) and in Chicago during 2003. Eligible controls in both locations were selected

from the general population, frequency matched to cases based on sex, age (5-year intervals), and race. The study was designed to maximize the number of black participants. Controls were frequency matched to cases at a 2:1 ratio for blacks and a 1:1 ratio for whites. A total of 1,217 cases (77% of the 1,571 that the researchers attempted to recruit) and 1,235 controls (54% of the 2,269 that the researchers attempted to recruit) participated in the study. Copies of medical records were obtained for all cases to confirm the kidney cancer diagnosis, and the original diagnostic slides were obtained for 706 cases for review by an experienced pathologist. Participants were interviewed for a wide variety of topics including work history for all jobs held for at least 12 months starting at age 16 years. For selected occupations, detailed histories were collected related to solvent exposures.

Job and task exposure matrices were developed for each of the six solvents included in the study by an industrial hygienist using information from a systematic review of the industrial hygiene literature (Purdue et al. 2017). Using the literature review, the exposure matrices, the occupational histories, and the information collected in the job modules, the industrial hygienist assessed levels of exposure probability, frequency, and intensity for each chlorinated solvent for each job. The job-specific estimates of probability, frequency, and intensity for each participant were integrated to develop metrics of exposure for each participant for each chlorinated solvent, including duration of exposure (sum of number of years worked at each job across all jobs with exposure probability ≥50%), cumulative hours exposed (sum of the product of the job-specific frequency midpoint and the job duration in weeks across all jobs with an exposure probability ≥50%), and average weekly exposure (cumulative hours exposed divided by the duration of exposure in weeks).

For the analysis, solvent exposures were split into tertiles among exposed controls, and unexposed participants were used as referents (Purdue et al. 2017). Unconditional logistic regression modelling was performed, including adjustment for location, age, race, sex, education, smoking history, body mass index, and self-reported history of hypertension. Additional analyses incorporated 5- or 15-year exposure lags, restricted participants to individuals with high confidence of exposure, or excluded participants with ≥50% probability of exposure to trichloroethylene.

Prevalence of PCE exposure was low, with <4% of cases and controls assessed as having exposure probability ≥50% (Purdue et al. 2017). Prevalence of exposure was low for other solvents as well, including trichloroethylene. The most common tasks associated with PCE exposure were degreasing and dry cleaning, accounting for 41% and 32% of exposures, respectively. Degreasing also accounted for most exposures to trichloroethylene, carbon tetrachloride, and 1,1,1-trichloroethane. In analyses among controls, after excluding participants unexposed to any chlorinated solvent, solvent exposure probabilities were moderately correlated with one another.

No significant association was found between kidney cancer risk and probability of exposure to PCE (e.g., OR = 1.2, 95% CI = 0.6-2.3, 22 cases/16 controls for those with probability of exposure ≥90%) or PCE exposure duration (e.g., OR = 1.1, 95% CI = 0.5-2.5, 13 cases/11 controls for those exposed ≥10 years), average weekly exposure (e.g., OR = 1.1, 95% CI = 0.4-3.1, 11 cases/14 controls for those exposed >15 hours/week), or cumulative hours of exposure (e.g., OR = 0.9, 95% CI = 0.3-3.3, 8 cases/11 controls for those in highest tertile) for those with ≥50% probability of exposure (Purdue et al. 2017). When the analysis was restricted to those with high-intensity exposure to PCE, however, there was a statistically significant increase in kidney cancer risk for those in the highest tertile of cumulative hours exposed (OR = 3.1, 95% CI = 1.3-7.4, 14 cases/8 controls, Ptrend = 0.03). This relationship was also seen in additional analyses that incorporated 5-year (OR = 3.5, 95% CI = 1.3 10.0, Ptrend = 0.03) or 15-year (OR = 6.2, 95% CI = 1.8-21.3, Ptrend = 0.003) exposure lag periods, included only jobs

assigned an exposure probability with high confidence (OR = 5.1, 95% CI = 1.5-7.2, Ptrend = 0.12), or excluded participants with ≥50% probability of exposure to trichloroethylene (OR = 3.0, 95% CI = 0.99-9.0, 17 cases/14 controls, Ptrend = 0.08). Similar analyses performed for trichloroethylene found no significant associations or exposure-response trends, although a nonsignificant increase in kidney cancer risk was seen in the high tertile of cumulative hours exposed among those with high-intensity exposure (OR = 1.7, 95% CI = 0.8-3.8, 18 cases and 8 controls, Ptrend = 0.28).

This study found no evidence of association between kidney cancer risk and exposure to chlorinated solvents other than PCE and trichloroethylene, and only limited evidence for trichloroethylene (Purdue et al. 2017). High exposure to PCE, however, was associated with kidney cancer, and the result was independent of exposure to trichloroethylene.

(Heck et al. 2013) conducted an exploratory study of exposure to air toxics during pregnancy in relation to risk of neuroblastoma in offspring. Cases of neuroblastoma among California residents younger than 6 years old, born and diagnosed between 1990 and 2007, and listed in the California Cancer Registry were matched to California birth certificates using first and last names and date of birth (89% matching rate). Controls, frequency matched by year of birth to all childhood cancer cases for the same time period, were randomly selected from California birth records of children who had no cancer diagnosis before the age of 6 years and matched to California death records to exclude those (n=1,522) who died of other causes prior to the age of 6. Birth address, as listed on the birth certificate, was used to estimate exposure to air toxics, including PCE, based on distance from each address to monitors in California's air toxics monitoring network (39 air monitors across the state, primarily positioned near heavily trafficked highways, industrial areas, and agriculturally intense rural regions) and measurements made at the nearest monitor to each residence, which were used to calculate average exposures for each trimester and the entire pregnancy period for each participant using date of birth and gestational age obtained from the birth certificate. The study included a total of 75 cases and 14,602 controls who lived within 5 km of a monitor and had measurement values for at least one pollutant. Unconditional logistic regression was used to calculate ORs and CIs, adjusted for mother's age, mother's race, birth year, and method of payment for prenatal care (proxy for family income). No increase in risk of neuroblastoma was seen with PCE exposure for cases within 5 km of a monitor (OR = 1.06, 95% CI = 0.84-1.33, 67 cases/12,041 controls) or within 2.5 km of a monitor (OR = 1.01, 95% CI = 0.62-1.64, 21 cases/3,635 controls).

(Bulka et al. 2016) looked at spatial patterns of diffuse large B-cell lymphoma (DLBCL) incidence in relation to residential proximity to toxic release sites in Georgia. The Georgia Comprehensive Cancer Registry was used to identify all DLBCL cases in adults (≥20 years) residing in Georgia at diagnosis during 1999-2008. Subjects without age, sex, or race information were excluded from the analysis. Included cases (n=3581) were aggregated by census tract, and standardized incidence ratios (SIR) were calculated for each tract by dividing the number of observed cases by expected cases, derived by standardizing DLBCL incidence rates from Georgia to national DLBCL incidence rates by age, sex, and race. GIS (geographic information system) software was used to examine the spatial distribution of TRI (Toxics Release Inventory) sites and SIRs by census tract. From 1988 to 1998, Georgia facilities reported the release of PCE at 33 TRI sites, with releases ranging from 5 to 1,575,644 lb. TRI sites for the other chemicals studied ranged from 3 to 86 sites. The study found that relative risk of DLBCL decreased as mean distance to TRI sites increased for TRI sites for most (8/9) of the contaminants studied, including PCE. The strongest such relationship was found for formaldehyde, which showed a 0.58% decrease in DLBCL risk for every mile of increase in distance to release site. For PCE, the decrease in risk was 0.27% per mile. The effect of mean distance on DLBCL incidence from all of the

15862 release sites was strongest for African Americans. Quantity of chemicals released was not included in
15863 the analysis.
15864

15865 ## F.2 Animal Studies

15866 In a 2-year inhalation study by (NTP 1986a), F344/N rats were exposed to PCE vapors at 0, 200, or 400
15867 ppm for 6 hours/day, 5 days/week for 103 weeks. The incidence of mononuclear cell leukemia (MCL)
15868 showed a positive trend in males (control: 28/50, 200 ppm: 37/50, 400 ppm: 37/50) and females
15869 (control: 18/50, 200 ppm: 30/50, 400 ppm: 29/50), with a dose-related increase in severity of MCL in
15870 both sexes. In addition, the time to onset was decreased in exposed females, compared to controls. When
15871 only advanced (stage 3) MCL was considered, the incidence was statistically significantly increased in
15872 male and female rats exposed to 400 ppm (males - control: 20/50, 200 ppm: 24/50, 400 ppm: 27/50;
15873 females - control: 10/50, 200 ppm: 18/50, 400 ppm: 21/50). The incidence of testicular interstitial cell
15874 tumors was increased in exposed male rats, with a statistically significant positive trend (control: 35/50,
15875 200 ppm: 39/49, 400 ppm: 41/50). Renal tubular cell hyperplasia was observed in exposed male rats
15876 (control: 0/49, 200 ppm: 3/49, 400 ppm: 5/50) and in one treated female rat (1/50 at 400 ppm only), and
15877 renal tubular adenomas and adenocarcinomas were observed in males (combined incidence - control:
15878 1/49, 200 ppm: 3/49, 400 ppm: 4/50) but not females. Although the increase in kidney tumors was not
15879 statistically significant, renal tubular carcinomas are considered rare in this strain of rat and (U.S. EPA
15880 2012c) concluded that a dose-response relationship is apparent when the combined incidence of
15881 proliferative and neoplastic lesions was considered in combination with tumor severity. A biologically
15882 significant elevation of brain gliomas, another rare tumor type, was observed in male (control: 1/50, 200
15883 ppm: 0/50, 400 ppm: 4/50) and female (control: 1/50, 200 ppm: 0/50, 400 ppm: 2/50) rats. The
15884 significance of the brain glioma findings is supported by the earlier occurrence of brain tumors in
15885 exposed animals (week 88 in males, week 75 in females), compared to controls (week 99 in males, week
15886 104 in females) (U.S. EPA 2012c).

15887 In the same study by (NTP 1986a), B6C3F1 mice were exposed to concentrations of PCE of 100 or 200
15888 ppm for 6 hours/day, 5 days/week for 103 weeks. Statistically significant dose-related increases were
15889 observed in the incidence of hepatocellular carcinoma (males - control: 7/49, 100 ppm: 25/49, 200 ppm:
15890 26/50; females - control: 1/48, 100 ppm: 13/50, 200 ppm: 36/50) and combined incidence of
15891 hepatocellular adenomas or carcinomas in male and female mice (males - control: 17/49, 100 ppm:
15892 31/49, 200 ppm: 41/50; females - control: 4/48, 100 ppm: 17/50, 200 ppm: 38/50). The incidences of
15893 hepatocellular carcinoma and hepatocellular adenomas or carcinomas combined were significantly
15894 increased, compared to controls, at both 100 and 200 ppm in males and females. In several instances,
15895 hepatocellular carcinomas metastasized to the lungs in males (control: 2/49, 100 ppm: 7/49, 200 ppm:
15896 1/50) and females (control: 0/48, 100 ppm: 2/50, 200 ppm: 7/50).

15897 In a 2-year inhalation study conducted by (JISA 1993), F344/DuCrj rats were exposed to PCE vapors at
15898 0, 50, 200, or 600 ppm. A statistically significant dose-related increase (statistical analysis by statistical
15899 analysis by statistical analysis by statistical analysis by U.S. EPA 2012c) was observed in the incidence
15900 of MCL in males (control: 11/50, 50 ppm: 14/50, 200 ppm: 22/50, 600 ppm: 27/50) and females
15901 (control: 10/50, 50 ppm: 17/50, 200 ppm: 16/50, 600 ppm: 19/50). The increase in MCL incidence
15902 achieved statistical significance in males exposed to 600 ppm, compared to control males. The time to
15903 first occurrence of MCL was decreased in exposed female rats (weeks 66-74 in exposed groups)
15904 compared to control female rats (week 100). Also, there was a dose-related increase in the overall
15905 number of unscheduled deaths attributed to MCL in males and females.

(JISA 1993) also exposed Crj:BDF1 mice to PCE at 0, 10, 50, or 250 ppm for 6 hours/day, 5 days/week for 104 weeks. Dose-related increases in the incidences of hepatocellular adenomas (males - control: 7/50, 10 ppm: 13/50, 50 ppm: 8/50, 250 ppm: 26/50; females - control: 3/50, 10 ppm: 3/47, 50 ppm: 7/49, 250 ppm: 26/49), hepatocellular carcinomas (males - control: 7/50, 10 ppm: 8/50, 50 ppm: 12/50, 250 ppm: 25/50; females - control: 0/50, 10 ppm: 0/47, 50 ppm: 0/49, 250 ppm: 14/49), and combined hepatocellular adenomas or carcinomas were observed in males and females (males - control: 13/50, 10 ppm: 21/50, 50 ppm: 19/50, 250 ppm: 40/50; females - control: 3/50, 10 ppm: 3/47, 50 ppm: 7/49, 250 ppm: 33/49). The incidences of hepatocellular adenoma, hepatocellular carcinoma, and combined hepatocellular adenoma or carcinoma were statistically significantly increased at 250 ppm, relative to controls, in both sexes. A small increase in liver and spleen hemangiosarcomas (reported as malignant hemangioendotheliomas) was also observed in treated male mice (liver - control: 1/50, 10 ppm: 1/50, 50 ppm: 5/50, 250 ppm: 5/50; spleen - control: 1/50, 10 ppm: 1/50, 50 ppm: 3/50, 250 pm: 5/50). The combined incidence of hemangiosarcomas or hemangiomas (reported as malignant or benign hemangioendotheliomas, respectively) occurring in the liver, spleen, fat, subcutaneous skin, and heart was statistically significantly increased in male mice (combined incidence - control: 4/50, 10 ppm: 2/50, 50 ppm: 7/50, 250 ppm: 11/50) (analysis by (U.S. EPA 2012c)). In addition, there was a statistically significant positive dose-related trend in the incidence of adenoma of the Harderian gland in male mice (control: 2/50, 10 ppm: 2/50, 50 ppm: 2/50, 250 ppm: 8/50).

In a lifetime bioassay by (NCI 1977), Osborne-Mendel rats were administered PCE for 78 weeks via gavage in corn oil for 5 days/week, followed by a 32-week observation period. Dose adjustments were made throughout the exposure period depending upon the tolerance of treated animals to the existing dose level. Administered doses were 500-700 mg/kg-day in the low dose and 1,000-1,400 mg/kg-day in the high-dose males, with 7 dose-free weeks occurring intermittently during the last 33 weeks of exposure. Time-weighted average (TWA) doses during the 78-week treatment period were approximately 470 mg/kg-day at the low dose and approximately 950 mg/kg-day at the high dose. Rats showed no significant treatment-related increases in neoplastic lesions, compared to controls, and there were no significant positive dose-related trends. A high rate of early death was observed in treated rats. At the high dose, mortality was 50% in males by week 44 and in females by week 66. Respiratory disease and pneumonia were observed in both treated and control rats, while toxic nephropathy occurred only in treated animals (males - low dose: 43/49, high dose: 47/50; females - low dose: 29/50, high dose: 39/50). Due to the high rate of early death in treated rats, (NCI 1977) determined that the carcinogenicity of PCE in rats could not be evaluated from the results of this study.

(NCI 1977) also exposed B6C3F1 mice to PCE by gavage in corn oil for 78 weeks (5 days/week), followed by a 12-week observation period. Male mice were administered 450 or 900 mg/kg-day for the first 11 weeks, after which the doses were increased to 550 or 1,100 mg/kg-day, respectively, for the next 67 weeks. Female mice received 300 or 600 mg/kg-day during the first 11 weeks, and doses were increased to 400 or 800 mg/kg-day, respectively, for the subsequent 67 weeks. The TWA doses (5 days/week for 78 weeks) were 536 and 1,072 mg/kg-day for males and 386 and 772 mg/kg-day for females. The incidence of hepatocellular carcinoma was statistically significantly increased in treated male and female mice of both dose groups, compared with controls (males - untreated control: 2/17, vehicle control: 2/20, 536 mg/kg-day: 32/49, 1,072 mg/kg-day: 27/48; females - untreated control: 2/20, vehicle control: 0/20, 386 mg/kg-day: 19/48, 772 mg/kg-day: 19/48); the time to first tumor was also decreased in treated mice (weeks 27-40 in males, weeks 41-50 in females) compared to controls (weeks 90-91 in males, week 91 in females). Metastasis of hepatocellular carcinomas to the lung was observed in 3/49 low-dose males, 1/49 low-dose females, and 1/48 high-dose females.

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Appendix G Chronic Inhalation Risk Estimates Using Occupational HECs

Table_Apx G-1 presents risk chronic inhalation risk estimates for each OES based on the occupational HECs for neurotoxicity presented in Table 3-8. These HECs are based on 8 hr or 12 hr LOAEC PODs and were compared to 8 or 12 hr TWA exposures for calculating MOEs. Risk estimates are shown without a respirator as well as with APF = 50 for workers, the highest plausible respiratory protection expected to be used by workers on a regular basis. Occupational Exposure Scenarios (OES) that are highlighted in gold demonstrate differing risk conclusions than shown in Section 4.3 (i.e. not using occupational HECs) based either on worker risk estimates with APF = 50 or ONU estimates without a respirator. Of note, occupational HECs were derived based on an expected normal, full time work schedule. For OES where exposure is expected for significantly less than 250 days/year (both of Other DOD uses), these HEC values are likely to overestimate risk.

Table_Apx G-1. Chronic Inhalation Risk Estimates by OES

8 hr HEC = 14.5 ppm 12 hr HEC = 9.7 ppm					**Benchmark MOE = 100**	
			MOEs for Chronic Exposure			
Occupational Exposure Scenario	**Occupational HEC (ppm)**	**Exposure Level**	**Worker No respirator**	**ONU No respirator**	**Worker APF 50**	**Benchmark MOE (= Total UF)**
Manufacturing (8 hr)	14.5	High-End	**5.6**	446	278	100
		Central Tendency	446		22308	
Manufacturing (12 hr)	9.7	High-End	**46**	472	2280	100
		Central Tendency	472		23577	
Repackaging	14.5	High-End	**18**	**33**	885	100
		Central Tendency	**33**		1666	
Processing as a reactant (8hr)	14.5	High-End	**5.6**	446	278	100
		Central Tendency	446		22308	
Processing as a reactant (12hr)	9.7	High-End	**46**	472	2280	100
		Central Tendency	472		23577	
Incorporation into Formulation - Aerosol Packing	14.5	High-End	**1.1**	**1.7**	**55**	100
		Central Tendency	**1.7**		**87**	
Incorporation into Formulation - Degreasing Solvent	14.5	High-End	**5.6**	**20**	279	100
		Central Tendency	**20**		994	
Incorporation into Formulation - Dry Cleaning Solvent	14.5	High-End	**1.0**	**3.7**	**51**	100
		Central Tendency	**3.7**		183	

8 hr HEC = 14.5 ppm 12 hr HEC = 9.7 ppm				Benchmark MOE = 100		
			MOEs for Chronic Exposure			
Occupational Exposure Scenario	**Occupational HEC (ppm)**	**Exposure Level**	**Worker No respirator**	**ONU No respirator**	**Worker APF 50**	**Benchmark MOE (= Total UF)**
Incorporation into Formulation - Miscellaneous	14.5	High-End	**10**	**36**	513	100
		Central Tendency	**36**		1825	
Batch Open-Top Vapor Degreasing	14.5	High-End	**0.5**	**2.8**	**23**	100
		Central Tendency	**6.9**	**24**	345	
Batch Closed-Loop Vapor Degreasing	14.5	High-End	**57**	151	2865	100
		Central Tendency	201	222	10043	
Conveyorized Vapor Degreasing	14.5	High-End	**7.80E-2**	**0.1**	**3.9**	100
		Central Tendency	**0.2**	**0.4**	**9.3**	
Web Degreasing	14.5	High-End	**8.0**	**12**	402	100
		Central Tendency	**24**	**45**	1187	
Cold Cleaning (Monitoring)	14.5	High-End	**3.5**	EPA did not identify ONU monitoring data	176	100
		Central Tendency	**10**		518	
Cold Cleaning (Modeling)	14.5	High-End	**9.4**	19	472	100
		Central Tendency	6048	11685	302423	
Aerosol Degreasing/ Lubricants (Monitoring)	14.5	High-End	**1.9**	EPA did not identify ONU monitoring data	93	100
		Central Tendency	**10**		504	
Aerosol Degreasing/ Lubricants (Modeling)	14.5	High-End	**0.8**	**20**	**42**	100
		Central Tendency	**2.6**	145	132	
Dry Cleaning and Spot Cleaning - Post-2006 (Monitoring)	14.5	High-End	**0.7**	**42**	**37**	100
		Central Tendency	**4.0**	**42**	199	
Dry Cleaning and Spot Cleaning - Post-2006 (Modeling)	14.5	High-End	**0.3**	**6.2**	**16**	100
		Central Tendency	**6.9**	**89**	346	
Dry Cleaning and Spot Cleaning - 4th/5th Gen Only	14.5	High-End	**2.6**	118	130	100
		Central Tendency	**15**	1039	741	
Paints/Coatings	14.5	High-End	**3.2**	**62**	159	100

8 hr HEC = 14.5 ppm 12 hr HEC = 9.7 ppm					Benchmark MOE = 100	
Occupational Exposure Scenario	**Occupational HEC (ppm)**	**Exposure Level**	**MOEs for Chronic Exposure**			**Benchmark MOE (= Total UF)**
			Worker No respirator	**ONU No respirator**	**Worker APF 50**	
		Central Tendency	**62**		3107	
Adhesives	14.5	High-End	**18**	164	894	100
		Central Tendency	164		8193	
Maskant for Chemical Milling	14.5	High-End	**6.9**	**12**	345	100
		Central Tendency	**12**		598	
Industrial Processing Aid	14.5	High-End	**12**	242	614	100
		Central Tendency	242		12083	
Metalworking Fluids	14.5	High-End	692	2521	34616	100
		Central Tendency	2521		126038	
Wipe Cleaning and Metal/Stone Polishes	14.5	High-End	**6.36E-02**	**0.6**	**3.2**	100
		Central Tendency	**0.1**	664	**5.5**	
Other Spot Cleaning/Spot Removers	14.5	High-End	**63**	483	3142	100
		Central Tendency	**84**		4219	
Other Industrial Uses	14.5	High-End	403	1822	20153	100
		Central Tendency	1822		91115	
Other Commercial Uses - Printing	14.5	High-End	**2.4**	**7.6**	122	100
		Central Tendency	**7.6**		378	
Other Commercial Uses - Photocopying	14.5	High-End	29000	77333	1450000	100
		Central Tendency	77333		3866667	
Other Commercial Uses - Photographic Film	14.5	High-End	**0.3**	**2.3**	**13**	100
		Central Tendency	**2.3**		115	
Other Commercial Uses - Mold Release	14.5	High-End	**73**	145	3625	100
		Central Tendency	145		7250	
Waste Handling, Disposal, Treatment, Recycling	14.5	High-End	403	1822	20153	100
		Central Tendency	1822		91115	

8 hr HEC = 14.5 ppm 12 hr HEC = 9.7 ppm				Benchmark MOE = 100		
			MOEs for Chronic Exposure			
Occupational Exposure Scenario	**Occupational HEC (ppm)**	**Exposure Level**	**Worker No respirator**	**ONU No respirator**	**Worker APF 50**	**Benchmark MOE (= Total UF)**
Other DOD Uses - Water Pipe Repair	14.5	High-End	**6.3**	**13**	314	100
		Central Tendency	**13**		627	
Other DOD Uses - Oil Analysis	14.5	High-End	**16**	**16**	823	
		Central Tendency				

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