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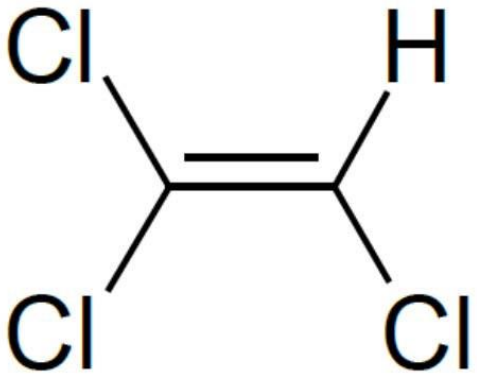

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Docket

Supporting information can be found in public docket (Docket: EPA-HQ-OPPT-2019-0500).

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ABBREVIATIONS

°C	Degrees Celsius
ε_0	Vacuum Permittivity
ACGIH	American Conference of Governmental Industrial Hygienists
AEGL	Acute Exposure Guideline Level
ADD	Average Daily Dose
AF	Assessment Factor
AQS	Air Quality System
ATCM	Airborne Toxic Control Measure
atm	Atmosphere(s)
ATSDR	Agency for Toxic Substances and Disease Registries
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BIOWIN	The EPI Suite™ module that predicts biodegradation rates
$BW^{3/4}$	body weight$^{3/4}$
CAA	Clean Air Act
CARB	California Air Resources Board
CASRN	Chemical Abstracts Service Registry Number
CBI	Confidential Business Information
CCR	California Code of Regulations
CDC	Centers for Disease Control and Prevention
CDR	Chemical Data Reporting
CEHD	Chemical Exposure Health Data
CEM	Consumer Exposure Model
CEPA	Canadian Environmental Protection Act
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFC	Chlorofluorocarbon
CFR	Code of Federal Regulations
CH	Chloral Hydrate
CHIRP	Chemical Risk Information Platform
ChV	Chronic Value
cm^3	Cubic Centimeter(s)
CNS	Central Nervous System
COC	Concentration of Concern
COU	Conditions of Use
CPCat	Chemical and Product Categories
CSCL	Chemical Substances Control Law
CWA	Clean Water Act
CYP	Cytochrome P450
DCA	Dichloroacetic acid
DCVC	S-dichlorovinyl-L-cysteine
DCVG	S-dichlorovinyl-glutathione
DEVL	Dermal Exposure to Volatile Liquids
DIY	Do-It-Yourself
DMR	Discharge Monitoring Report
EC_{50}	Effect concentration at which 50% of test organisms exhibit an effect
ECCC	Environment and Climate Change Canada
ECHA	European Chemicals Agency

EDC	Ethylene Dichloride
E-FAST	Exposure and Fate Assessment Screening Tool
EG	Effluent Guidelines
EPA	Environmental Protection Agency
EPCRA	Emergency Planning and Community Right-to-Know Act
EPI Suite™	Estimation Program Interface Suite™
ESD	Emission Scenario Document
EU	European Union
FDA	Food and Drug Administration
FFDCA	Federal Food, Drug, and Cosmetic Act
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FR	Federal Register
g	Gram(s)
GACT	Generally Available Control Technology
GS	Generic Scenario
GSH	Glutathione
GST	Glutathione-S-transferase
HAP	Hazardous Air Pollutant
HCFC	Hydrochlorofluorocarbon
HCl	Hydrochloric Acid
HC_{05}	Hazardous Concentration threshold for 5% of species in a Species Sensitivity Distribution
HEC	Human Equivalent Concentration
HED	Human Equivalent Dose
HFC	Hydrofluorocarbon
HHE	Health Hazard Evaluation
HPV	High Production Volume
Hr	Hour
IARC	International Agency for Research on Cancer
ICIS	Integrated Compliance Information System
IDLH	Immediately Dangerous to Life and Health
IMIS	Integrated Management Information System
IRIS	Integrated Risk Information System
ISHA	Industrial Safety and Health Act
ISOR	Initial Statement of Reasons
IUR	Inhalation Unit Risk
K_{oc}	Soil Organic Carbon-Water Partitioning Coefficient
K_{ow}	Octanol/Water Partition Coefficient
kg	Kilogram(s)
L	Liter(s)
lb	Pound(s)
LC_{50}	Lethal Concentration at which 50% of test organisms die
LOAEL	Lowest-observed-adverse-effect-level
LOEC	Lowest-observable-effect Concentration
m^3	Cubic Meter(s)
MACT	Maximum Achievable Control Technology
MATC	Maximum Acceptable Toxicant Concentration
MCCEM	Multi-Chamber Concentration and Exposure Model
MCL	Maximum Contaminant Level

MCLG	Maximum Contaminant Level Goal
mg	Milligram(s)
mmHg	Millimeter(s) of Mercury
MOA	Mode of Action
mPa·s	Millipascal(s)-Second
MSDS	Material Safety Data Sheet
MSW	Municipal Solid Waste
NAICS	North American Industry Classification System
NATA	National Scale Air-Toxics Assessment
NCEA	National Center for Environmental Assessment
NICNAS	Australia National Industrial Chemicals Notification and Assessment Scheme
NCP	National Contingency Plan
NEI	National Emissions Inventory
NESHAP	National Emission Standards for Hazardous Air Pollutants
NHANES	National Health and Nutrition Examination Survey
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIH	National Institute of Health
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NIOSH	National Institute for Occupational Safety and Health
NITE	National Institute of Technology and Evaluation
NOAEL	No-Observed-Adverse-Effect-Level
NOEC	No-observable-effect Concentration
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulation
NRC	National Research Council
NTP	National Toxicology Program
NWIS	National Water Information System
OCPSF	Organic Chemicals, Plastics and Synthetic Fibers
OCSPP	Office of Chemical Safety and Pollution Prevention
OECD	Organization for Economic Co-operation and Development
OEHHA	Office of Environmental Health Hazard Assessment
OES	Occupational Exposure Scenario
OEL	Occupational Exposure Limits
ONU	Occupational Non-User
OPPT	Office of Pollution Prevention and Toxics
OR	Odds Ratio
OSHA	Occupational Safety and Health Administration
OSF	Oral Slope Factor
OST	Office of Science and Technology
OTVD	Open-Top Vapor Degreaser
OW	Office of Water
PBPK	Physiologically-Based Pharmacokinetic
PBZ	Personal Breathing Zone
PCE	Tetrachloroethylene
PECO	Population, Exposure, Comparator, and Outcome
PEL	Permissible Exposure Limit
PESS	Potentially Exposed or Susceptible Subpopulations
POD	Point of Departure

POTW	Publicly Owned Treatment Works
ppb	Part(s) per Billion
PPE	Personal Protective Equipment
ppm	Part(s) per Million
PSD	Particle Size Distribution
PV	Production Volume
QC	Quality Control
QSAR	Quantitative Structure Activity Relationship
RCRA	Resource Conservation and Recovery Act
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
REL	Relative Exposure Limit
RR	Relative Risk
RTR	Risk and Technology Review
SDS	Safety Data Sheet
SDWA	Safe Drinking Water Act
SIDS	Screening Information Dataset
SNUN	Significant New Use Notice
SNUR	Significant New Use Rule
SOCMI	Synthetic Organic Chemical Manufacturing Industry
SPARC	SPARC Performs Automated Reasoning in Chemistry
SpERC	Specific Environmental Release Categories
STEL	Short-Term Exposure Limit
STP model	Sewage Treatment Plant model
STORET	STOrage and RETrieval
SSD	Species Sensitivity Distribution
TCCR	Transparent, clear, consistent, and reasonable
TCA	Trichloroacetic acid
TCE	Trichloroethylene
TCOH	Trichloroethanol
TCOG	Trichloroethanol, gluuronide conjugate
TNSSS	Targeted National Sewage Sludge Survey
TLV	Threshold Limit Value
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	Time Weighted Average
UIC	Underground Injection Control
U.S.	United States
UV	Ultraviolet
USGS	United States Geological Survey
VOC	Volatile Organic Compound
VP	Vapor Pressure
Yr	Year(s)

EXECUTIVE SUMMARY

This draft risk evaluation for trichloroethylene 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 trichloroethylene. 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 or no unreasonable risk under the conditions of use, in accordance with TSCA section 6, and are not intended to represent any findings under TSCA section 7.

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

Trichloroethylene has a wide-range of uses in consumer and commercial products and in industry. An estimated 83.6% of TCE's annual production volume is used as an intermediate in the manufacture of the hydrofluorocarbon, HFC-134a, an alternative to the refrigerant chlorofluorocarbon, CFC-12. Another 14.7% of TCE production volume is used as a degreasing solvent, leaving approximately 1.7% for other uses. The total aggregate production volume decreased from 220.5 to 171.9 million pounds between 2012 and 2015.

EPA evaluated TCE's conditions of use (COUs), including the following categories of use: solvent for cleaning and degreasing, lubricants and greases, adhesives and sealants, functional fluids in a closed system, paints and coatings, laundry and dishwashing products, arts, crafts and hobby materials, and process solvent recycling and worker handling of wastes. Trichloroethylene is subject to federal and state regulations and reporting requirements. Trichloroethylene 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 as a Hazardous Air Pollutant (HAP) under the Clean Air Act (CAA), is a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), and is regulated as a hazardous waste under the Resource Conservation and Recovery Act (RCRA). 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. Under TSCA, EPA previously assessed risks from use of trichloroethylene in commercial solvent degreasing (aerosol and vapor), consumer use as a spray applied protective coating for arts and crafts and commercial use as a spot remover at dry cleaning facilities (U.S. EPA, 2014b).

Approach

EPA used reasonably available information (defined in 40 Code of Federal Regulations (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 scope document and 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 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). EPA quantitatively evaluated the risk to aquatic species from exposure to surface water. EPA evaluated the risk to workers, from inhalation and dermal exposures, and occupational non-users (ONUs)[1], from inhalation exposures, by comparing the estimated exposures to acute and chronic human health hazards. EPA also evaluated the risk to consumers, from inhalation and dermal exposures, and bystanders, from inhalation exposures, by comparing the estimated exposures to acute human health hazards.

EPA used environmental fate parameters, physical-chemical properties, modeling, and monitoring data to assess ambient water exposure to aquatic organisms. While trichloroethylene is present in various environmental media, such as groundwater, surface water, and air, EPA determined during problem formulation that no further analysis beyond what was presented in the problem formulation document (Section 2.5.3.3 in (U.S. EPA, 2018d)) would be done for environmental exposure pathways for land application of biosolids and sediment, and water or soil pathways for terrestrial organisms, in this draft risk evaluation because TCE is not anticipated to partition to biosolids during wastewater treatment. It is expected to primarily volatilize. However, exposures to aquatic organisms from ambient surface water, are assessed and presented in this draft risk evaluation. These analyses are described in Sections 2.1 and 2.2.

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). As stated in Section 3.1, the reasonably available environmental hazard data indicate that TCE presents hazard to aquatic organisms. For acute exposures, aquatic invertebrates are the most sensitive species with toxicity values ranging from 7.8 mg/L to 33.85 mg/L (resulting in a geometric mean of 16 mg/L). For chronic exposures, toxicity values for fish and aquatic invertebrates are as low as 7.88 mg/L and 9.2 mg/L, respectively. The data also indicated that TCE presents hazard for aquatic plants, with toxicity values in algae as low as 0.03 mg/L, and a wide range in toxicity between algae species. TCE is not expected to accumulate in aquatic organisms.

EPA evaluated exposures to trichloroethylene in occupational and consumer settings for the conditions of use included in the scope of the risk evaluation, listed in Section 1.4. In occupational settings, EPA evaluated acute and chronic inhalation exposures to workers and ONUs, and acute and chronic dermal

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

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 were estimated since inhalation and dermal monitoring data were not reasonably available. These analyses are described in Section 2.3 of this draft risk evaluation.

EPA evaluated reasonably available information for human health hazards and identified hazard endpoints including acute and chronic toxicity for non-cancer effects and cancer, as described in Section 3.2. EPA used the *Framework for Human Health Risk Assessment to Inform Decision Making* (U.S. EPA, 2014a) to evaluate, extract, and integrate trichloroethylene's human health hazard and dose-response information. EPA reviewed key and supporting information from previous hazard assessments [TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Use (U.S. EPA, 2014b), Toxicological Review of Trichloroethylene (U.S. EPA, 2011e), and other national and international assessments listed in Table 1-3], (however all data sources from prior assessments were independently reviewed for this risk evaluation). EPA also screened and evaluated studies that were published since these reviews (i.e., from 2010 – 2017, in addition to select studies published after completion of the literature search).

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, chronic and non-cancer endpoints, and inhalation unit risk (IUR) and cancer slope factors (CSF) for cancer risk estimates. Health hazards of TCE described and reviewed in this risk evaluation include: acute overt toxicity, liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity (including sensitization), reproductive toxicity, developmental toxicity, and cancer. Following dose-response analysis, representative PODs were identified for multiple non-cancer endpoints within the domains of liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity, reproductive toxicity, and developmental toxicity.

For cancer, EPA performed meta-analyses in order to statistically evaluate the epidemiological data for non-Hodgkin Lymphoma (NHL), kidney cancer, and liver cancer. EPA utilized similar methodology as was employed in the 2011 EPA TCE IRIS Assessment (U.S. EPA, 2011e) and included sensitivity analyses, as needed, to partition the results based on both heterogeneity and study quality. See Appendix H for full details and results. The 2019 meta-analysis of all relevant studies examining kidney cancer, liver cancer, or NHL (Appendix H) concluded that there is a statistical significant association between TCE exposure and increased incidence of all three cancers. For context, this was the same conclusion as the previous EPA meta-analysis in the 2011 IRIS Assessment (U.S. EPA, 2011e), which evaluated older literature than the current assessment. Therefore, EPA utilized the same inhalation unit risk and oral slope factor estimates as were derived in (U.S. EPA, 2011e) and cited in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). A linear non-threshold assumption was applied to the TCE cancer dose-response analysis because there is sufficient evidence that TCE-induced kidney cancer operates primarily through a mutagenic mode of action while it cannot be ruled out for the other two cancer types.

Risk Characterization

Environmental Risk: For environmental risk, EPA utilized a risk quotient (RQ) to compare the environmental concentration to the effect level to characterize the risk to aquatic organisms. EPA included a qualitive assessment describing trichloroethylene exposure from sediments for aquatic organisms, and land-applied biosolids, water, and soil for terrestrial organisms. Trichloroethylene is not expected to accumulate in sediments, and is expected to be mobile in soil, and migrate to water or volatilize to air. The results of the risk characterization are in Section 4.1, including a table (Table 4-1). that summarizes the RQs for acute and chronic risks. Surface water concentrations of TCE were modeled for 214 releases.

EPA identified the expected environmental exposures for aquatic species under the conditions of use in the scope of the risk evaluation. Estimated releases from specific facilities result in modeled surface water concentrations that exceed the aquatic benchmark (RQ ≥ 1) for either chronic, acute, and/ or algae concentrations of concern for the following conditions of use in various locations (see Table 4-1): processing as a reactant; open top vapor degreasing; repackaging; adhesives; sealants; paints and coatings; industrial processing aid; other industrial uses; other commercial uses; process solvent recycling and worker handling of wastes; and waste water treatment plants. Details of these estimates are in Section 4.1.2.

Qualitative consideration of the physical-chemical and fate characteristics, as well as consideration of the conditions of use for TCE indicated limited presence in terrestrial environments and aquatic sediments (Section 4.1.3 and 4.1.4). Therefore EPA did not find risks for sediment or terrestrial organisms.

Human Health Risks: Risks were estimated following both acute and chronic exposure for representative endpoints from every hazard domain.

For workers and ONUs, EPA estimated potential cancer risk from chronic exposures to trichloroethylene using inhalation unit risk or dermal cancer slope factor values multiplied by the chronic exposure for each COU. For workers and ONUs, EPA also estimated potential non-cancer risks resulting from acute and chronic inhalation and dermal exposures using a Margin of Exposure (MOE) approach. For workers, EPA estimated risks using several occupational exposure scenarios, with scenario-specific assumptions regarding the expected use of personal protective equipment (PPE) for respiratory and dermal exposures for workers directly handling trichloroethylene. More information on respiratory and dermal protection, including EPA's approach regarding the occupational exposure scenarios for trichloroethylene, is in Section 2.3.1.

For the majority of exposure scenarios, risks to workers were identified for multiple endpoints in both acute and chronic exposure scenarios. Based on the most robust and well-supported PODs selected from among the most sensitive acute and chronic endpoints, acute and chronic non-cancer and cancer risks were indicated for all exposure scenarios and occupational conditions of use under high-end[2] inhalation exposure levels. Non-cancer risks following chronic exposure were also identified for all exposure scenarios at high-end exposure levels with expected use of respiratory protection up to APF = 50. When

[2] A high-end is assumed to be representative of occupational exposures that occur at probabilities above the 90th percentile but below the exposure of the individual with the highest exposure. EPA provided results at the 95th percentile when available.

only considering the central tendency[3] inhalation exposure level, risks were not identified for three out of 18 occupational exposure scenarios. Acute and chronic non-cancer and cancer risks were indicated for all exposure scenarios and occupational conditions of use under both high-end and central tendency dermal exposure levels. Risks are still identified for all exposure scenarios (at high-end exposure levels following acute exposure and at both exposure levels following chronic exposure) when gloves are worn even when assuming the maximum applicable glove protection (either PF 10 or 20).

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. Therefore, for those instances where monitoring data or modeling did not distinguish between worker and far-field ONU inhalation exposure estimates, EPA considered the worker risk estimates when determining far-field ONU risk. There is significant uncertainty in these ONU inhalation risk estimates. While the difference between the exposures of ONUs and the exposures of workers directly handling TCE 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 TCE, nor they are in the immediate proximity of TCE.

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 trichloroethylene used in their vicinity. ONUs are not expected to be dermally exposed to trichloroethylene 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 and Table 4-54 in Section 4.5.1.0.

For consumers and bystanders for consumer use, EPA estimated non-cancer risks resulting from acute inhalation or dermal exposures (applicable to consumers only) that were modeled with a range of user intensities, described in detail in Section 2.3.2. Bystanders are assumed to not have direct dermal contact with TCE. Based on reasonably available information, EPA determined that consumers or bystanders would not use PPE and that all exposures would be acute, rather than chronic.

For consumers, risks were identified for multiple acute endpoints acute risks were indicated for all consumer conditions of use except Pepper Spray at both medium and high-intensity acute inhalation and dermal consumer exposure scenarios. Acute risks were also indicated for most conditions of use for bystanders at both medium and high-intensity acute inhalation levels. EPA's estimates for consumer and bystander risks for each consumer use exposure scenario are presented in Section 4.2.3 and summarized in Table 4-55 in Section 4.5.2.2.

Uncertainties: Key assumptions and uncertainties in the environmental risk estimation include uncertainties regarding the hazard data for aquatic species and surface water concentrations. Additionally the reasonably available environmental monitoring data was limited temporally and

[3] A central tendency is assumed to be representative of occupational exposures in the center of the distribution for a given condition of use. For risk evaluation, EPA used the 50th percentile (median), mean (arithmetic or geometric), mode, or midpoint values of a distribution as representative of the central tendency scenario.

geographically.

For the human health risk estimation, key assumptions and uncertainties are related to data on exposures, exposure model input parameters, and the estimates for ONU inhalation exposures for COUs in which monitoring data or probabilistic modeling data was not reasonably available. Additional sources of uncertainty related to human health hazard include selection of the appropriate Physiologically-Based Pharmacokinetic (PBPK) dose-metric for each endpoint, the dose-response for the congenital heart defect endpoint, and the adjustment of the cancer PODs to account for cancer at multiple sites. Assumptions and key sources of uncertainty in the risk characterization are detailed in Section 4.3.

Potentially Exposed or Susceptible Subpopulations (PESS): TSCA § 6(b)(4) requires that EPA conduct a risk evaluation to "*determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use*." 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*."

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 potentially exposed groups, EPA considered trichloroethylene exposures to be higher among workers using trichloroethylene and ONUs in the vicinity of trichloroethylene use than the exposures experienced by the general population. Risk estimates were also provided separately for ONUs when sufficient data were reasonably available. EPA was unable to provide separate risk estimates when insufficient information was reasonably available for quantifying ONU exposure. EPA considered the central tendency risk estimate when determining ONU risk for those conditions of use for which ONU exposures were not separately estimated. Consumer risk estimates were provided for low, medium, and high intensities of use, accounting for differences in duration, weight fraction, and mass used. Dermal risk estimates were calculated for both average adult workers and women of childbearing age. The use of the 99th percentile Human Equivalent Concentration/Dose $(HEC/HED)_{99}$ POD values derived from relevant (PBPK) dose metrics also account for the vast majority of toxicokinetic variation across the population. By relying on the 99th percentile output of the PBPK model, these values are expected to be protective of particularly susceptible subpopulations, including those with genetic polymorphisms resulting in increased activity of bioactivating enzymes. While there may not be a risk for all endpoints to all individuals or to an individual at all times, assessment of risks for all relevant endpoints using toxicokinetic values for the most sensitive 1% of the population is expected to sufficiently cover any particularly susceptible subpopulations.

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

pathways at this time within a condition of use because of the uncertainties present in the current exposure estimation procedures, which may may lead to an underestimate or overestimate of the actual total 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. EPA considered sentinel exposures by considering risks to populations who may have upper bound (e.g., high-end, high intensities of use) exposures.

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 identified risks from acute and chronic exposures for aquatic organisms (e.g., aquatic invertebrates and fish) near two facilities releasing TCE to surface water. One facility had an acute RQ greater than 1 (RQ = 3.11), exceeding the acute COC of 3,200 ppb and indicating risk to aquatic organisms from acute exposures. This facility is one of 59 facilities modeled by EPA that use TCE for open-top vapor degreasing (see Section 4.5.1). This facility and one other facility (one of 11 facilities that process TCE as a reactant) had chronic RQs greater than 1, exceeding the chronic COC of 788 ppb for 20 days (see Section 4.5.1). Monitored data from the Water Quality Portal and grey literature show no exceedances of the acute COC and the chronic COC in ambient water. Monitored data from literature showed some exceedances of the algae COC of 3 ppb in ambient water; however, the data show no exceedances of the algae COC of 52,000 ppb. Therefore, EPA did not identify risks for acute or chronic exposure durations in ambient water for areas where monitored data were reasonably available. Given the uncertainties in the modeling data and exceedance of the acute RQ for only one data point and of the chronic RQ for only two data points out of 70 facilities modeled, EPA does not consider these risks unreasonable (see Section 5.1).

Risks of Injury to Health: EPA's determination of unreasonable risk for specific conditions of use of TCE 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 relevant for these conditions of use. TCE 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. Risks from acute exposures include developmental toxicity and pulmonary immunotoxicity. For chronic exposures, EPA identified risks of non-cancer effects (liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity,

reproductive toxicity, and developmental toxicity) as well as cancers of liver, kidney, and non-Hodgkin Lymphoma.

Risk to the General Population: General population exposures to TCE 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 TCE, EPA found those exposure pathways are covered under the jurisdiction of other environmental statutes, administered by EPA, which adequately assess and effectively manage those exposures, i.e., CAA, SDWA, CWA, and RCRA. EPA believes this 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 (U.S. EPA, 2018d).

Risk to Workers: EPA evaluated workers' acute and chronic inhalation and dermal occupational 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 immunosuppression resulting from acute inhalation and dermal exposures, autoimmunity resulting from chronic inhalation and dermal exposures, and cancer resulting from chronic inhalation and dermal exposures. For workers, EPA determined that all applicable conditions of use for TCE presented unreasonable risks. The determinations reflect the severity of the effects associated with the occupational exposures to TCE and incorporate consideration of expected PPE (frequently estimated to be a respirator of APF 10 or 50 and gloves with PF 5 – 20). A full description of EPA's determination for each condition of use is in Section 5.3.

Risk to Occupational Non-Users (ONUs): EPA evaluated ONU acute and chronic inhalation occupational 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 immunosuppression resulting from acute inhalation exposures, autoimmunity resulting from chronic inhalation exposures, and cancer resulting from chronic inhalation exposures. The determinations reflect the severity of the effects associated with the occupational exposures to TCE and the expected absence of PPE for ONUs. For dermal exposures, because ONUs are not expected to be dermally exposed to TCE, dermal risks to ONUs generally 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 directly exposed workers. 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, and determined that most of applicable conditions of use present unreasonable risks. Estimated numbers of occupational non-users are in Section 2.3.1.2.7.

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 determination of unreasonable risk is immunosuppression 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 consumers, EPA determined that consumer conditions of use present unreasonable

risks, except for pepper spray. A full description of EPA's determination for each condition of use is in Section 5.1.

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 is immunosuppression from acute inhalation exposures. 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 TCE, dermal non-cancer risks to bystanders were not identified. For bystanders, EPA determined that consumer conditions of use present unreasonable risks, except for pepper spray. A full description of EPA's determination for each condition of use is in Section 5.1.

Summary of risk determinations:

EPA's preliminary determination regarding environmental risks are summarized above and presented in more detail in Section 5.1.

EPA has preliminarily determined that the following condition of use of TCE does not present an unreasonable risk of injury under any scenarios. The details of this determination are presented in Table 5-1 in Section 5.2.

Conditions of Use that Do Not Present an Unreasonable Risk
• Pepper Spray (consumers and bystanders)

EPA has preliminarily determined that the following conditions of use of TCE present an 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 (including repackaging and loading/unloading)

Processing that Presents an Unreasonable Risk
• Processing as a reactant/intermediate • Incorporation into a formulation, mixture or reaction product (solvents for cleaning or degreasing) • Incorporation into a formulation, mixture or reaction product (adhesives and sealant chemicals) • Incorporation into a formulation, mixture or reaction product (solvents which become part of product formulation or mixture) • Incorporation into articles • Repackaging

- Recycling

1502

Distribution that Presents an Unreasonable Risk

- Distribution

1503

Industrial/Commercial 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
- As a solvent for mold release
- As a lubricant and grease in tap and die fluid
- As a lubricant and grease in penetrating lubricant
- As an adhesive and sealant in solvent-based adhesives and sealants
- As an adhesive and sealant in solvent in tire repair cement/sealer
- As an adhesive and sealant in solvent in mirror edge sealant
- As a functional fluid in heat exchange fluid
- In paints and coatings as a diluent in solvent-based paints and coatings
- In cleaning and furniture care products as carpet cleaner
- In cleaning and furniture care products as wipe cleaning
- In laundry and dishwashing products as spot remover
- In arts, crafts, and hobby materials as fixatives and finishing spray coatings
- As corrosion inhibitors and anti-scaling agents
- As processing aids in process solvent use in battery manufacture
- As processing aids in process solvent used in polymer fiber spinning, fluoroelastomer manufacture and Alcantara manufacture
- As processing aids in extraction solvent used in caprolactam manufacture
- As processing aids in precipitant used in beta-cyclodextrin manufacture
- As ink, toner and colorant products in toner aid
- In automotive care products as brake parts cleaner
- In apparel and footwear care products as shoe polish
- As hoof polish
- As gun scrubber
- As pepper spray
- Other miscellaneous industrial and commercial uses

1504

Disposal that Presents an Unreasonable Risk

- Disposal

1505

Consumer Uses that Present an Unreasonable Risk
• As a solvent in brake and parts cleaner • As a solvent in aerosol electronic degreaser/cleaner • As a solvent in liquid electronic degreaser/cleaner • As a solvent in aerosol spray degreaser/cleaner • As a solvent in liquid degreaser/cleaner • As a solvent in aerosol gun scrubber • As a solvent in liquid gun scrubber • As a solvent in mold release • As a solvent in aerosol tire cleaner • As a solvent in liquid tire cleaner • As a lubricant and grease (tap and die fluid) • As a lubricant and grease (penetrating lubricant) • As an adhesive and sealant (solvent-based adhesive and sealant) • As an adhesive and sealant (mirror edge sealant) • As an adhesive and sealant (tire repair cement/sealer) • As a cleaning and furniture care product (carpet cleaner) • As a cleaning and furniture care product (aerosol spot remover) • As a cleaning and furniture care product (liquid spot remover) • In arts, crafts, and hobby materials as fixative and finishing spray coating • In apparel and footwear products as shoe polish • As fabric spray • As film cleaner • As hoof polish • As toner aid

1506
1507

1 INTRODUCTION

This document presents the draft risk evaluation for trichloroethylene (TCE) under the Frank R. Lautenberg Chemical Safety for the 21st Century Act which amended the Toxic Substances Control Act, the Nation's primary chemicals management law, in June 2016.

The EPA published the scope of the risk evaluation for TCE (U.S. EPA, 2017i) in June 2017, and the problem formulation in May, 2018 (U.S. EPA, 2018d), which represented the analytical phase of risk evaluation in which "the purpose for the assessment is articulated, the problem is defined, and a plan for analyzing and characterizing risk is determined" as described in Section 2.2 of the *Framework for Human Health Risk Assessment to Inform Decision Making*. The problem formulation (U.S. EPA, 2018d) presented three conceptual models and an analysis plan. Based on EPA's analysis of the conditions of use, physical-chemical and fate properties, environmental releases, and exposure pathways, the problem formulation preliminarily concluded that further analysis was necessary for exposure pathways to ecological receptors exposed via surface water along with human workers and consumers. The conclusions of the problem formulation were that no further analysis was necessary in the risk evaluation for sediment, soil and land-applied biosolid pathways leading to exposure to terrestrial and aquatic organisms and for water pathways leading to exposure to terrestrial organisms. Further analysis was not conducted for biosolid, soil and sediment pathways, and for water pathways of exposure to terrestrial organisms, based on a qualitative assessment of the physical-chemical properties and fate of trichloroethylene in the environment and a quantitative comparison of hazards and exposures for aquatic and terrestrial organisms. The qualitative assessment for trichloroethylene is presented in Appendix E. 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 trichloroethylene and has considered the comments specific to trichloroethylene, 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.

The EPA indicated in the analysis plan of the problem formulation that it would review the full study reports obtained for physical and chemical properties, environmental fate properties, environmental hazard and human health hazard studies. For human exposure pathways, the EPA further analyzed inhalation exposures to vapors and mists for workers, occupational non-users consumers, and bystanders. Dermal exposures were analyzed for skin contact with liquids for workers and consumers. For environmental release pathways, the EPA further analyzed surface water exposure to aquatic vertebrates, invertebrates, and plants.

In this draft risk evaluation, Section 1.1 presents the basic physical-chemical characteristics of trichloroethylene, 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 1 provides a discussion and analysis of the exposures, both health and environmental, that can be expected based on the conditions of use for trichloroethylene. Section 3 discusses environmental and health hazards of trichloroethylene. 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, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726 (July 20, 2017)), 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 trichloroethylene. 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* and other methods consistent with section 26 of TSCA (*See* 40 CFR 702.45). As explained in the *Risk Evaluation Rule* (82 FR 33726 (July 20, 2017)), the purpose of peer review is for the independent review of the science underlying the risk assessment. 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* (82 FR 33726 (July 20, 2017)), it is important for peer reviewers to consider how the underlying risk evaluation analyses fit together to produce an integrated risk characterization, which forms the basis of an unreasonable risk determination. EPA 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. For this reason, and consistent with standard Agency practice, the public comment period will precede peer review on this draft risk evaluation. 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 trichloroethylene 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 trichloroethylene. 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.

EPA continues to review the recent court decision in *Safer Chemicals Healthy Families v. EPA*, Nos. 17-72260 et al. (9th Cir. 2019). This draft risk evaluation does not reflect any changes that may occur as a result of that decision. EPA is still seeking public comment on and peer review of this version, however. EPA will communicate the Agency's plans, including the possibility of supplemental versions, in response to the court decision as appropriate.

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

TCE is a colorless liquid with a pleasant, sweet odor resembling that of chloroform. It is considered a volatile organic compound (VOC) because of its moderate boiling point, 87.2°C, and high vapor pressure, 73.46 mm Hg at 25°C. TCE is moderately water soluble (1.280 g/L at 25°C) and has a log octanol/water partition coefficient (Kow) of 2.42. The density of TCE, 1.46 g/m^3 at 20°C, is greater than that of water.

Table 1-1. Physical and Chemical Properties of TCE

Property	Value [a]	References
Molecular Formula	C_2HCl_3	
Molecular Weight	131.39 g/mole	
Physical Form	Colorless, liquid, sweet, pleasant odor, resembles chloroform	(O'Neil et al., 2006)
Melting Point	-84.7°C	(Lide, 2007)
Boiling Point	87.2°C	(Lide, 2007)
Density	1.46 g/cm^3 at 20°C	(ECB, 2000)
Vapor Pressure	73.72 mmHg at 25°C[b]	(Daubert and Danner, 1995)
Vapor Density	4.53	(O'Neil et al., 2006)
Water Solubility	1,280 mg/L at 25°C	(Horvath et al., 1999)
Octanol/Water Partition Coefficient (Log K_{ow})	2.42	(Banerjee et al., 1980)
Henry's Law Constant	9.85E-03 atm·m^3/mole	(Leighton and Calo, 1981)
Flash Point	90°C (closed cup)	(ECB, 2000)
Auto Flammability	410°C (Estimated)	(WHO, 1985)
Viscosity	0.545 mPa·s at 25°C	(Lide, 2007)
Refractive Index	1.4775 at 20°C	(O'Neil et al., 2001)
Dielectric Constant	3.4 ε_0 at 16°C	(Weast and Selby, 1966)

[a] Measured unless otherwise noted
[b] This value was updated based on systematic review re-analysis of original values. The original value of 73.46 mmHg, from (Daubert and Danner, 1989), was used for occupational and consumer modeling of inhalation exposures. The effect of this small difference is expected to be negligible for associated exposure estimates.

1.2 Uses and Production Volume

This section contains use and production volume information for TCE.

1.2.1 Data and Information Sources

The summary of use and production volume information for TCE that is presented below is based on research conducted for the *Problem Formulation Document Trichloroethylene* (EPA-740-R1-7014) and any additional information that was learned since the publication of that document. The previous research was based on reasonably available information, including the *Use and Market Profile for Trichloroethylene,* (EPA-HQ-OPPT-2016-0737-0056), public meetings, and meetings with companies, industry groups, chemical users and other stakeholders to aid in identifying conditions of use and verifying conditions of use identified by the EPA. The information and input received from the public, stakeholder meetings and the additional contacts was incorporated into this section to the extent appropriate. Thus, EPA believes the manufacture, processing, distribution, use and disposal activities constitute the conditions of use within the scope of the risk evaluation for trichloroethylene, based on reasonably available information.

1.2.2 Domestic Manufacture of Trichloroethylene

A life cycle diagram is provided (Figure 1-1) depicting the conditions of use that are within the scope of the risk evaluation during various life cycle stages including manufacturing, processing, use (industrial, commercial, consumer; when distinguishable), distribution and disposal. The information is grouped according to Chemical Data Reporting (CDR) processing codes and use categories (including functional use codes for industrial uses and product categories for industrial, commercial and consumer uses), in combination with other data sources (e.g., published literature and consultation with stakeholders), to provide an overview of conditions of use. The EPA notes that some subcategories of use may be grouped under multiple CDR categories.

For the purposes of this risk evaluation, CDR definitions were used. CDR use categories include the following: "industrial use" means use at a site at which one or more chemicals or mixtures are manufactured (including imported) or processed. "Commercial use" means the use of a chemical or a mixture containing a chemical (including as part of an article) in a commercial enterprise providing saleable goods or services. "Consumer use" means the use of a chemical or a mixture containing a chemical (including as part of an article, such as furniture or clothing) when sold to or made available to consumers for their use (U.S. EPA, 2016d).

To understand conditions of use relative to one another and associated potential exposures under those conditions of use, the life cycle diagram includes the production volume associated with each stage of the life cycle, as reported in the 2016 CDR reporting (U.S. EPA, 2016d) when the volume was not claimed confidential business information (CBI). The 2016 CDR reporting data for TCE are provided in Table 1-2 for TCE from the EPA's CDR database (U.S. EPA, 2016d). For the 2016 CDR reporting period, non-confidential data indicate a total of 13 manufacturers and importers of TCE in the United States.

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

Reporting Year	2012	2013	2014	2015
Total Aggregate Production Volume (lbs)	220,536,812	198,987,532	191,996,578	171,929,400

Reporting Year	2012	2013	2014	2015
[a] The CDR data for the 2016 reporting period is available via ChemView (https://java.epa.gov/chemview). Because of an ongoing CBI substantiation process required by amended TSCA, the CDR data available in the risk evaluation is more specific than currently in ChemView.				

As reported in the Use Document [EPA-HQ-OPPT-2016-0737-0003 (U.S. EPA, 2017c)], as well as in the 2014 TCE risk assessment (U.S. EPA, 2014b), an estimated 83.6% of TCE's annual production volume is used as an intermediate in the manufacture of the hydrofluorocarbon, HFC-134a, an alternative to the refrigerant chlorofluorocarbon, CFC-12. Another 14.7% of TCE production volume is used as a degreasing solvent, leaving approximately 1.7% for other uses. Also reflected in the life cycle diagram is the fact that TCE, as a widely used solvent, has numerous applications across industrial, commercial and consumer settings.

Descriptions of the industrial, commercial and consumer use categories identified from the 2016 CDR and included in the life cycle diagram (Figure 1-1) are summarized below (U.S. EPA, 2016d). The descriptions provide a brief overview of the use category; the [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500)]* contains more detailed descriptions (e.g., process descriptions, worker activities, process flow diagrams, equipment illustrations) for each manufacture, processing, use and disposal category. The descriptions provided below are primarily based on the corresponding industrial function category and/or commercial and consumer product category descriptions from the 2016 CDR and can be found in the EPA's Instructions for Reporting 2016 TSCA Chemical Data Reporting (U.S. EPA, 2016b).

The following describes several industrial/commercial CDR use categories where TCE has been used; the [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500)]* provides additional process-related information on the remaining categories and life cycle stages.

The **"Solvents for Cleaning and Degreasing"** category encompasses chemical substances used to dissolve oils, greases and similar materials from a variety of substrates including metal surfaces, glassware and textiles. This category includes the use of TCE in vapor degreasing, cold cleaning and in industrial and commercial aerosol degreasing products.

The **"Lubricants and Greases"** category encompasses chemical substances contained in products used to reduce friction, heat generation and wear between solid surfaces. This category includes the use of TCE in penetrating lubricants, and tap and die fluids for industrial, commercial and consumer uses.

The **"Adhesives and Sealants"** category encompasses chemical substances contained in adhesive and sealant products used to fasten other materials together. This category includes the use of TCE in mirror-edge sealants and other adhesive products.

The "**Functional Fluids (closed system)"** category encompasses liquid or gaseous chemical substances used for one or more operational properties in a closed system. Examples are heat transfer agents (e.g., coolants and refrigerants).

The **"Paints and Coatings"** category encompasses chemical substances contained in paints, lacquers, varnishes and other coating products that are applied as a thin continuous layer to a surface. Coating may provide protection to surfaces from a variety of effects such as corrosion and ultraviolet (UV) degradation; may be purely decorative; or may provide other functions. The EPA anticipates that the

primary subcategory to be the use of TCE in solvent-based coatings. This category covers industrial, commercial and consumer uses of paints and coatings.

The **"Cleaning and Furniture Care Products"** category encompasses chemical substances contained in products that are used to remove dirt, grease, stains and foreign matter from furniture and furnishings, or to cleanse, sanitize, bleach, scour, polish, protect or improve the appearance of surfaces. This category includes the use of TCE for spot cleaning and carpet cleaning.

The **"Laundry and Dishwashing Products"** category encompasses chemical substances contained in laundry and dishwashing products and aids formulated as a liquid, granular, powder, gel, cakes, and flakes that are intended for consumer or commercial use.

The **"Arts, Crafts and Hobby Materials"** category encompasses chemical substances contained in arts, crafts, and hobby materials that are intended for consumer or commercial use.

1.3 Regulatory and Assessment History

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

Federal Laws and Regulations

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

State Laws and Regulations

TCE 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.2

Laws and Regulations in Other Countries and International Treaties or Agreements

TCE is subject to statutes or regulations in countries other than the United States and/or international treaties and/or agreements. A summary of these laws, regulations, treaties and/or agreements is provided in Appendix A.3

The EPA has identified assessments conducted by other agency programs and organizations (see Table 1-3). Depending on the source, these assessments may include information on conditions of use, hazards, exposures, and potentially exposed or susceptible subpopulations (PESS)—information useful to the EPA in preparing this risk evaluation. Table 1-3 shows the assessments that have been conducted. In addition to using this information, EPA conducted a full review of the data collected [see *Trichloroethylene (CASRN 79-01-6) Bibliography: Supplemental File for the TSCA Scope Document,* EPA-HQ-OPPT-2016-0737) using the literature search strategy (see *Strategy for Conducting Literature Searches for Trichloroethylene: Supplemental File for the TSCA Scope Document,* EPA-HQ-OPPT-2016-0737] to ensure that the EPA is considering information that has been made available since these assessments were conducted.

In its previous TCE Risk Assessment (U.S. EPA, 2014b), risks from use of TCE in commercial and consumer solvent degreasing (aerosol and vapor), consumer use as a spray-applied protective coating for arts and crafts and commercial use as a spot remover at dry-cleaning facilities were assessed. The TCE Risk Assessment was used to support two proposed rules under TSCA section 6 (81 FR 91592;

December 12, 2016; 82 FR 7432; January 19, 2017) to address risks from use of TCE. Along with other reasonably available information, the EPA used the existing TSCA risk assessments to inform its development of the TCE risk evaluation.

Table 1-3. Assessment History of TCE

Authoring Organization	Assessment
EPA Assessments	
Office of Chemical Safety and Pollution Prevention (OCSPP)/ Office of Pollution Prevention and Toxics (OPPT)	TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Use (U.S. EPA, 2014b)
OCSPP/OPPT	Supplemental Occupational Exposure and Risk Reduction Technical Report in Support of Risk Management Options for Trichloroethylene (TCE) Use in Aerosol Degreasing (U.S. EPA, 2016f)
OCSPP/OPPT	Supplemental Exposure and Risk Reduction Technical Report in Support of Risk Management Options for Trichloroethylene (TCE) Use in Consumer Aerosol Degreasing (U.S. EPA, 2016e)
OCSPP/OPPT	Supplemental Occupational Exposure and Risk Reduction Technical Report in Support of Risk Management Options for Trichloroethylene (TCE) Use in Spot Cleaning (U.S. EPA, 2016g)
OCSPP/OPPT	Supplemental Occupational Exposure and Risk Reduction Technical Report in Support of Risk Management Options for Trichloroethylene (TCE) Use in Vapor Degreasing [RIN 2070-AK11] (U.S. EPA, 2016h)
Integrated Risk Information System (IRIS)	Toxicological Review of Trichloroethylene (U.S. EPA, 2011e)
National Center for Environmental Assessment (NCEA)	Sources, Emission and Exposure for Trichloroethylene (TCE) and Related Chemicals (U.S. EPA, 2001)
Office of Water (OW)/ Office of Science and Technology (OST)	Update of Human Health Ambient Water Quality Criteria: Trichloroethylene (TCE) 79-01-6 (U.S. EPA, 2015b)
Other U.S.-Based Organizations	
Agency for Toxic Substances and Disease Registries (ATSDR)	Final Toxicological Profile for Trichloroethylene (ATSDR, 2019)
National Research Council (NRC)	Assessing the Human Health Risks of Trichloroethylene: Key Scientific Issues (NRC, 2006)
Office of Environmental Health Hazard Assessment (OEHHA), Pesticide and Environmental Toxicology Section	Public Heath Goal for Trichloroethylene in Drinking Water (CalEPA, 2009)
International	

Authoring Organization	Assessment
Institute for Health and Consumer Protection, European Chemicals Bureau	European Union Risk Assessment Report, Trichloroethylene (ECB, 2004)
Australia National Industrial Chemicals Notification and Assessment Scheme (NICNAS)	Trichloroethylene: Priority Existing Chemical Assessment Report No. 8 (NICNAS, 2000)

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 and Table 1-5. No additional information was received by the EPA following the publication of the problem formulation (U.S. EPA, 2018d) that would update or otherwise require changes to the life cycle diagram (Figure 1-1) as presented in the problem formulation (U.S. EPA, 2018d). Nonetheless, EPA decided to reorganize the conditions of use for this risk evaluation. In this risk evaluation, the COUs as described in (U.S. EPA, 2018d) were evaluated for occupational scenarios based on corresponding occupational exposure scenarios (OES) (Table 1-4). The occupational COUs are also applicable to environmental receptors based on water releases from these activities.

“Lace wig and hair extension glues” have been eliminated as a COU since the publication of the problem formulation (U.S. EPA, 2018d). EPA, after consultation with the FDA, has determined that this use, previously identified in the problem formulation as a conditions of use, is not a condition of use because it falls outside the scope of EPA’s jurisdiction. TSCA sec. 3(2) excludes from the definition of “chemical substance” cosmetics as they are defined in the Federal Food, Drug and Cosmetic Act (FFDCA) when manufactured, processed, or distributed in commerce for use as a cosmetic. Because the glue for lace wigs and hair extensions is a cosmetic within section 201(i) of the FFDCA, any TCE used for these purposes is exempted from TSCA.

Consumer scenarios were evaluated separately from occupational scenarios, and EPA re-categorized certain COUs based on product function. None of these changes resulted in any difference in how these products are or would have been assessed, they simply reflect a recategorization in order to improve clarity. Additionally, subcategories were added based on availability of differing forms of a product (e.g., aerosol vs liquid). The updated consumer conditions of use and explanations for the changes are presented in Table 1-5.

1783 **Table 1-4. Categories and Subcategories of Occupational Conditions of Use and Corresponding Occupational Exposure Scenario**

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
Manufacture	Domestic manufacture	Domestic manufacture	Manufacturing	(U.S. EPA, 2016d)
	Import	Import	Repackaging	(U.S. EPA, 2016d)
Processing	Processing as a reactant/ intermediate	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants, foam blowing agents and solvents)	Processing as a reactant	(U.S. EPA, 2016d); EPA-HQ-OPPT-2016-0737-0013; EPA-HQ-OPPT-2016-0737-0013; EPA-HQ-OPPT-2016-0737-0026; EPA-HQ-OPPT-2016-0737-0027
	Processing - Incorporation into formulation, mixture or reaction product	Solvents (for cleaning or degreasing)	Formulation of Aerosol and Non-Aerosol Products	(U.S. EPA, 2016d)
		Adhesives and sealant chemicals		(U.S. EPA, 2016d)
		Solvents (which become part of product formulation or mixture) (e.g., lubricants and greases, paints and coatings, other uses)		(U.S. EPA, 2016d); EPA-HQ-OPPT-2016-0737-0003; EPA-HQ-OPPT-2016-0737-0056
	Processing – incorporated into articles	Solvents (becomes an integral components of articles)		(U.S. EPA, 2016d)
	Repackaging	Solvents (for cleaning or degreasing)	Repackaging	(U.S. EPA, 2016d)
	Recycling	Recycling	Process Solvent Recycling and Worker Handling of Wastes	(U.S. EPA, 2017f)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
Distribution in commerce	Distribution	Distribution	Not assessed as a separate operation; exposures/releases from distribution are considered within each condition of use.	EPA-HQ-OPPT-2016-0737-0003
Industrial/commercial use	Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop) [c]	Batch Open-Top Vapor Degreasing; Batch Closed-Loop Vapor Degreasing	EPA-HQ-OPPT-2016-0737-0003, (U.S. EPA, 2014b), (U.S. EPA, 2016h), EPA-HQ-OPPT-2016-0737-0056
		In-line vapor degreaser (e.g., conveyorized, web cleaner) [c]	Conveyorized Vapor Degreasing; Web Vapor Degreasing	EPA-HQ-OPPT-2016-0737-0003, (U.S. EPA, 2014b), (U.S. EPA, 2016h), EPA-HQ-OPPT-2016-0737-0056
		Cold cleaner	Cold Cleaning	EPA-HQ-OPPT-2016-0737-0003; (U.S. EPA, 2017h); EPA-HQ-OPPT-2016-0737-0056
		Aerosol spray degreaser/ cleaner [c]	Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	EPA-HQ-OPPT-2016-0737-0003, (U.S. EPA, 2014b), (U.S. EPA, 2016f), (U.S. EPA, 2016e), EPA-HQ-OPPT-2016-0737-0056
		Mold release		EPA-HQ-OPPT-2016-0737-0003; EPA-HQ-OPPT-2016-0737-0056

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
	Lubricants and greases/lubricants and lubricant additives	Tap and die fluid	Metalworking Fluids	(U.S. EPA, 2016d); EPA-HQ-OPPT-2016-0737-0003; EPA-HQ-OPPT-2016-0737-0028, EPA-HQ-OPPT-2016-0737-0056
		Penetrating lubricant	Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases; Metalworking Fluids	(U.S. EPA, 2016d), EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003; EPA-HQ-OPPT-2016-0737-0028
	Adhesives and sealants	Solvent-based adhesives and sealants	Adhesives, Sealants, Paints, and Coatings	(U.S. EPA, 2016d), EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Tire repair cement/sealer		(U.S. EPA, 2016d), EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Mirror edge sealant		EPA-HQ-OPPT-2016-0737-0003; (U.S. EPA, 2014b), EPA-HQ-OPPT-2016-0737-0056
	Functional fluids (closed systems)	Heat exchange fluid	Other Industrial Uses	(U.S. EPA, 2017h)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
	Paints and coatings	Diluent in solvent-based paints and coatings	Adhesives, Sealants, Paints, and Coatings	(U.S. EPA, 2016d), EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003; EPA-HQ-OPPT-2016-0737-0010; EPA-HQ-OPPT-2016-0737-0015; EPA-HQ-OPPT-2016-0737-0027;
	Cleaning and furniture care products	Carpet cleaner	Spot Cleaning, Wipe Cleaning and Carpet Cleaning	EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Wipe cleaning [d]		EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
	Laundry and dishwashing products	Spot remover [c]		EPA-HQ-OPPT-2016-0737-0003, (U.S. EPA, 2014b), (U.S. EPA, 2016g), EPA-HQ-OPPT-2016-0737-0056
	Arts, crafts and hobby materials	Fixatives and finishing spray coatings [c]	Adhesives, Sealants, Paints, and Coatings	(U.S. EPA, 2014b)
	Corrosion inhibitors and anti-scaling agents	Corrosion inhibitors and anti-scaling agents	Industrial Processing Aid	(U.S. EPA, 2016d)
	Processing aids	Process solvent used in battery manufacture		(U.S. EPA, 2017h)
		Process solvent used in polymer fiber spinning, fluoroelastomer manufacture and Alcantara manufacture		(U.S. EPA, 2017h)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
		Extraction solvent used in caprolactam manufacture		(U.S. EPA, 2017h)
		Precipitant used in beta-cyclodextrin manufacture		(U.S. EPA, 2017h)
	Ink, toner and colorant products	Toner aid	Commercial Printing and Copying	EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
	Automotive care products	Brake and parts cleaner	Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
	Apparel and footwear care products	Shoe polish	Other Commercial Uses	(U.S. EPA, 2017h)
	Other uses	Hoof polishes [e]		EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Pepper spray		EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Gun scrubber		EPA-HQ-OPPT-2016-0737-0056; EPA-HQ-OPPT-2016-0737-0003
		Other miscellaneous industrial and commercial uses		(U.S. EPA, 2017h)
Disposal	Disposal	Industrial pre-treatment		(U.S. EPA, 2017f)

Life Cycle Stage	Category [a]	Subcategory [b]	Occupational Exposure Scenario (OES)	References
		Industrial wastewater treatment	Process Solvent Recycling and Worker Handling of Wastes	
		Publicly owned treatment works (POTW)		

[a] These categories of conditions of use appear in the Life Cycle Diagram, reflect CDR codes, and broadly represent conditions of use of TCE in industrial and/or commercial settings.
[b] These subcategories reflect more specific uses of TCE.
[c] This includes uses assessed in the (U.S. EPA, 2014b) risk assessment.
[d] This condition of use involves wipe cleaning. Note that the problem formulation described "cleaning wipes" as a condition of use. This referred to the application of a product that is then wiped off, rather than a pre-wet towelette.
[e] "Hoof polish" would remain within EPA's jurisdiction unless the article in question was also *intended for the diagnosis, cure, mitigation, treatment, of disease or intended to affect the structure or function of the body of animals*, as described in the FFDCA. EPA identified a single product for hoof polish containing TCE (U.S. EPA, 2017h), and this product is intended for only cosmetic and not medical use. Therefore, "hoof polish" was evaluated as a COU, applicable only to products restricted to cosmetic function.

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Life Cycle Stage	Category	Subcategory
Use	Solvents for Cleaning and Degreasing	Brake & Parts Cleaner[2]
		Aerosol Electronic Degreaser/Cleaner[1]
		Liquid Electronic Degreaser/Cleaner[1]
		Aerosol Spray Degreaser/Cleaner[1]
		Liquid Degreaser/Cleaner[1]
		Aerosol Gun Scrubber[1,3]
		Liquid Gun Scrubber[1,3]
		Mold Release
		Aerosol Tire Cleaner[1,4]
		Liquid Tire Cleaner[1,4]
	Lubricants and Greases	Tap & Die Fluid
		Penetrating Lubricant[5]
	Adhesives and Sealants	Solvent-based Adhesive & Sealant
		Mirror-edge Sealant
		Tire Repair Cement/Sealer
	Cleaning and Furniture Care Products[10]	Carpet Cleaner
		Aerosol Spot Remover[1,6]
		Liquid Spot Remover[1,6]
	Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings[7]
	Apparel and Footwear Care Products	Shoe Polish
	Other Consumer Uses	Fabric Spray[8]
		Film Cleaner
		Hoof Polish
		Pepper Spray
		Toner Aid[9]

Life Cycle Stage	Category	Subcategory

[1] Form was determined based on the specific products identified as representative of the associated product subcategories. Distinct subcategories based on differing forms (aerosol and liquid) were not specifically defined in the Problem Formulation. They were added due to product availability based on additional research that helped to differentiate specific product forms (i.e., liquid or aerosol) and types.
[2] The brake cleaner subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the automotive care products category; however, the same brake cleaning conditions of use are now associated with the broader solvents for cleaning and degreasing category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the brake cleaner product(s) and not a broader category of use.
[3] The gun scrubber subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the other consumer uses category; however, the same gun scrubber conditions of use are now associated with the broader solvents for cleaning and degreasing category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the gun scrubber product(s) and not a broader category of use.
[4] Tire cleaner products / subcategories of use were not specifically called out in the Problem Formulation; however, such products were identified in the 2017 Use and Market Report (U.S. EPA, 2017f) and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: TCE (U.S. EPA, 2017c) and fit within the broader Solvents for Cleaning and Degreasing category.
[5] Based on additional research into the specific product(s) associated with the broader lubricants and greases category, the subcategory name was updated from penetrating lubricant to lubricant.
[6] The spot remover subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the laundry and dishwashing products category; however, the same spot remover conditions of use are now associated with the cleaning and furniture care products category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the spot remover product(s) and not a broader category of use.
[7] Note that this subcategory is referred to as "clear protective coating spray" in U.S. EPA (2014b) and as "spray fixative" in the TCE Significant New Use Rule (80 FR 47441).
[8] Fabric spray (specifically an anti-fray spray) was added following Problem Formulation based on identification in the final 2014 TCE Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).
[9] The toner aid subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the Ink, toner, and colorant products category; however, the toner aid use is not like use of a toner or pigment; therefore, the same toner aid condition of use is now associated with the other consumer use category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the toner aid product(s) and not a broader category of use.
[10] Note that the problem formulation described "cleaning wipes" as a condition of use for this category. However, that referred to the application of a product that is then wiped off, rather than a pre-wet towelette. A number of consumer conditions of use involve wipe cleaning and are described in detail in Section 2.3.2.6.2 as leading to dermal contact with impeded evaporation.

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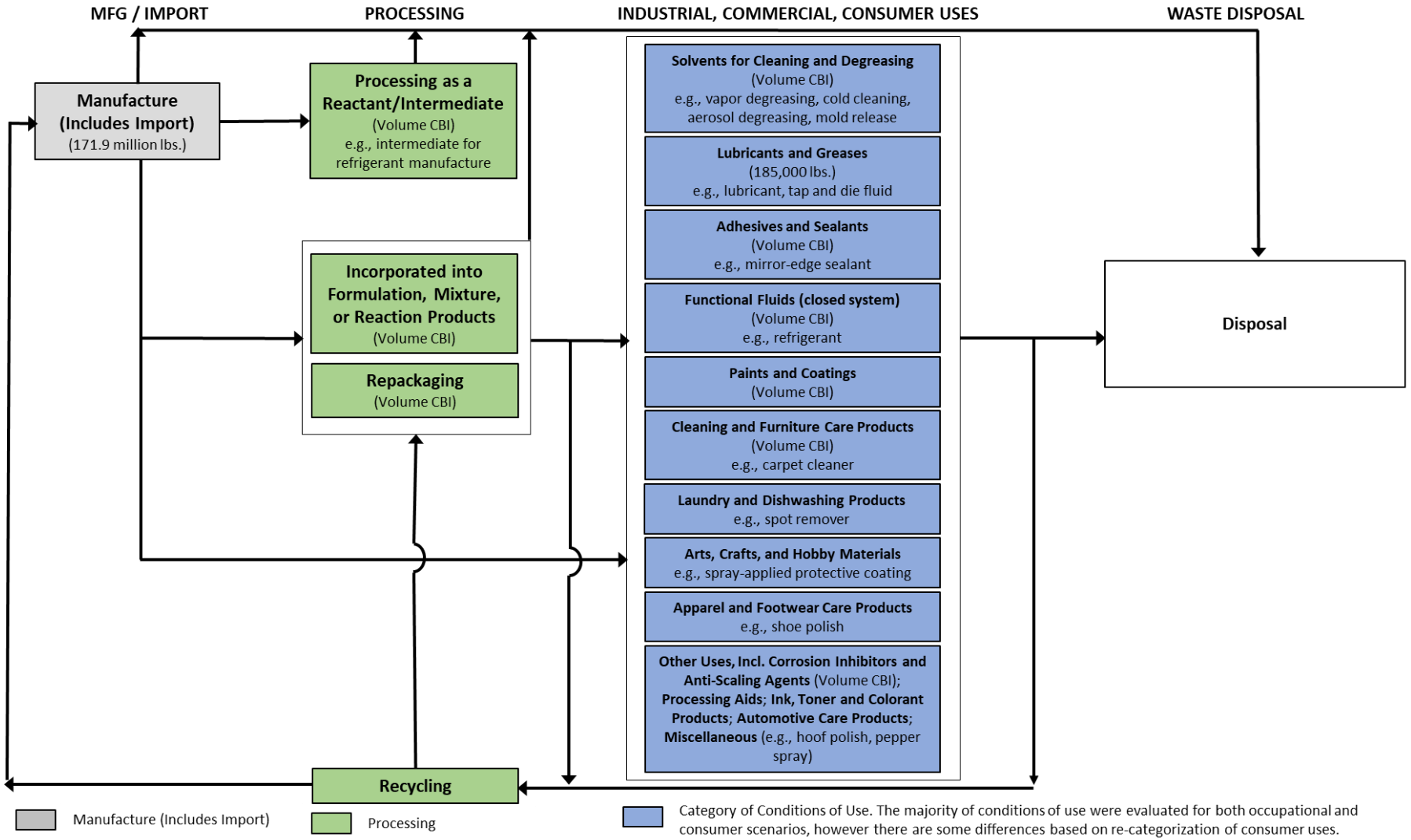


Figure 1-1. TCE Life Cycle Diagram

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

1.4.2 Conceptual Models

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

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, exposure to organisms through the sediment compartment, and exposure to terrestrial organisms. In the problem formulation, the EPA determined that no further evaluation of these pathways is needed due to the physical/chemical properties associated with TCE (high vapor pressure) and its rapid volatilization to air from soil and water or rapid migration through soil into groundwater. Due to TCE's fate properties, a significant portion of TCE would not be available to enter the sediment compartment.

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

- Exposure to aquatic species (i.e. aquatic plants) via contaminated surface water.
- Inhalation and dermal exposures to workers and consumers, and inhalation exposures to ONUs and 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 TCE confirmed the preliminary conclusions in the problem formulation (U.S. EPA, 2018d) and as a result, the EPA confirms further analysis of the pathways outlined in the conceptual models. The conceptual models from the problem formulation are shown below in Figure 1-2, Figure **1-3**, and Figure **1-4**.

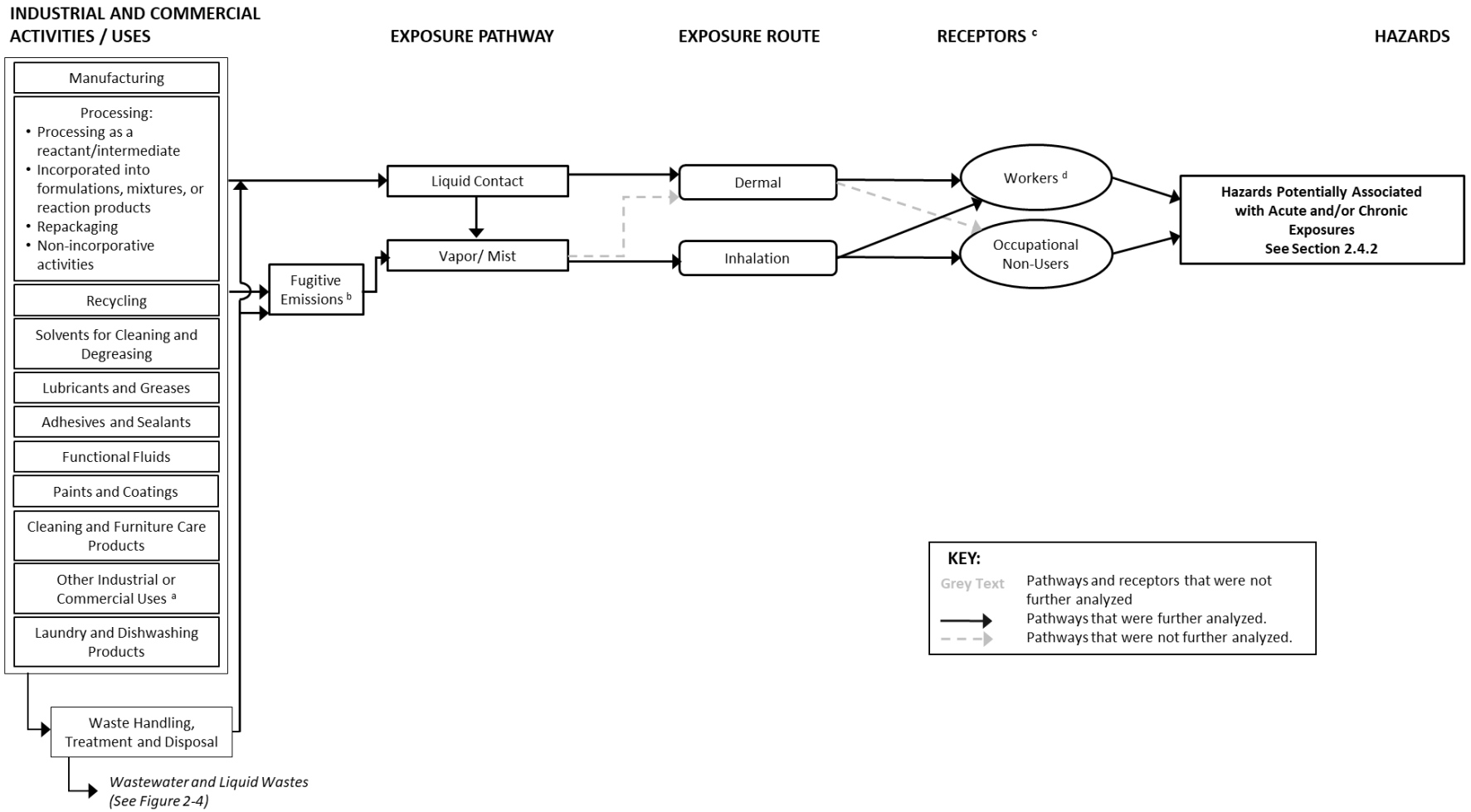


Figure 1-2. TCE Conceptual Model for Industrial and Commercial Activities and Uses: Potential Exposures and Hazards

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

[a] Some products are used in both commercial and consumer applications. Additional uses of TCE 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 (PESS) including women of childbearing age and their children and genetically susceptible populations.

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

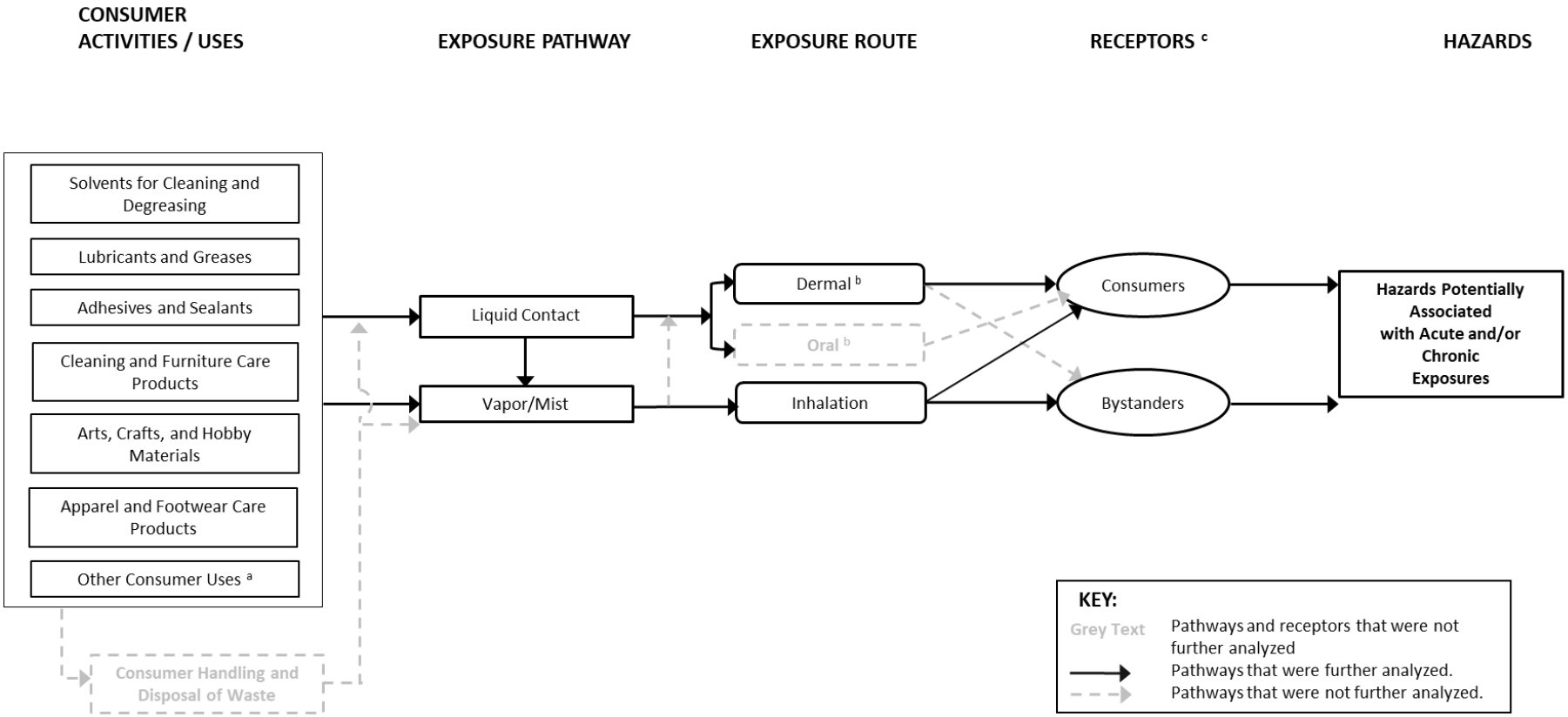


Figure 1-3. TCE Conceptual Model for Consumer Activities and Uses: Potential Exposures and Hazards

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

[a] Some products are used in both commercial and consumer applications. Additional uses of TCE are included in Table 1-4.

[b] Exposure may occur through mists that deposit in the upper respiratory tract however, based on physical chemical properties, mists of TCE will likely be rapidly absorbed in the respiratory tract or evaporate and not result in an oral exposure. Although less likely given the physical-chemical properties, oral exposure may also occur from incidental ingestion of residue on hand/body.

[c] Receptors include Potentially Exposed or Susceptible Subpopulations (PESS).

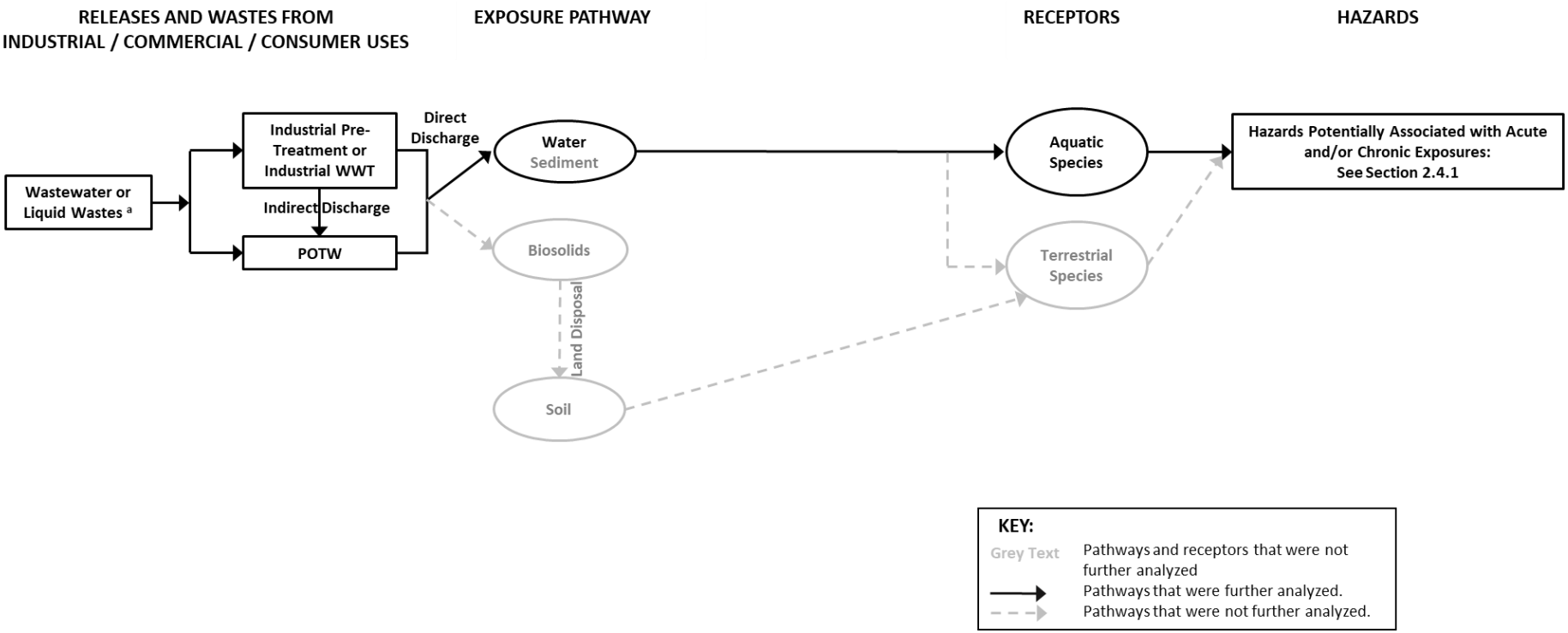


Figure 1-4. TCE Conceptual Model for Environmental Releases and Wastes: Potential Exposures and Hazards

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

[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 the 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, 2018b). The process complements the risk evaluation process in that the data collection, data evaluation and data integration stages of the systematic review process are used to develop the exposure and hazard assessments based on reasonably available information. EPA defines "reasonably available information" to mean information that EPA possesses, or can reasonably obtain and synthesize for use in risk evaluations, considering the deadlines for completing the evaluation (40 CFR 702.33).

EPA is implementing systematic review methods and approaches within the regulatory context of the amended TSCA. Although EPA 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 aggressive 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; engineering releases and occupational exposure; consumers and environmental exposure; and environmental and human health hazard). EPA then developed and applied inclusion and exclusion criteria during the title and abstract screening to identify information potentially relevant for the risk evaluation process. The literature and screening strategy as specifically applied to TCE is described in the *Strategy for Conducting Literature Searches for Trichloroethylene (TCE): Supplemental File for the TSCA Scope Document* (U.S. EPA, 2017e) and the results of the title and abstract screening process were published in the [*Trichloroethylene (CASRN 79-01-6) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA, 2017i)].

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

[4] 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.

Although EPA conducted a comprehensive search and screening process as described above, EPA made the decision to leverage the literature published in previous assessments[5] when identifying relevant key and supporting data[6] and information for developing the TCE risk evaluation. This is discussed in the *Strategy for Conducting Literature Searches for Trichloroethylene: Supplemental Document to the TSCA Scope Document* (U.S. EPA, 2017e). In general, many of the key and supporting data sources were identified in the comprehensive *Trichloroethylene (CASRN 79-01-6) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA, 2017i). However, there were instances that 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 the *Application of Systematic Review for TSCA Risk Evaluations* (U.S. EPA, 2018b). Other relevant key and supporting references were identified through targeted supplemental searches to support the analytical approaches and methods in the trichloroethylene risk evaluation (e.g., to locate specific information for exposure modeling) or to identify new data and information published after the date limits of the initial search.

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 the *Strategy for Conducting Literature Searches for Trichloroethylene: Supplemental Document to the TSCA Scope Document* (U.S. EPA, 2017e). 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. All other literature from previous authoritative assessments were considered as supplemental information. A 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 considering the deadlines specified in TSCA section 6(b)(4)(G) for completing such evaluation for most chemical substances especially those that have a data rich database such as TCE. Furthermore, EPA evaluated how EPA's evaluation of the key and supporting data and information and newer information would change the previous conclusions presented in the previous assessments.

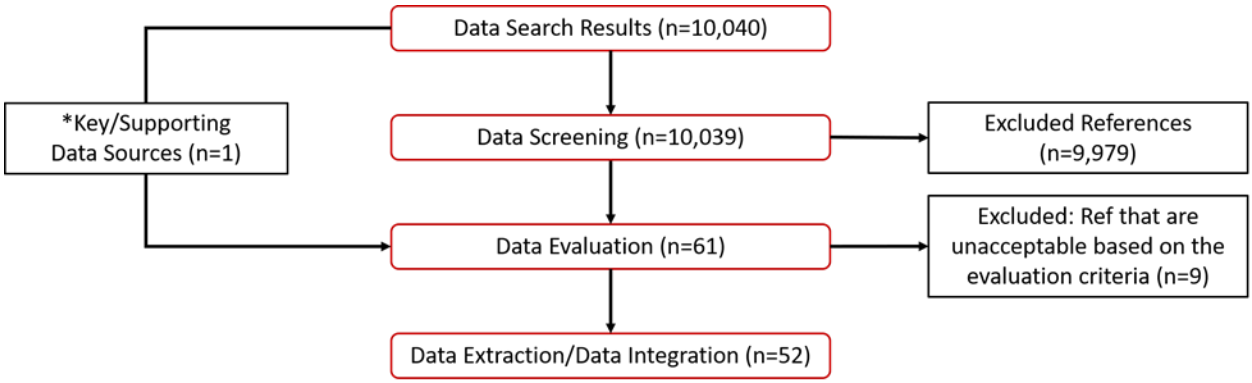
This pragmatic approach allowed EPA to maximize the scientific and analytical efforts of other regulatory and non-regulatory agencies by accepting for the most part the relevant scientific knowledge gathered and analyzed by others except for influential information sources that may have an impact on 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.

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

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

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

EPA made the decision to bypass the data screening step for data sources that were highly relevant to the 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 engineering environmental releases and occupational exposure data sources that were subject to a combined data extraction and evaluation step (Figure 1-6).



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

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

Note: Literature search results for the environmental fate and transport of TCE yielded 10,040 studies. During problem formulation, following data screening, most environmental exposure pathways were removed from the conceptual models. As a result, 9,979 studies were deemed off-topic and excluded. One key source (U.S. EPA, 2012b) and the remaining 61 studies related to environmental exposure pathways retained in the conceptual models entered data evaluation, where 9 studies were deemed unacceptable and 52 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, [*Data Quality Evaluation of Physical-Chemical Properties Studies. Docket: EPA-HQ-OPPT-2019-0500*] and the extracted data are presented in Table 1-1.

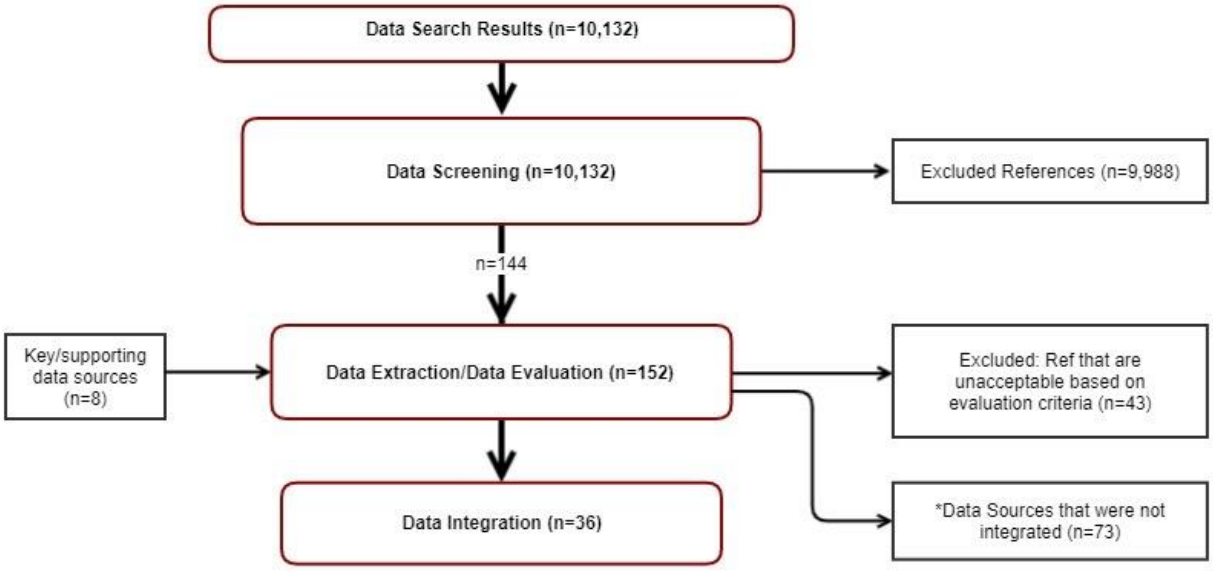
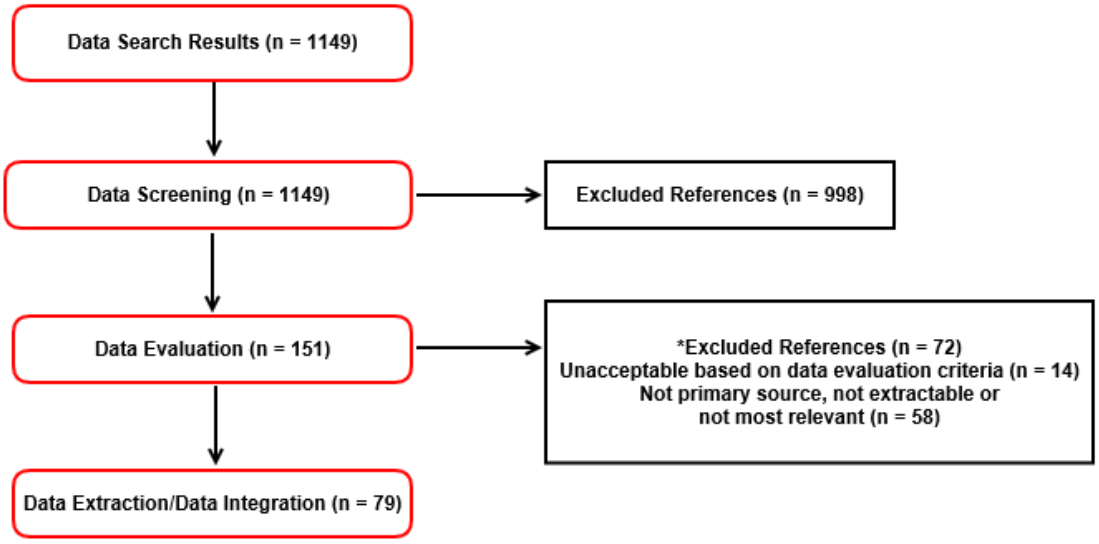


Figure 1-6. Literature Flow Diagram for Engineering Releases and Occupational Exposure

Note: Literature search results for environmental release and occupational exposure yielded 10,132 data sources. Of these data sources, 159 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 8 relevant data sources that bypassed the data screening step [*List of Key and Supporting Studies for Environmental Releases and Occupational Exposure. Docket: EPA-HQ-OPPT-2019-0500)*] 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, 2018b). Of the 152 sources from which data were extracted and evaluated, 43 sources only contained data that were rated as unacceptable based on serious flaws detected during the evaluation. Of the 124 sources forwarded for data integration, data from 36 sources were integrated, and 73 sources contained data that were not integrated (e.g., lower quality data that were not needed due to the existence of higher quality data, data for release media that were removed from scope after data collection).

*The quality of data in these sources (n=73) 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.



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

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

Note: EPA conducted a literature search to determine relevant data sources for assessing exposures for trichloroethylene within the scope of the risk evaluation. This search identified 1149 data sources including relevant supplemental documents. Of these, 998 were excluded during the screening of the title, abstract, and/or full text and 151 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, 79 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.

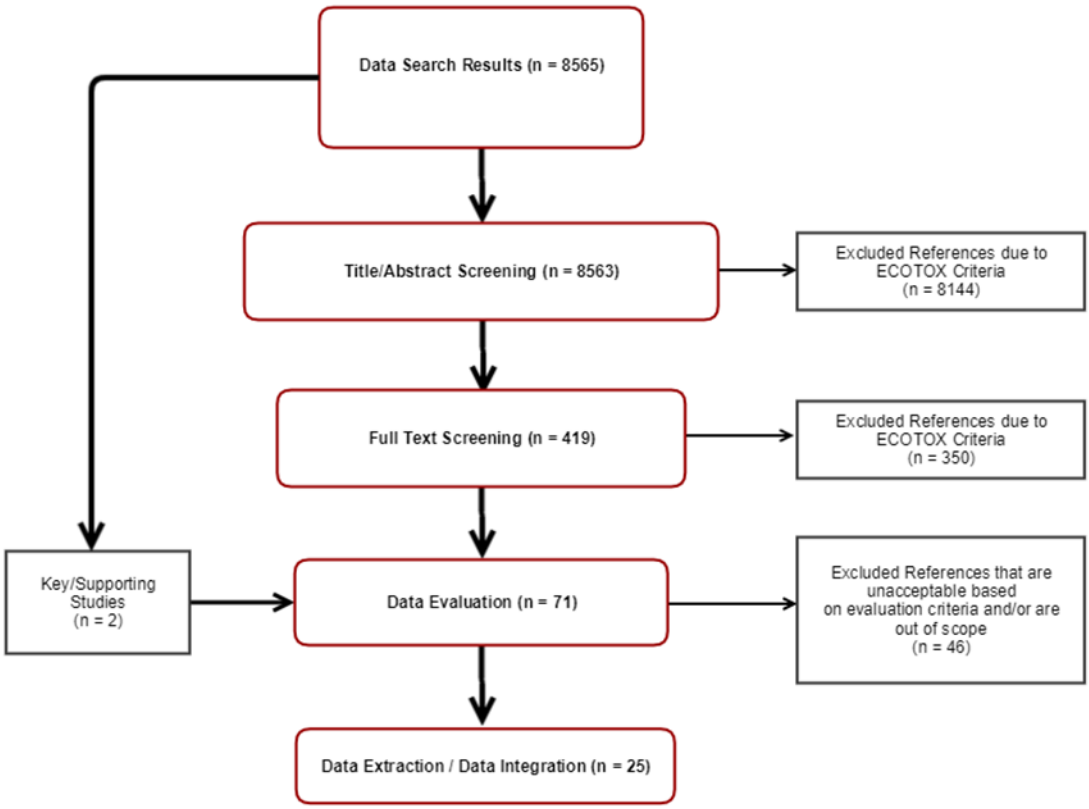


Figure 1-8. Literature Flow Diagram for Environmental Hazard

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

The "Key/Supporting Studies" box represents data sources cited in an existing assessment (Environment Canada and Health Canada, 1993) that were considered highly relevant for the TSCA risk evaluation because they were used as key and supporting information by another regulatory organization to support their chemical hazard and risk assessment. These citations were found independently from the ECOTOX process. These studies bypassed the data screening step and moved directly to the data evaluation step. These two studies were ultimately excluded because they examined hazard to terrestrial species and the relevant exposure pathway of air releases has since been determined to be out of scope.

The literature search process for environmental hazard data found 8,565 citations for TCE. At the title and abstract screening phase, 8,144 citations were excluded as off-topic using ECOTOXicology knowledgebase criteria. The remaining 419 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 71 citations that went to data evaluation for TCE, which included the above-mentioned two additional citations gathered from (Environment Canada and Health Canada, 1993) that were later excluded as out of scope. EPA analyzed each of these studies using the DQE results to determine overall study quality. Twenty-five studies were considered acceptable and were rated high, medium, or low quality during this analysis. The extracted data from these 25 studies were used during data integration for TCE.

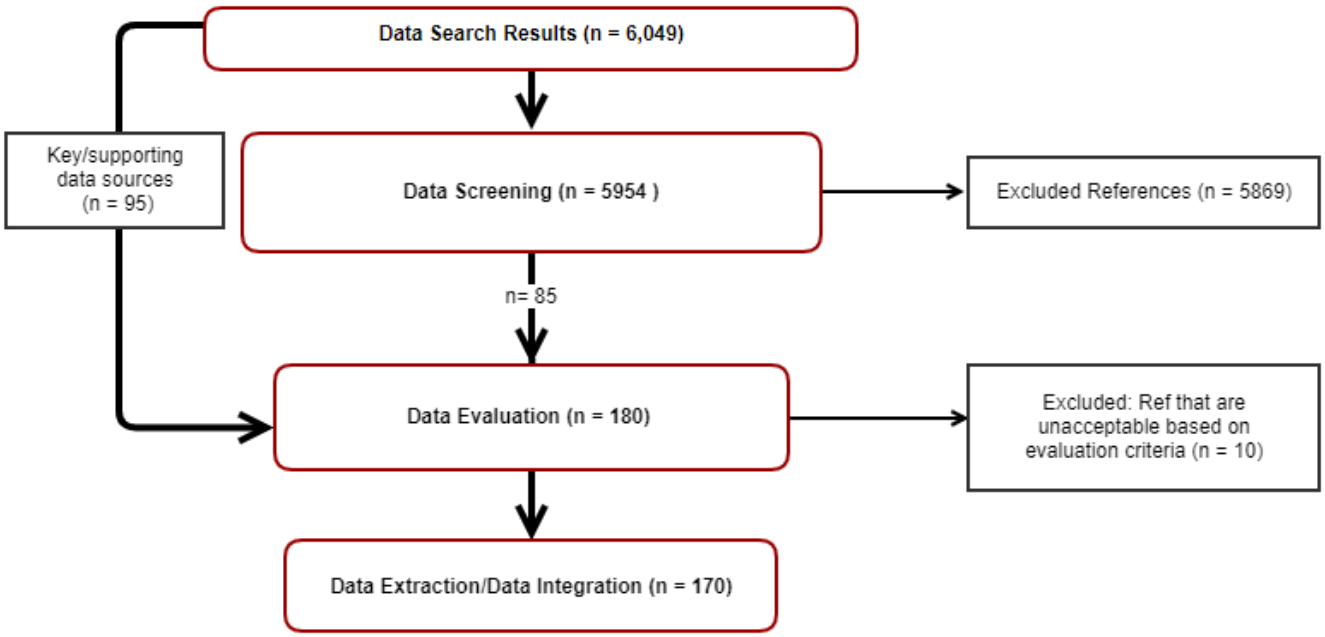


Figure 1-9. Literature Flow Diagram for Human Health Hazard

Note: The literature search results for human health hazard of TCE yielded 6,049 studies. This included 95 key and supporting studies identified from previous EPA assessments. Of the 5,954 new studies screened for relevance, 5,869 were excluded as off topic. The remaining 85 new studies together with the 95 key and supporting studies entered data evaluation. Ten studies were deemed unacceptable based on the evaluation criteria for human health hazard data sources and the remaining 170 studies were carried forward to data extraction/data integration. Additional details can be found in the *Strategy for Conducting Literature Searches for Trichloroethylene Supplemental Document to the TSCA Scope Document* (U.S. EPA, 2017e).

The "Key/Supporting Studies" box represents data sources cited in an existing assessment (U.S. EPA, 2011e) that were considered highly relevant for the TSCA risk evaluation because they were used as key and supporting information by another regulatory organization to support their chemical hazard and risk assessment. For a list of the key and supporting studies, see *[List of Key and Supporting Studies for Human Health Hazard. Docket # EPA-HQ-OPPT-2019-0500]*.

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 TCE study reports identified in [*Trichloroethylene (CASRN 79-01-6) Bibliography: Supplemental File for the TSCA Scope Document;* (U.S. EPA, 2017i)], 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 2.2.2 (Releases to the Environment), Section 2.2.6 (Environmental Exposures), Section 2.3 (Human Exposures), Section 3.1 (Environmental Hazards) and Section 3.2 (Human Health Hazards). Supplemental files[7] also provide details of the data evaluations including individual metric scores and the overall study score for each data source (Docket: EPA-HQ-OPPT-2019-0500).

[7] See Appendix B for the list of all supplemental files.

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 (*Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726).

EPA used previous assessments (see Table 1-3) to identify key and supporting information and then analyzed and synthesized available evidence regarding TCE'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 (Section 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

For TSCA exposure assessments, EPA evaluated exposures and releases to the environment resulting from the conditions of use applicable to TCE. Post-release pathways and routes were described to characterize the relationship or connection between the conditions of use for TCE (Section 1.4.1) and the exposure to human receptors, including potentially exposed or susceptible subpopulations (PESS) and ecological receptors. EPA considered, where relevant, the duration, intensity (concentration), frequency and number of exposures in characterizing exposures to TCE.

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 expects to consider in the risk evaluation. Table 2-1 presents environmental fate data that EPA identified and considered in the Scoping and Problem Formulation documents as well as additional data extracted form the systematic review process.

Table 2-1 Environmental Fate Characteristic of TCE

Property or Endpoint	Value [a]	References	Data Quality Rating
Indirect photodegradation	1-11 days (atmospheric oxidation based on measured hydroxyl radical oxidation)	(U.S. EPA, 2014b)	High
Hydrolysis half-life	10.7 months (average; decomposition in aerated water in the dark; part of the reaction may have occurred in the vapor phase)	(Dilling et al., 1975)	High
Biodegradation	38.9% after 28 days (aerobic OECD 302B Inherent biodegradability test)	(Tobajas et al., 2016)	High
	100% degradation after 60 days (anaerobic serum bottle test)	(Long et al., 1993)	High
	100% degradation after 40 days (anaerobic groundwater microcosms with added hydrogen/acetate)	(Schmidt and Tiehm, 2008)	High
	TCE removed slowly with a reduction of 40% after 8 weeks (TCE (200 µg/L) incubated with batch bacterial cultures under methanogenic conditions)	(Bouwer and McCarty, 1983)	High

Property or Endpoint	Value [a]	References	Data Quality Rating
	99.98% degradation after 2 or 4 days (anaerobic continuous flow)	(Vogel and McCarty, 1985)	High
	100% degradation after 20 days (aerobic with Methane culture, aerobic with phenol culture)	(Long et al., 1993)	High
Bioconcentration factor (BCF)	17 (Bluegill)	(Barrows et al., 1980)	High
Bioaccumulation factor (BAF)	24 (estimated)	(U.S. EPA, 2012b)	High
Organic carbon:water partition coefficient (Log K_{oc})	1.8 (estimated)	(U.S. EPA, 2012b)	High
[a] Measured unless otherwise noted			

2.1.1 Fate and Transport Approach and Methodology

EPA gathered and evaluated environmental fate information according to the process described in the Application of *Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018b). Reasonable available environmental fate data, including biotic and abiotic degradation rates, removal during wastewater treatment, volatilization from lakes and rivers, and organic carbon:water partition coefficient (Koc) were selected for use in this assessment document.

Other fate estimates were based on modeling results from EPI (Estimation Programs Interface) Suite™ (U.S. EPA, 2012b), a predictive tool for physical/chemical and environmental fate properties (https://www.epa.gov/tsca-screening-tools/epi-suitetm-estimation-program-interface). EPI Suite™ was reviewed by the EPA Science Advisory Board (http://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/CCF982BA9816F9CFCFA8525735200739805/$File/sab-07-011.pdf) and the individual models have been peer reviewed in numerous articles published in technical journals. Citations for such articles are available in the EPI Suite™ help files. Table 2-1 provides environmental fate data that EPA considered while assessing the fate of TCE.

2.1.2 Summary of Fate and Transport

The EPI Suite™ (U.S. EPA, 2012b) STP model was run using default settings (set biodegradation half-life to 10,000 hours) to evaluate the potential for TCE to volatilize to air or adsorb to sludge during wastewater treatment. In order to improve the accuracy of the EPI Suite™ estimations, physical and chemical properties (Log Kow, Boiling point, Melting point, Vapor Pressure, Water solubility, Henry's Law Constant) from Table 1-1 were entered into EPI Suite along with TCE's SMILES notation entry (C(=CCL)(CL)CL) before running the module.

If TCE is released to the air, TCE does not absorb radiation well at wavelengths that are present in the lower atmosphere (>290 nm) so direct photolysis is not a main degradation process. Degradation by reactants in the atmosphere has a half-life of several days meaning that long range transport is possible.

If TCE is released to water, sediment or soil, the fate of TCE is influenced by volatilization from the water surface or from soil as indicated by its physical chemical properties (e.g., Henry's law constant) and by microbial biodegradation under some conditions. The EPI Suite™ model that estimates volatilization from lakes and rivers ("Volatilization" model) was run using default settings to evaluate the volatilization half-life of TCE in surface water. The volatilization model estimates that the half-life of TCE in a model river is 1.2 hours and the half-life in a model lake is 110 hours. Therefore, the volatilization is likely to be a significant removal process.

If TCE is released to wastewater treatment, the removal percentage of TCE is estimted by using the STP model in EPI Suite™ as 81%, including 80% removal via volatilization and 1% removal via adsorption. This value (81%) is used for the calculation of exposure assement in this document. Therefore, TCE is not anticipated to partition to biosolids during wastewater treatment. Any TCE present in the water portion of biosolids following wastewater treatment and land application would be expected to rapidly volatilize into air. To further support this analysis, TCE was not detected in EPA's Targeted National Sewage Sludge Survey (TNSSS) nor was it reported in biosolids during EPA's Biennial Reviews for Biosolids, a robust biennial literature review conducted by EPA's Office of Water (U.S. EPA, 2019d). Furthermore, TCE is not anticipated to remain in soil, as it is expected to either volatilize into air or migrate through soil into groundwater.

The biodegradation of TCE in the environment is dependent on a variety of factors and thus, a wide range of degradation rates have been reported (ranging from days to years). The BIOWIN module in the EPI Suite™ was run using default settings to estimate biodegradation rates of TCE in soil and sediment. Three out of the four models built in the BIOWIN module (BIOWIN 1, 2, and 5) estimate that TCE will not rapidly biodegrade in aerobic environments, while a fourth (BIOWIN 6) estimates that TCE will rapidly biodegrade in aerobic environments. The weight of the scientific evidence from these estimates suggests that TCE does not biodegrade quickly under aerobic condition. This conclusion is supported by test results in a frequently cited publication (Rott et al.,1982) which indicates 19% aerobic biodegradation in 28 days (OECD 301D) and 2.4% aerobic biodegradation in 14 days (OECD 301C), respectively. The data was also cited in the 2004 EU TCE Risk Assessment (ECB, 2004).

During the systematic review process, a high-quality aerobic serum bottle biodegradation study, in which 100% degradation occurred in 20 days was reported in methane and phenol cultures. The result indicates that the aerobic degradation rate with either methane or phenol culture is "fast", is different from the BIOWIN predictions. However, the "fast" aerobic biodegradation with special cultures cannot represent general environmental conditions, so the "slow aerobic biodegradation" considered in the scoping and problem formulation documents was not changed in this risk evaluation document.

During the systematic review for fate endpoints, several high-quality anaerobic biodegradation test data were identified and inserted into the original fate table summarized in the Problem Formulation document (U.S. EPA, 2018c). The added anaerobic biodegradation data confirms that TCE anaerobic biodegradation rate is "fast".

The systematic review did not identify any additional studies for sorption coefficient to soil and sediments, therefore, the log K_{OC} value was estimated with EPI Suite™ as 1.8, which is close to the measured values ranged from 1.86 to 2.17 with different soils in the previous TCE assessments (U.S.

EPA, 2014b). These log K_{OC} values (1.8-2.17) suggest that the sorption of TCE to soil and sediment is low and TCE is mobile in soil and sediment.

The systematic review identified a high quality bioconcentration data with low BCF (BCF=17; Barrows, 1980). The BAF of TCE is also low (BAF=24) based on EPI Suite™ estimation. Therefore, TCE is not expected to accumulate in aquatic organisms due to low BCF and BAF.

2.1.3 Assumptions and Key Sources of Uncertainty for Fate and Transport

A range of biodegradation rates have been reported for TCE. The range of degradation rates reported were measured in laboratory studies for biodegradation in water, soil and sediment. These studies are subject to several sources of variability including variability inherent in the methodology, interlaboratory variability and variability due to factors such as the specific microbial populations used, water, soil and sediment chemistry, oxygen concentration/redox potential, of the collected samples used in the study, temperature and test substance concentration. No single value is universally applicable as it is influenced by these variables and possibly others. However, the weight of evidence shows the aerobic biodegradation of TCE is slow and the anerobic biodegradation in anaerobic condition is fast.

That range of Log K_{OC} values (1.8-2.17) is supported by the basic principles of environmental chemistry which states that the K_{OC} is typically within one order of magnitude (one log unit) of the octanol:water partition coefficient (K_{ow}).

2.2 Environmental Exposures

2.2.1 Environmental Exposures Overview

In this section, EPA presents environmental exposures to TCE for aquatic organisms. Exposure to terrestrial organisms is expected to be low since physical chemical properties do not support an exposure pathway through water and soil pathways to these organisms. To characterize environmental exposure, EPA assessed exposures derived from both predicted and measured concentrations of TCE in surface water in the U.S.

Aquatic exposures associated with the industrial and commercial conditions of use evaluated were predicted through modeling. Predicted surface water concentrations resulting from facility releases in the EPA Lifecycle Release Analysis were generated for reporting year 2016. Release estimates were based on loading and/or production volume information obtained from TRI, DMR, and CDR (See Section 2.2.2). The surface water modeling was conducted with EPA's Exposure and Fate Assessment Screening Tool, version 2014 (E-FAST 2014), using reported annual release/loading amounts (kg/yr) and estimates of the number of days per year that the annual load is released. The Probabilistic Dilution Model (PDM), a module of E-FAST 2014, was run to predict the number of days per year predicted stream concentrations are expected to exceed the designated chronic aquatic concentration of concern (COC) value.

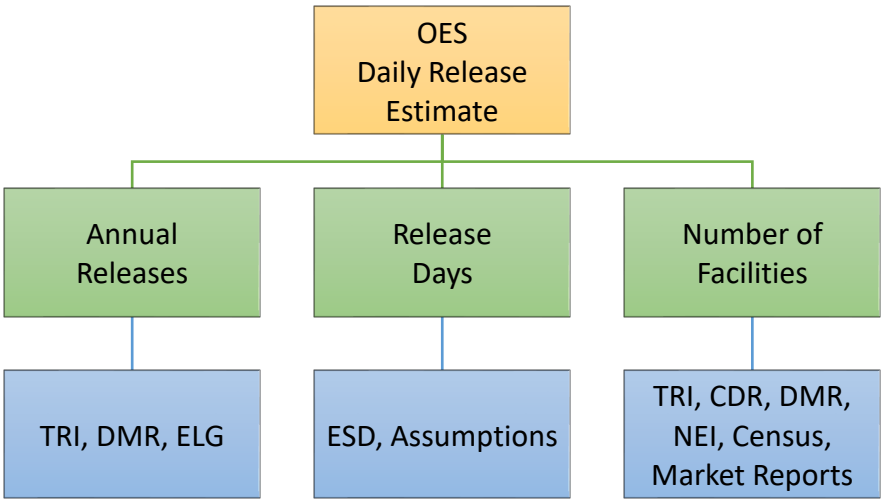
The aquatic exposure assessment also includes an analysis of collected measured surface water concentrations from monitoring data in EPA's Water Quality Exchange (WQX) using the online Water Quality Portal (WQP) tool and published literature obtained and evaluated through a systematic review process. WQX is the nation's largest source of water quality monitoring data and includes results from EPA's STORage and RETrieval (STORET) Data Warehouse, the United States Geological Service (USGS) National Water Information System (NWIS), and other federal, state, and tribal sources. A literature search was also conducted to identify other peer-reviewed or gray sources of measured surface water concentrations in the US. The measured concentrations reflect ambient surface water

concentrations at the monitoring sites but cannot be directly attributed to specific industrial or commercial conditions of use. A geospatial analysis at the watershed level was conducted to compare the measured and predicted surface water concentrations and investigate whether modeled facility releases may be located within the same watershed as observed concentrations in surface water.

2.2.2 Environmental Releases to Water

EPA categorized the conditions of use (COUs) listed in Table 1-4 into 18 Occupational Exposure Scenarios (OES). For each OES, a daily water release was estimated based on annual releases, release days, and the number of facilities (Figure 2-1). In this section, EPA describes its approach and methodology for estimating daily water releases, and for each OES, provides a summary of release days, number of facilities, and daily water releases. For detailed facility level results, see Appendix P of this document and the "Water Release Assessment" section for each OES in [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500)*].

Figure 2-1: An overview of how EPA estimated daily water releases for each OES.[8]



2.2.2.1 Results for Daily Release Estimate

EPA combined its estimates for annual releases, release days, and number of facilities to estimate a range for daily water releases for each OES. A summary of these ranges across facilities is presented in Table 2-2. See Table 2-5 for more details on deriving the overall confidence score for each OES. For some OES, EPA was not able to estimate or did not expect water releases. For example:

- **OES Aerosol Application:** Water releases were not expected due to the volatile nature of TCE; releases from this OES are expected to be to air.
- **OES Formulation of Aerosol and Non-Aerosol Products:** All releases reported in TRI were to off-site land, incineration, or recycling.

[8] TRI = Toxics Release Inventory; DMR = Discharge Monitoring Report; NEI = National Emissions Inventory; CDR = Chemical Data Reporting; ELG = Effluent Limitation Guidelines; ESD = Emission Scenario Document

Table 2-2: Summary of EPA's daily water release estimates for each OES and also EPA's Overall Confidence in these estimates.

Occupational Exposure Scenario (OES)	Estimated Daily Release Range Across Sites (kg/site-day)		Overall Confidence	Source and Notes
	Minimum	Maximum		
Manufacturing	0	1.27	M	From TRI, DMR
Processing as a Reactant	1.7E-03	0.02	M	From TRI, DMR
Formulation of Aerosol and Non-Aerosol Products	-	-	-	No information identified to estimate water releases
Repackaging	6.8E-06	1.1	M	From TRI, DMR
Batch Open-Top Vapor Degreasing	2.53E-07	1.96	M	From TRI, DMR
Batch Closed-Loop Vapor Degreasing	2.53E-07	1.96	M	Same as Batch Open-Top Vapor Degreasing[a]
Conveyorized Vapor Degreasing	2.53E-07	1.96	M	Same as Batch Open-Top Vapor Degreasing[a]
Web Vapor Degreasing	2.53E-07	1.96	M	Same as Batch Open-Top Vapor Degreasing[a]
Cold Cleaning	2.53E-07	1.96	M	Same as Batch Open-Top Vapor Degreasing[a]
Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	-	-	H	EPA expects releases of TCE to be to air for this OES
Metalworking Fluids	2.53E-07	1.96	M	Same as Batch Open-Top Vapor Degreasing[a]
Adhesives, Sealants, Paints, and Coatings	3.68E-06	0.30	M	From TRI, DMR
Other Industrial Uses	9.2E-06	1.6	M	From DMR
Spot Cleaning and Wipe Cleaning	2.9E-05	8.0E-05	M	From DMR
Industrial Processing Aid	5.5E-04	0.4	M	From TRI, DMR
Commercial Printing and Copying	2.0E-04	2.0E-04	-	Based on only one reported release in DMR
Other Commercial Uses	1.9E-06	0.013	M	From DMR
Process Solvent Recycling and Worker Handling of Wastes	1.6E-06	24.1	M	From TRI, DMR

[a] Water releases from OTVD were repeated for other degreasing operations and for MWF because the releases were estimated using TRI and DMR data. Due to the limited information in these reporting programs, these sites may in fact not operate OTVDs, but may operate other solvent cleaning machines or perform metalworking activities (e.g., closed-loop degreasing, conveyorized degreasing, web cleaning, or cold cleaning) or use of TCE as a metalworking fluid. They are included in the OTVD assessment as EPA expects OTVDs to be the most likely condition of use. EPA assessed annual releases as reported in the 2016 TRI or 2016 DMR and assessed daily releases by assuming 260 days of operation per year, as recommended in the 2017 ESD on Use of Vapor Degreasers, and averaging the annual releases over the operating days.

2.2.2.2 Approach and Methodology

2.2.2.2.1 Water Release Estimates

Where available, EPA used 2016 TRI (U.S. EPA, 2017g) and 2016 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 TCE and 10,000 pounds for users of TCE). Due to these limitations, some sites that manufacture, process, or use TCE may not report to TRI and are therefore not included in these datasets.

For the 2016 DMR (U.S. EPA, 2016a), EPA used the Water Pollutant Loading Tool within EPA's Enforcement and Compliance History Online (ECHO) to query all TCE 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 TCE may not be included in the DMR dataset.

Where releases are expected but TRI and DMR data were not available or where EPA determined TRI and DMR data did not sufficiently represent releases of TCE to water for a condition of use, releases were estimated using data from literature, relevant Emission Scenario Documents (ESDs) or Generic Scenarios (GSs), existing EPA models (e.g., EPA Water Saturation Loss Model), and/or relevant Effluent Limitation Guidelines (ELG). ELG are national regulatory standards set forth by EPA for wastewater discharges to surface water and municipal sewage treatment plants. For more details, please refer to Appendix I.

2.2.2.2.2 Estimates of Number of Facilities

Where available, EPA used 2016 CDR (U.S. EPA, 2016c), 2016 TRI (U.S. EPA, 2017g), 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 TCE within a condition of use. Generally, information for reporting sites in CDR and NEI was sufficient to accurately characterize each reporting site's 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 reported NAICS codes could include multiple conditions of use; 2) the site reported 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:

- Information on known uses of the chemical and market data identifying the most prevalent conditions of use of the chemical.

- 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).

The National Emissions Inventory (NEI) is a comprehensive and detailed estimate of air emissions of criteria pollutants, criteria precursors, and hazardous air pollutants from air emissions sources. The NEI is released every three years based primarily upon data provided by State, Local, and Tribal air agencies for sources in their jurisdictions and supplemented by data developed by the US EPA. The inventory includes emissions estimates for larger sources that are located at a fixed, stationary location (point sources) and emissions estimates for sources which individually are too small in magnitude to report as point sources (nonpoint sources). In NEI, facilities report on the equipment or process sources for their facility emissions. Based on these reported point sources for TCE emissions, EPA could generally determine which condition of use the facility fell in.

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

- Identify the North American Industry Classification System (NAICS) codes for the industry sectors associated with these uses.
- Estimate total number of sites using the U.S. Census' Statistics of US Businesses (SUSB) (U.S. Census Bureau, 2015) data on total establishments by 6-digit NAICS.
- Use market penetration data to estimate the percentage of establishments likely to be using TCE instead of other chemicals.
- Combine the data generated in Steps 1 through 3 to produce an estimate of the number of sites using TCE 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: Summary of EPA's estimates for the number of facilities for each OES.

Occupational Exposure Scenario (OES)	Number of Facilities	Notes
Manufacturing	5	Based on CDR reporting
Processing as a Reactant	5 to 440	Based on TRI and DMR reporting, and Census data for NAICS 325120 (Industrial Gas Manufacturing)
Formulation of Aerosol and Non-Aerosol Products	19	Based on TRI reporting
Repackaging	22	Based on TRI and DMR reporting
Batch Open-Top Vapor Degreasing	194	Based on NEI and TRI reporting
Batch Closed-Loop Vapor Degreasing	4	Based on NEI reporting
Conveyorized Vapor Degreasing	8	Based on NEI reporting
Web Vapor Degreasing	1	Based on NEI reporting

Occupational Exposure Scenario (OES)	Number of Facilities	Notes
Cold Cleaning	13	Based on NEI reporting
Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	4,366	Based on Census data and market penetration estimates based on California Air Resources Board (CARB) survey of automotive maintenance and repair facilities
Metalworking Fluids	-	No information identified to estimate number of facilities
Adhesives, Sealants, Paints, and Coatings	70	Based on NEI, TRI, and DMR reporting
Other Industrial Uses	49	Based on TRI and DMR reporting
Spot Cleaning and Wipe Cleaning	63,748	Based on Census data for NAICS codes 812300, 812320, 561740; assumed 100% market penetration for TCE.
Industrial Processing Aid	18	Based on TRI and DMR reporting
Commercial Printing and Copying	-	No information identified to estimate number of facilities
Other Commercial Uses	-	No information identified to estimate number of facilities
Process Solvent Recycling and Worker Handling of Wastes	30	Based on TRI and DMR reporting

2.2.2.2.3 Estimates of Release Days

EPA referenced Emission Scenario Documents (ESDs) 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 expected for each OES.

Occupational Exposure Scenario (OES)	Release Days	Notes
Manufacturing	350	Assumed seven days per week and 50 weeks per year with two weeks per year for shutdown activities.
Processing as a Reactant	350	Assumed seven days per week and 50 weeks per year with two weeks per year for shutdown activities.
Formulation of Aerosol and Non-Aerosol Products	-	Water releases not estimated for this OES.
Repackaging	250	Assumed 5 days per week and 50 weeks per year.
Batch Open-Top Vapor Degreasing	260	2017 ESD on Use of Vapor Degreasing
Batch Closed-Loop Vapor Degreasing	260	2017 ESD on Use of Vapor Degreasing
Conveyorized Vapor Degreasing	260	2017 ESD on Use of Vapor Degreasing
Web Vapor Degreasing	260	2017 ESD on Use of Vapor Degreasing
Cold Cleaning	260	2017 ESD on Use of Vapor Degreasing
Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	-	Water releases not expected from this OES.
Metalworking Fluids	260	2017 ESD on Use of Vapor Degreasing

Occupational Exposure Scenario (OES)	Release Days	Notes
Adhesives, Sealants, Paints, and Coatings	250	2011 ESD on the Application of Radiation Curable Coatings, Inks, and Adhesives via Spray, Vacuum, Roll and Curtain Coating
Other Industrial Uses	250	Assumed 5 days per week and 50 weeks per year.
Spot Cleaning and Wipe Cleaning	300	Assumed 6 days per week and 50 weeks per year.
Industrial Processing Aid	300	Assumed 6 days per week and 50 weeks per year.
Commercial Printing and Copying	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.
Process Solvent Recycling and Worker Handling of Wastes	250	Assumed 5 days per week and 50 weeks per year.

2.2.2.3 Assumptions and Key Sources of Uncertainty for Environmental Releases

EPA estimated water releases using reported discharges from the 2016 TRI and the 2016 DMR. TRI and DMR data were determined to have a "medium" confidence rating through EPA's systematic review process. Due to reporting requirements for TRI and DMR, the number of sites for a given OES may be underestimated. It is uncertain, the extent to which, sites not captured in these databases discharge wastewater containing TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT.

In addition, information on the use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether the number of facilities estimated for a given OES do in fact represent that specific OES. If 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 release days expected for the different OES.

Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA estimated the release days and averaged the annual releases over these days. There is some uncertainty that all sites for a given OES operate for the assumed duration; therefore, the average daily discharges may be higher if sites have fewer release days or lower if they have greater release days. TRI-reporting facilities are required to submit their "best available data" to EPA for TRI reporting purposes. Some facilities are required to measure or monitor emission or other waste management quantities due to regulations unrelated to the TRI Program (e.g., permitting requirements), or due to company policies. These existing, reasonably available data are often used by facilities for TRI reporting purposes, as they represent the best available data. When monitoring or direct measurement data are not reasonably available, or are known to be non-representative for TRI reporting purposes, the TRI regulations require that facilities determine release and other waste management quantities of TRI-listed chemicals by making reasonable estimates. These reasonable estimates may be obtained through various Release Estimation Techniques, including mass-balance calculations, the use of emission factors, and engineering calculations. There may be greater uncertainty in data resulting from estimates compared to monitoring measurements. However, available monitored data that showed ambient water concentrations were not useful in corroborating the modeling approach because most of them were far downstream from the near-facility modeled concentration estimates.

Furthermore, TCE 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.

In some cases, the number of facilities for a given OES was estimated using data from the U.S. Census. In such cases, the average daily release calculated from sites reporting to TRI or DMR was applied to the total number of sites reported in (U.S. Census Bureau, 2015). It is uncertain how accurate this average release is to actual releases at these sites; therefore, releases may be higher or lower than the calculated amount.

The 2014 NEI was also used to estimate the number of facilities for various OES. NEI does not report water release information, therefore, an average release was calculated from the sites reporting water releases to TRI and DMR and applied to sites reported in NEI. It is uncertain how accurate this average release is to actual releases at these sites; therefore, releases may be higher or lower than the calculated amount.

2.2.2.3.1 Summary of Overall Confidence in Release Estimates

Table 2-5 provides a summary of EPA's overall confidence in its release estimates for each of the Occupational Exposure Scenarios assessed.

Table 2-5: Summary of Overall Confidence in Release Estimates by OES.

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
Manufacturing	Wastewater discharges are assessed using reported discharges from the 2016 TRI for three sites. TRI data were determined to have a "medium" confidence rating through EPA's systematic review process. Facilities reporting to TRI only report annual discharges; to assess daily discharges, EPA assumed 350 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites manufacturing TCE will operate for this duration; 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, TCE 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. One of the three sites reporting to TRI also reported to DMR. This information was also assessed. The same uncertainties discussed above for TRI releases also apply to the DMR data. Based on this information, EPA has a medium confidence in the wastewater discharge estimates for the four sites in the 2016 TRI and 2016 DMR. Water discharges from the remaining two sites were estimated using the maximum daily and monthly discharge limits in the OCPSF EG and the estimated volume of wastewater produced per pound of TCE production from the Specific Environmental Release Category (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. Based on this information EPA has a medium confidence in the wastewater discharge estimates for these two sites.

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
Processing as a Reactant	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are processing TCE as a reactant rather than a different OES. 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. Facilities reporting to TRI and DMR only report annual discharges; to assess daily discharges, EPA assumed 350 days/yr of operation and averaged the annual discharges over the operating days. There is some uncertainty that all sites processing TCE as a reactant will operate for this duration; 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, TCE 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.
Formulation of Aerosol and Non-Aerosol Products	All sites reporting in TRI show zero water releases; EPA does not expect water releases from this OES.
Repackaging	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are performing repackaging activities rather than a different OES. 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. 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 TCE will operate for this duration; 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, TCE

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
	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.
Batch Open-Top Vapor Degreasing	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" confidence rating through EPA's systematic review process. Due to reporting requirements for TRI and DMR, EPA does not expect all sites using TCE in OTVD to be captured in the databases. It is uncertain the extent that sites not captured in these databases discharge wastewater containing TCE 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 TCE 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 TCE in OTVD rather than a different OES (including other vapor degreasing and cold cleaning operations and use of TCE 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. 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 TCE in OTVDs will operate for this duration; 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 260 days/yr. Furthermore, TCE 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.
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 Vapor Degreasing	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Cold Cleaning	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	EPA assessed no wastewater discharges for this OES. There is some uncertainty as to whether and how much TCE may deposit on shop floors. However, due to the volatility of TCE, EPA expects TCE 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)	Overall Confidence in Release Estimates
Metalworking Fluids	Same as the Open-Top Vapor Degreasing (OTVD) OES.
Adhesives, Sealants, Paints, and Coatings	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are performing adhesive, sealant, paint or coating activities rather than a different OES. 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. 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 TCE in adhesives, sealants, paints and coatings will operate for this duration; 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, TCE 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. There is further uncertainty that the number of sites obtained from the 2014 NEI represent the total number of sites using adhesives, sealants, paints or coatings containing TCE. NEI data only covers specific industries which may not capture the entirety of industries using these products and NEI 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Also, NEI do not report water release information, therefore, an average release was calculated from the sites reporting water releases to TRI and DMR and applied to sites reported in NEI. It is uncertain how accurate this average release is to actual releases as these sites; therefore, releases may be higher or lower than the calculated amount. Based on this information, EPA has a medium confidence in the wastewater discharge estimates.
Other Industrial Uses	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT.

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
	Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are performing other industrial uses rather than a different OES. 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. 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 TCE for other industrial uses will operate for this duration; 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, TCE 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.
Spot Cleaning and Wipe Cleaning	Wastewater discharges from spot cleaning facilities at industrial launderers are assessed using reported discharges from the 2016 DMR. DMR data were determined to have a "medium" confidence rating through EPA's systematic review process. DMR only contains information for 2 sites. Additional 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). 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 TCE will operate for this duration; 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, TCE 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. There is further uncertainty that the releases estimated for the total number of sites obtained from the U.S. Census' Bureau for spot, carpet and wipe cleaning accurately reflect releases from these sites. An average release was calculated from the sites reporting water releases to DMR and applied to the total number of sites reported in (U.S. Census Bureau, 2015). It is uncertain how accurate this average release is to actual releases as these sites; therefore, releases may be higher or lower than the calculated amount. It is also uncertain the extent that sites not captured in this assessment discharge wastewater containing TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Based on this information, EPA has a medium confidence in the wastewater discharge estimates.

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
Industrial Processing Aid	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are using TCE as an industrial processing aid rather than a different OES. 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. 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 TCE as an industrial processing aid will operate for this duration; 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, TCE 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.
Commercial Printing and Copying	Wastewater discharges from one commercial printing and copying site was found in the 2016 DMR. DMR data were determined to have a "medium" confidence rating through EPA's systematic review process. However, EPA acknowledges this site does not represent the entirety of commercial printing and copying sites using TCE; data was not reasonably available to estimate water releases from additional sites.
Other Commercial Uses	Wastewater discharges are assessed using reported discharges from the 2016 DMR. DMR data were determined to have a "medium" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are performing other commercial uses rather than a different OES. 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. Facilities reporting to DMR only report annual discharges; to assess daily discharges, EPA assumed 250 days/yr of operation and averaged the annual

Occupational Exposure Scenario (OES)	Overall Confidence in Release Estimates
	discharges over the operating days. There is some uncertainty that all sites using TCE in other commercial uses will operate for this duration; 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, TCE 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.
Process Solvent Recycling and Worker Handling of Wastes	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" confidence rating through EPA's systematic review process. 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 TCE and whether any such discharges would be to surface water, POTW, or non-POTW WWT. Additionally, information on the conditions of use of TCE at facilities in TRI and DMR is limited; therefore, there is some uncertainty as to whether all the sites assessed in this section are recycling/disposing of TCE rather than a different OES. 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. 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 recycling/disposing of TCE will operate for this duration; 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, TCE 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.

2.2.3 Aquatic Exposure Modeling Approach

Surface water concentrations resulting from wastewater releases of TCE from facilities that use, manufacture, or process TCE related to the evaluated industrial and commercial conditions of use were modeled using EPA's Exposure and Fate Assessment Screening Tool, Version 2014 (U.S. EPA, 2014c). E-FAST 2014 estimates chemical concentrations in surface water resulting from releases to surface water, resulting in exposure estimates at the point of release. Advantages to this model are that it requires minimal input parameters and it has undergone extensive peer review by experts outside of EPA. A brief description of the calculations performed within the tool, as well as a description of required inputs and the methodology to obtain and use inputs specific to this assessment is described below. To obtain more detailed information on the E-FAST 2014 tool from the model documentation (U.S. EPA, 2007), 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/.

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

The required modeling inputs are discussed below.

Chemical release to wastewater (WWR)

Annual wastewater loading estimates (kg/site/year or lb/site/year) were predicted in Section 2.2.2 and based on reported production loading or production volume estimates**.** To model these releases within Exposure and Fate Assessment Screening Tool 2014, the annual release is converted to a daily release using an estimated days of release per year. Below is an example calculation:

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

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

Release Days (days/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 Eq. 3). 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 a range of surface water concentrations predicted by E-FAST 2014. The two scenarios modeled are a higher release frequency (200 to 365 days) based on release estimates in Section 2.2.2 and a low-end release frequency of 20 days of release per year as an estimate of releases that could lead to chronic risk for aquatic organisms. 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 discharges from water treatment facilities (e.g., POTWs, STPs, WWTPs), only the higher release frequency was modeled because such treatment sites are anticipated to discharge more frequently than non-treatment facilities.

Removal from wastewater treatment (WWR%)

The WWR% is the percentage of the chemical removed from wastewater during treatment before discharge to a body of water. As discussed in Section 2.1.1, the WWR% for TCE is estimated as 81%. The WWR% of 81% was applied, when appropriate, to volumes characterized as being transferred off-site for treatment at a water treatment facility prior to discharge to surface water. A WWR% of zero was used for direct releases to surface water because the release estimates are based on estimated release (post-treatment). In cases where it wasn't clear whether the release was direct or indirect, both possible scenarios were modeled.

Facility or Industry Sector

The required site-specific stream flow or dilution factor information is contained in the E-FAST 2014 database, which is accessed by querying a facility National Pollutant Discharge Elimination System (NPDES) number, facility name, or reach code. For facilities that directly discharge to surface water (i.e., "direct dischargers"), the NPDES of the direct discharger is selected from the database. For facilities that indirectly discharge to surface water (i.e., "indirect dischargers" because the release is sent to a water treatment facility prior to discharge to surface water), the NPDES of the receiving treatment facility is 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. If a facility NPDES was not available in the E-FAST-2014 database, the release was modeled using water body data for a surrogate NPDES (preferred) or an industry sector, as described below.

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

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. This option uses the 10th and 50th percentile receiving stream flows for dischargers in a given industry sector, as defined by the Standard Industrial Classification (SIC) codes of the industry. Table 2-6 below provides the industrial sectors that were applied as needed for each condition of use category.

Table 2-6 Industry Sector Modeled for Facilities without Site-Specific Flow Data in E-FAST 2014

Condition of Use	Industry Sector in E-FAST 2014 for Stream Flow Data [1]
OES: Adhesives, Sealants, Paints, and Coatings	Adhesives and Sealants Manufacture
OES: Commercial Printing and Copying	Printing
OES: Industrial Processing Aid	POTW[2] (Industrial)
OES: Manufacturing	Organic Chemicals Manufacture
OES: N/A Water Treatment Facility	POTW[2] (Industrial)
OES: Other Commercial Uses	POTW[2] (Industrial)
OES: Other Industrial Uses	POTW[2] (Industrial)
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, Cold Cleaning, and Metalworking Fluids)	Primary Metal Forming Manufacture
OES: Process Solvent Recycling and Worker Handling of Wastes	POTW[2] (Industrial)
OES: Processing as a Reactant	Organic Chemicals Manufacture
OES: Repackaging	n/a
OES: Spot Cleaning and Carpet Cleaning	n/a

[1] n/a = Not applicable because a NPDES or surrogate NPDES was available in E-FAST 2014 to obtain a site-specific stream flow for all facilities within the OES.
[2] POTW = Publicly Owned Treatment Works

Concentration of Concern

Concentrations of Concern (COCs) are threshold concentrations below which adverse effects on aquatic life are expected to be minimal. See Section 3.1.5 for a full discussion of acute and chronic COCs for TCE. For E-FAST modeling, only the chronic COCs are entered for use in PDM runs, which compare estimated stream concentrations calculated based on an annual stream flow distribution to the chronic COCs and return the number of days per year the selected COCs are exceeded. The COCs used in the PDM module of E-FAST 2014 for TCE were 3, 788, and 52,000 ppb.

2.2.3.2 E-FAST 2014 Equations

Surface Water Concentrations

E-FAST 2014 estimates 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 can calculate surface water concentrations for four streamflow conditions (7Q10, harmonic mean, 30Q5, and 1Q10 flows) using the following equation:

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

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)
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 accounts for dilution by incorporating an acute or chronic dilution factor for the water body of interest instead of streamflows. Dilution factors in E-FAST 2014 are typically 1 (representing no dilution) to 200. The following equation is used to calculate surface water concentrations in still water bodies:

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

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)

Days of COC Exceedance

The Probabilistic Dilution Model (PDM) portion of E-FAST 2014 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 that can affect the chemical concentration and flow rate of the effluent. PDM does not estimate exceedances for chemicals discharged to still waters, such as lakes, bays, or estuaries. For these water bodies, the days of exceedance is assumed 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).

2.2.3.3 E-FAST 2014 Outputs

E-FAST 2014 provides esitmates of surface water concentration for multiple stream flow parameters. The concentrations reflect predicted levels of TCE in the receiving water body at the point of release and do not incorporate downstream transport or post-release chemical fate processes. For this aquatic exposure assessment, 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 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 reasonably 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. Estimates from this calculation method are reported for the 10th Percentile harmonic mean and 10th Percentile 7Q10 stream flows.

2.2.4 Surface Water Monitoring Data Gathering Approach

2.2.4.1 Method for Systematic Review of Surface Water Monitoring Data

EPA conducted a full systematic review of published literature to identify studies reporting concentrations of TCE in surface water in the United States. Studies clearly associated with releases from Superfund sites, improper disposal methods, and landfills were considered not to meet the PECO statement and excluded from data evaluation and extraction. The systematic review process is described in detail in Section 1.5. A total of 28 surface water studies were extracted and the results are summarized in Section 2.2.6.2.2. No concentration data from the US were identified prior to 2000.

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

For this aquatic exposure assessment, the primary source for the occurrence of TCE in surface water is monitoring data retrieved from the Water Quality Portal (WQP), which integrates publicly available US water quality data from multiple databases: 1) the United States Geological Survey National Water Information System (USGS NWIS); 2) EPA's STOrage and RETrieval (STORET); and 3) the United States Department of Agriculture Agricultural Research Service (USDA ARS) Sustaining The Earth's Watersheds - Agricultural Research Database System (STEWARDS). NWIS is the Nation's principal repository of water resources data USGS collects from over 1.5 million sites, including sites from the National Water-Quality Assessment (NAWQA). STORET refers to an electronic data system originally created by EPA in the 1960's to compile water quality monitoring data. NWIS and STORET now use common web services, allowing data to be published through the WQP tool. The WQP tool and User Guide is accessed from the following website: (http://www.waterqualitydata.us/portal.jsp).

Data Retrieval from WQP

Surface water data for TCE were downloaded from the WQP on October 3, 2018. The WQP can be searched through three different search options: Location Parameters, Site Parameters, and Sampling Parameters. Three queries were performed using the Sampling Parameters search, as shown in Figure 2-2. One query obtained STORET data using the Characteristics parameter (selected "Trichlorethylene (STORET)" and two queries obtained NWIS data using the Parameter Codes (34485 for "Trichloroethene, water, filtered, recoverable, micrograms per liter" and 39180 for "Trichloroethene, water, unfiltered, recoverable, micrograms per liter"). Parameters codes were obtained from the USGS website https://nwis.waterdata.usgs.gov/usa/nwis/pmcodes using the chemical CASRN. All queries were performed using a Date Range of 01-01-2013 to 12-31-2017. Both the "Site data only" and "Sample results (physical/chemical metadata)" were selected for download in "MS Excel 2007+" format. The "Site data only" file contains monitoring site information (i.e., location in hydrologic cycle, HUC and geographic coordinates); whereas the "Sample result" file contains the sample collection data and analytical results for individual samples.

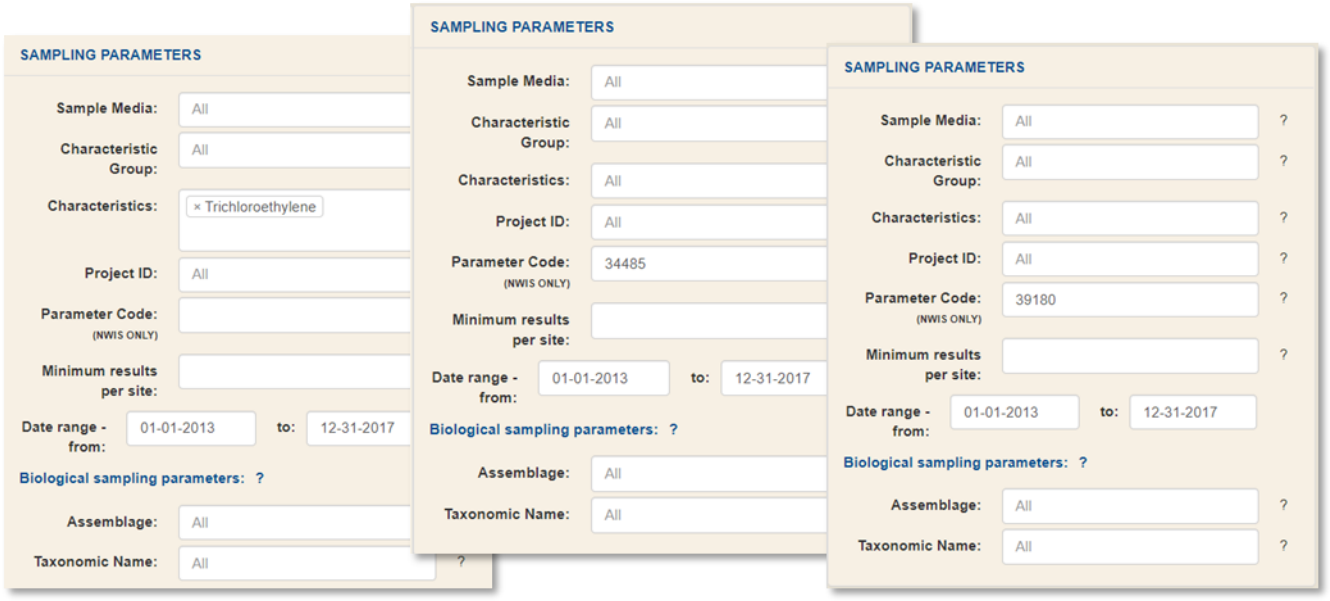


Figure 2-2. WQP Search Option. Surface water data were obtained from the WQP by querying the Sampling Parameters search option for the characteristic (STORET data), Parameter Code (NWIS data), and date range parameter

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. 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).

Following filtering to obtain the final dataset, the domains "ResultDetectionConditionText," "ResultCommentText," and "MeasureQualifierCode" 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).

2.2.5 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 Hydrologic Unit Code (HUC) 8 and HUC 12 level. The purpose of the analysis is to identify if any the observed surface water concentrations could be associated with 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. 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.

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.

Surface Water Concentrations

The surface water concentrations associated with the monitoring stations and facility releases are denoted on the maps using COCs to determine the concentration thresholds:

red	≥52,000 µ/L (exceeds all COC for algae, aquatic invertebrate, and fish
orange	788-51,999 µ/L (exceeds the COC for algae and aquatic invertebrate, but not for fish)
green	3-787 µ/L (exceeds the COC for algae, but not for aquatic invertebrate or fish)
blue	Detected, but less than 3 µ/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, 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: ≥52,000 µ/L (red), 788-51,999 µ/L (orange), 3-787 µ/L (green), <3µ/L (blue), and not detected (purple).

2.2.6 Environmental Exposure Results

2.2.6.1 Terrestrial Environmental Exposures

Exposure to terrestrial organisms is expected to be low since physical chemical properties do not support an exposure pathway through water, biosolids, and soil pathways to these organisms. The partition of TCE into sediments is very low. Furthermore, the primary fate of TCE released to surface waters or surface soils is volatilization.

2.2.6.2 Aquatic Environmental Exposures

To characterize environmental exposure, EPA assessed surface water concentrations derived from both predicted concentrations of TCE in surface water (using E-FAST modeling results) and measured concentrations (using monitored data from WQP and the published literature). Generally, the modeled concentrations reflect near-site estimates at the point of release, and the measured concentrations reflect localized ambient water concentrations at the monitoring sites. However, there were several sources in the published literature that represent near facility concentrations and are labeled as such.

2.2.6.2.1 Predicted Surface Water Concentrations: E-FAST 2014 Modeling

A summary of the surface water concentration estimates modeled using E-FAST 2014, based on the lifecycle release analysis for the year 2016, is summarized by OES category in Table 2-7 through Table 2-9. A break-out of facility-specific modeling results organized per OES, with predicted surface water concentrations and associated days of COC exceedance, are included in Appendix C. These facility-specific modeling results are utilized and discussed in environmental risk characterization presented in Section 4.1.2.

For the higher release frequency scenarios (250-365 days of release/year), predicted surface water concentrations under 7Q10 flow conditions ranged from 1.27E-5 to 765.63 ppb (Table 2-7). For the 20 days of release/year scenario for direct dischargers, predicted surface water concentrations under 7Q10 flow conditions ranged from 0.00019 to 9,937.5 ppb (Table 2-8). 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 0.2 to 339.11 ppb (Table 2-9).

Table 2-7. Summary of Surface Water Concentrations by OES for Maximum Days of Release Scenario

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (ppb)	
		Min	Max
Manufacturing	6	0.00514	2.77
Processing as a Reactant (low-end # of sites)	3	0.0000518	169
Processing as a Reactant	4	0.18	0.92
Repackaging	4	0.0000189	27.18
OTVD	51	0.0000822	765.63
Adhesives, Sealants, Paints, and Coatings	104	0.000818	10.83
Other Industrial Uses	16	0.0000941	9.5
Spot Cleaning and Carpet Cleaning	1	0.00388	0.00388
Industrial Processing Aid	6	0.000419	9.3
Commercial Printing and Copying	1	0.00292	0.00292
Other Commercial Uses	5	0.00564	9
Process Solvent Recycling and Worker Handling of Wastes	4	0.98	11.76
N/A (WWTP)	9	0.0000127	0.7
Grand Total	**214**	**1.27E-5**	**765.63**

Table 2-8. Summary of Surface Water Concentrations by OES for 20 Days of Release Scenario For Direct Releases

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (ppb)	
		Min	Max
Manufacturing	3	0.0897	49.91
Processing as a Reactant (low-end # of sites)	3	0.000907	3000
Processing as a Reactant	2	16.45	16.45
Repackaging	3	0.000235	89.13
OTVD	51	0.00103	9937.5
Adhesives, Sealants, Paints, and Coatings	52	0.0101	133.33
Other Industrial Uses	16	0.00154	200
Spot Cleaning and Carpet Cleaning	1	0.0485	0.0485
Industrial Processing Aid	3	0.00335	2.2
Commercial Printing and Copying	1	0.0365	0.0365
Other Commercial Uses	5	0.0658	110
Process Solvent Recycling and Worker Handling of Wastes	1	138.24	138.24
N/A (WWTP)	9	0.00019	12.79
Grand Total	**150**	**0.00019**	**9,937.5**

Table 2-9. Summary of Surface Water Concentrations by OES for 20 Days of Release Scenario for Indirect Releases to a non-POTW WWTP

OES	No. of Releases Modeled	Surface Water Concentration (7Q10) (ppb)	
		Min	Max
Manufacturing	3	9.48	42.14
Processing as a Reactant	1	3.13	3.13
Repackaging	1	339.11	339.11
Industrial Processing Aid	3	0.2	138.34
Process Solvent Recycling and Worker Handling of Wastes	3	11.26	106.75
Grand Total	**11**	**0.2**	**339.11**

On a site-specific basis, the predicted surface water concentrations did not exceed the highest COC (52,000 ppb) for any facility and only exceeded the COC of 788 ppb for two releasing facilities (US Nasa Michoud Assembly Facility in New Orleans, LA and Praxair Technology Center in Tonawanda, NY). These release scenarios were 20-day scenarios involving release to a still water body, which applied no additional dilution. There were 102 modeled releases that exceeded the lowest COC of 3 ppb. A detailed summary table by facility is provided in Appendix C.

Characterization of Modeled Releases

As discussed in Section 2.2.2, releases of TCE were estimated based on data from TRI, DMRs, and CDR (primarily TRI and DMR) for the 2016 calendar year. Release estimates were generally facility-specific and releasing facilities were assigned to one of 13 occupational exposure scenarios (OES). Overall, modeling was conducted on 157 unique active releasing facilities plus one OES with sites nationwide (440 unknown sites in OES Processing as a Reactant). As shown in Figure **2-3.**, the releases occurred in 39 states. With respect to watersheds, the releases occurred across 122 HUC-8 areas and 144-HUC 12 areas.

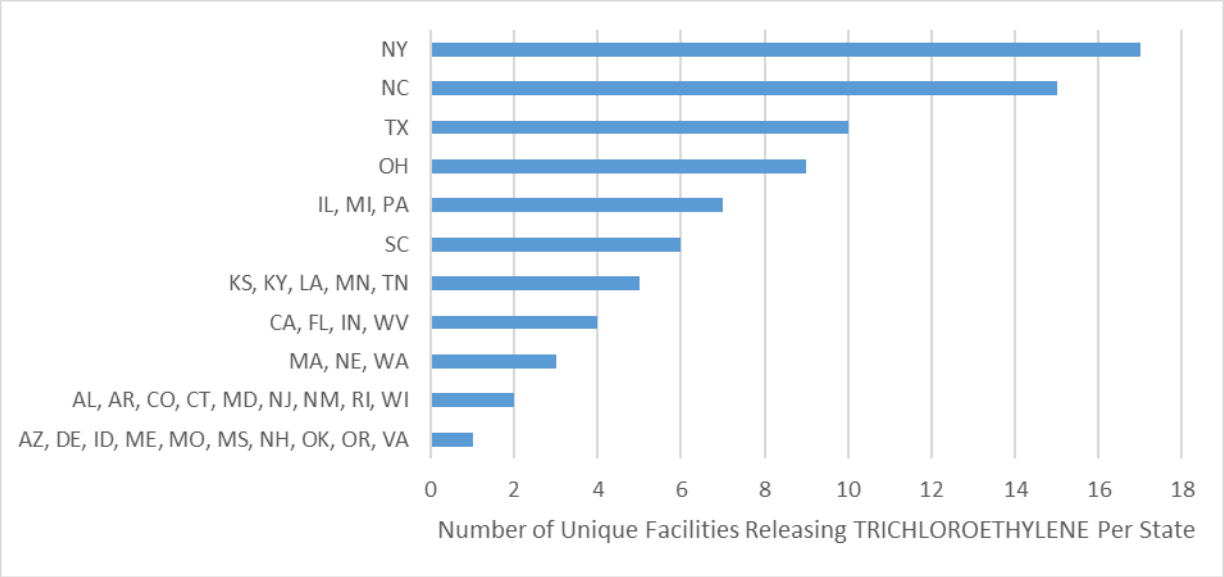


Figure 2-3. Distribution of Active Facility Releases Modeled

As shown in Figure 2-4, direct and indirect dischargers accounted for 70% and 30% of the total releases modeled, respectively. Site-specific waterbody flow/dilution data (identified via NPDES) were available in E-FAST 2014 for the majority of the releases (58%); surrogate waterbody flow/dilution data were used in only 15% of the cases, with the remaining cases (26%) 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 86% of the cases, and still water bodies for the remaining cases (14%).

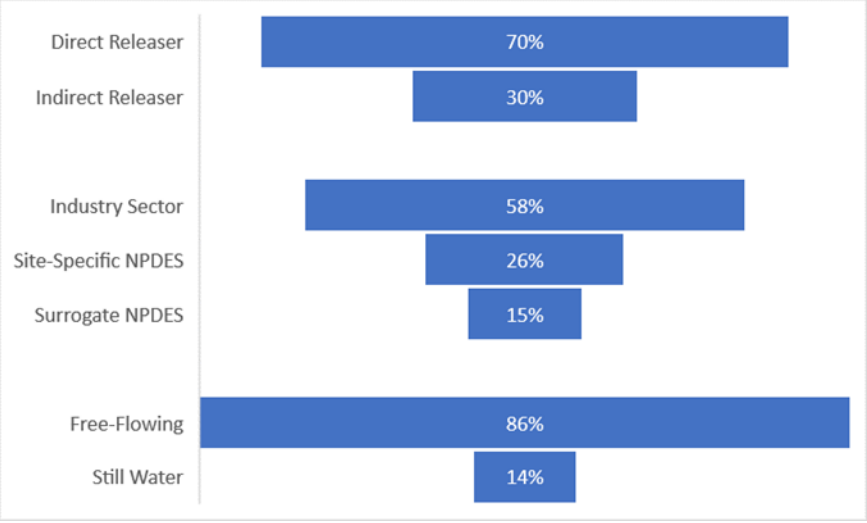


Figure 2-4. Modeled Release Characteristics (Percent Occurrence)

2.2.6.2.2 Monitored Surface Water Concentrations

Measured Concentrations of TCE from WQX/WQP

A summary of the WQX data obtained from the WQP is provided in Table 2-10 below for years 2013-2017. Per year, the cleansed datasets evaluated contained between 46 and 793 surface water samples collected from 89 to 193 unique monitoring stations. Detection frequencies were low, ranging from 0 to

8.7%. Concentrations ranged from not detected (ND; <0.022-5) to 0.11 µg/L in 2013, ND (<0.022-5) to 1.86 µg/L in 2014, ND (<0.025-2.4) to 0.011 µg/L in 2015, all ND (<0.025-5) in 2016, and ND (<0.025-5) to 2.0 µg/L in 2017. Peaks were observed in 2014 and 2017; however, caution should be used in interpreting trends with these data due to the small number of samples and the lack of samples collected from the same sites over multiple years. The quantitative environmental assessment used the 2016 data set only. For the 2016 data, concentrations in all samples were non-detect. No samples in the 2013-2017 dataset had concentrations exceeding the lowest COC of 3 µg/L.

Table 2-10. Measured Concentrations of TCE in Surface Water Obtained from the Water Quality Portal: 2013-2017[1]

Year	Detection Frequency	Concentration (µg/L) in all samples			Concentrations (µg/L) in only samples above the detection limit		
		No. of Samples (No. of Unique Stations)	Range[2]	Average (Standard Deviation)[3]	No. of Samples (No. of Unique Stations)	Range	Average (Standard Deviation)3
2013	4.67%	793 (164)	ND (<0.022-<5) to 0.11	0.21 (0.26)	37 (22)	0.008 to 0.11	0.051 (0.016)
2014	3.78%	609 (155)	ND (<0.022-<5) to 1.86	0.33 (0.31)	23 (13)	0.0055 to 1.86	0.17 (0.41)
2015	1.42%	352 (91)	ND (<0.025-<2.4) to 0.011	0.42 (0.16)	5 (2)	0.0075 to 0.011	0.009 (0.001)
2016	0.0%	473 (109)	ND (<0.025-<5)	0.44 (0.27)	0 (0)	NA	NA
2017	8.70%	46 (25)	ND (<0.025-<5) to 2.0	0.47 (0.53)	4 (1)	1.0 to 2.0	1.5 (0.71)
All Years	3.04%	2273 (384)	ND (<0.022-<5) to 2.0	0.33 (0.29)	69 (39)	0.0055 to 2.0	0.13 (0.35)

[1]Data were downloaded from the Water Quality Portal (www.waterqualitydata.us) on 10/3/2018. STORET surface water data was obtained by selecting "TCE (STORET)" for the Characteristic. NWIS surface water data were obtained by selecting "34485; 39180" for the Parameter Codes. Samples were filtered for surface water media and locations only. Results were reviewed and cleansed (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.).

[2]ND = Not Detected. Reported detection limits in all samples ranged from 0.022 to 5 µg/L.

[3]Calculations were performed using ½ the reported detection limit when results were reported as not detected. If a detection limit was not provided, calculations were performed using the average of the reported detection limits in all samples (0.65 µg/L).

Characterization of WQX Data

The original dataset downloaded contained 31,456 samples for years 2013 through 2017. Following the filtering and cleansing procedure, only 7% of the samples remained (2,273 samples). The majority of the samples 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, spring, or well). A smaller number of samples were excluded because they were quality control samples, estimated values, or had other quality control issues. Samples associated with one Superfund site (Palermo Wellfield Superfund Site) were also excluded.

For the 2016 cleansed dataset (473 samples), observations were made in 10 states/territories (AZ, KS, MN, MO, NJ, NM, NC, PA, TN, and TX) at 109 unique monitoring sites, with 1 to 13 samples collected per sampling site.

Measured Concentrations of TCE from Published Literature

Systematic review of published literature yielded only a minimal amount of surface water monitoring data for TCE; a summary of the individual studies is provided in Table 2-11**.** In six U.S. studies encompassing 1,177 surface water samples collected from river and oceans throughout the nation between 1979 and 2001, reported concentrations of TCE ranged from below the detection limit (0.0001 to 0.08) to 17.3 µg/L, with reported central tendency values ranging from 0.0002 to 1.17 µg/L. The maximum concentration was collected from the Charles River in Boston, Massachusetts (an urban area) between 1998 and 2000 (Robinson et al., 2004). The next highest TCE concentration was 2.0 µg/L, collected during a large nationwide survey of surface water for drinking water sources (rivers and reservoirs) between 1999 and 2000 (USGS, 2003). Robinson et al. (2004) reported the results of sampling conducted between 1996 and 2000 from 26 urban sites nationwide (n=711 samples), as part of the National Water-Quality Assessment (NAWQA) Program; the median TCE concentration was only 0.09 µg/L (detection frequency of 41%). One US study (U.S. EPA, 1977) reported much higher concentrations of TCE in surface water, up to 447 µg/L. These samples were collected in 1976/1977 from the vicinity of facilities producing and/or using methylchloroform, thus the concentrations reflect historical levels of TCE and are not considered to be representative of current conditions. Not enough information is reasonably available to provide a trend analysis of US surface water concentrations identified in published literature.

Systematic review also identified data from various other countries and regions, including China, Korea, United Kingdom, Russia, Portugal, Belgium, Greece, Japan, France, Italy, and Antarctica (see [*Data Extraction Tables for Environmental Monitoring Data. Docket: EPA-HQ-OPPT-2019-0500*]).

Table 2-11. Ambient Levels of TCE in U.S. Surface Water from Published Literature

Location Type	Site Information	Dates Sampled	N (Det. Freq.)	Concentration (µg/L)		Source	Data Quality Score
				Range	Central Tendency (Standard Deviation)		
Ambient	Anchorage, AK; Chester Creek (6 urban sampling sites)	1998-2001	11 (0)	All samples ND (<0.08)		(USGS, 2006)	Medium
	Nation-wide; Surface water for drinking water sources (rivers and reservoirs)	1999-2000	375 (0.008)	ND (<0.2) - 2.0	NR	(USGS, 2003)	Medium
	Nation-wide; Urban Rivers (26 sites, as part of the NAWQA Program)	1996-2000	711 (0.41)	NR	Median: 0.09	(Robinson et al., 2004)	Medium
	Boston, MA; Charles Rivers	1998-2000	29 (1)	NR - 17.3	Median: 1.17	(Robinson et al., 2004)	Medium
	Gulf of Mexico, near mouth of the Mississippi River and on the Louisiana Shelf (11 stations in the open ocean and coast representing both unpolluted and anthropogenic influences)	1980	11 (0.27)	ND - 0.05	NR	(Sauer, 1981)	Medium

Location Type	Site Information	Dates Sampled	N (Det. Freq.)	Concentration (µg/L)		Source	Data Quality Score
				Range	Central Tendency (Standard Deviation)		
	Two Bridges, NJ; Passaic River	1996-1998	10 (0.4)	NR	Median: 0.1	(Robinson et al., 2004)	Medium
	Eastern Pacific Ocean (California, US to Valparaiso, Chile)	1979-1981	30 (0.9)	ND (<0.0001) - 0.0007	Mean: 0.3 (0.002); Median: 0.0002	(Singh et al., 1983)	Medium
Near Facility (methyl-chloroform producer or user)	Baton Rouge, LA (Ethyl Corporation); Stream samples (surface) collected upstream and downstream of the outfall.	1976	2 (1.0)	0.4 - 37	NR	(U.S. EPA, 1977)	High
	Freeport, TX (Dow Chemical Plant); Stream samples (bottom and surface) collected from the receiving stream at the plant outfall and upstream and downstream of the outfall.	1976	6 (1.0)	0.9 - 126	NR	(U.S. EPA, 1977)	High
	Geismar, LA (Vulcan Materials Plant); 3 surface water samples collected from the receiving stream at the plant outfall and upstream and downstream of the outfall.	1976	3 (1.0)	5 - 74	NR	(U.S. EPA, 1977)	High
	Lake Charles, LA (PPG Industries); Stream samples (bottom and surface) collected from the receiving stream at the plant outfall and upstream and downstream of the outfall.	1976	5 (1.0)	29 - 447	Mean: 282 (156); Median: 353	(U.S. EPA, 1977)	High
	Auburn, WA (Boeing Company); Stream samples (surface) collected from the receiving stream at outfalls and/or upstream and downstream of the outfall.	1977	5 (1.0)	5 - 30	NR	(U.S. EPA, 1977)	High

NR = Not reported
ND = Not detected; detection limit reported in parethesis if reasonably available

2.2.6.2.3 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. Overall, there are 39 US states/territories with either a measured concentration or a predicted concentration; at the watershed level, there are 155 HUC-8 areas and 241 HUC-12 areas with either measured or predicted concentrations.

The monitoring stations co-located with facilities in the same HUC in the 2016 set were assessed for proximity to Superfund sites to determine if the Superfund sites could be contributing to TCE releases, and thus would not fall under the scope of this evaluation. No Superfund sites were identified within 5 miles of these sites.

Co-location of releasing facilities and monitoring sampling locations was examined for presence in the same watershed (HUC-8 and HUC-12). Co-location does not mean there is an upstream/downstream connection between release and sampling sites.

2.2.6.3 Assumptions and Key Sources of Uncertainty for Environmental Exposures

E-FAST 2014 estimates surface water concentrations at the point of release, without post-release accounting for environmental fate or degradation such as volatilization, biodegradation, photolysis, hydrolysis, or partitioning. Additionally, E-FAST does not estimate stream concentrations based on the potential for downstream transport and dilution. These considerations tend to lead to higher predicted surface water concentrations. Dilution is incorporated, but it is based on the stream flow applied. Therefore, there is uncertainty regarding the level of TCE that would be predicted downstream of a releasing facility or after accounting for potential volatilization from the water surface, which is dependent on the degree of mixing in a receiving water body. Despite these uncertainties, E-FAST is considered an appropriate screening model for near-field environmental concentrations.

Releases modeled using E-FAST 2014 were predicted based on engineering site-specific estimates, as based on DMR, TRI, and/or CDR databases. These data that form the basis for engineering estimates are self-reported by facilities subject to minimum reporting thresholds; therefore, they may not capture releases from certain facilities not meeting reporting thresholds (i.e., environmental releases may be underestimated).

The days of release applied in modeling have a direct impact on predicting surface water concentrations. The greater the number of release days assumed, the more the per-day release is diluted (assuming the same overall annual loading estimate). Both the higher release frequency and lower release frequency scenarios were based on estimates and were not based on actual facility reporting. Therefore, there is uncertainty regarding which release scenario is more likely, although the determination was made to consider only the higher release frequency for scenarios involving water treatment facilities.

Another key parameter in modeling is the applied stream flow distribution, which provides for the immediate dilution of the release estimate. The flow distributions are applied by selecting a facility-specific NPDES code in E-FAST. When site-specific or surrogate site-specific stream flow data were not reasonably available, flow data based on a representative industry sector were used in the assessment. This includes cases where a receiving facility for an indirect release could not be determined. In such cases, it is likely that the stream concentration estimates are higher than they would be if a facility-specific NPDES code was able to be applied, except in certain cases (e.g., NODES

associated with low-flow or intermittent streams or bays). Additionally, the stream flow data currently available in E-FAST 2014 are 15 to 30 years old. More recent flow data are available through the National Hydrological Dataset (NHD) but are not available within the E-FAST model.

With respect to the geospatial comparison of modeled estimates with ambient data obtained from WQX, one limitation is the accuracy of the latitudes and longitudes. The geographic coordinates for facilities were obtained from the FRS Interests geodatabase, which are assigned through various methods including photo-interpretation, address matching, and GPS. These are considered "Best Pick" coordinates. While EPA does assign accuracy values for each record based on the method used, the true accuracy of any individual point is unknown. Also, in some cases the receiving facilities for indirect releases could not be determined. In these cases, the location of the active releaser was mapped. As such, the co-location of facilities and monitoring sites may have been missed. As the number of unknown receiving facilities was small and most monitoring sites had samples with concentrations below the detection limit, this would have minimal impact on the watershed analysis. It is also important to note that only a few USGS-NWIS and STORET monitoring station locations aligned with the watersheds of the TCE -releasing facilities identified under the scope of this assessment, and the two co-located monitoring stations had samples with concentrations below the detection limit; therefore, no direct correlation can be made between them. While these data reflect low levels of trichlorethylene in ambient surface water samples, they cannot be interpreted as reflecting concentrations downstream of direct release sites, which could be higher than reported measured levels.

The WQP Tool contains data from USGS-NWIS and STORET databases, and is one of the largest environmental monitoring databases in the US; however, comprehensive information needed for data interpretation is not always reasonably available. For example, specific details regarding analytical techniques may be unclear, or not reported at all. As a result, there are uncertainties in the reported data that are difficult to quantify with regard to impacts on exposure estimates.

The quality of the data provided in the USGS-NWIS and STORET datasets varies, and some of the information provided is non-quantitative. While many individual sampling results were obtained from these datasets, the monitoring studies used to collect the data were not specifically designed to evaluate TCE distribution across the US. The reasonably 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 US. While these data reflect low levels of trichlorethylene in ambient surface water samples, they directly reflect sampling done in specific states.

2.2.6.4 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 2.2.2.1, 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) and its associated default and user-selected values and related uncertainties. As described in Section 2.2.6.3, there are uncertainties related to the ability of E-FAST 2014 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. Of note, as stated on the EPA website, "modeled

estimates of concentrations and doses are designed to reasonably overestimate exposures, for use in an exposure assessment in the absence of or with reliable monitoring data".

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 (see Table 2-10 and Table 2-11). Samples characterizing background levels in surface water ranged from non-detect (ND) to 17.3 µg/L, from both literature and the Water Quality Portal database. However, based on the modeling approach using site-specific releases and considering that the predicted concentrations reflect near-site concentrations prior to any additional fate and transport processes, these background exposure levels are not as useful in corroborating the modeling approach. Near-facility monitoring data collected between 1976 and 1977 show levels of TCE ranging from 0.4 to 447 µg/L, which encompasses the range of the modeled estimates across all OES (with the exception of two sites, which are associated with releases into a still water body) (see [*Aquatic Exposure Modeling Outputs from E-FAST. Docket: EPA-HQ-OPPT-2019-0500*]). However, these data are not attributable to any of the specific sites modeled, nor are they reflective of ongoing TCE use or release patterns.

Based on the above considerations, the aquatic exposure assessment scenarios have an overall moderate confidence.

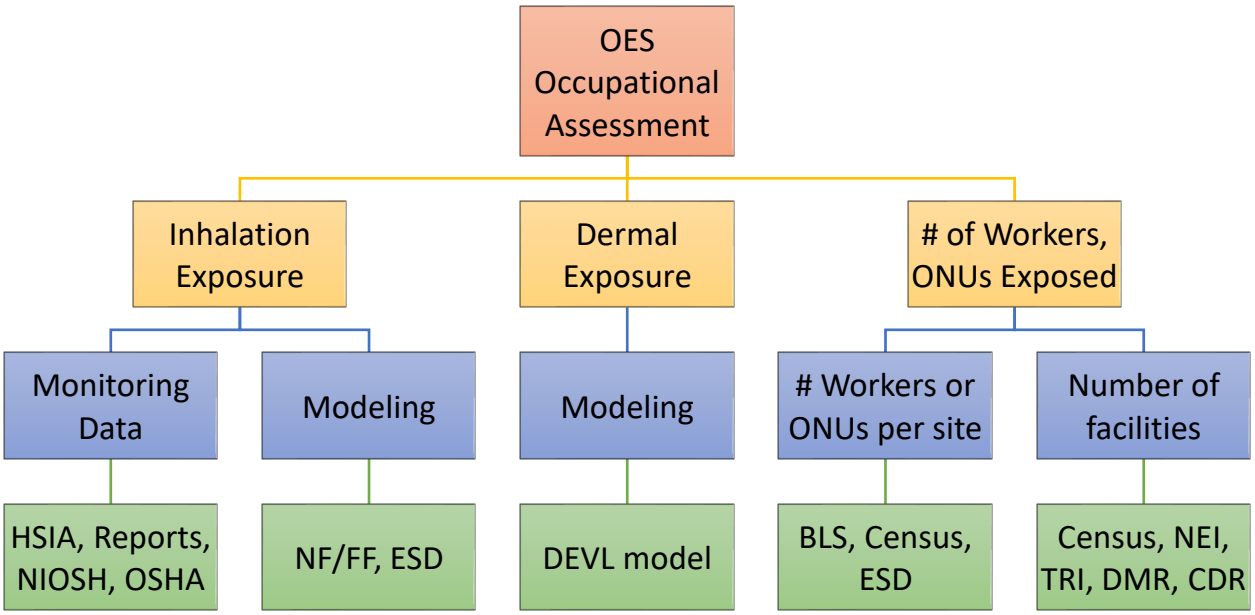
2.3 Human Exposures

2.3.1 Occupational Exposures

EPA categorized the conditions of use (COUs) listed in Table 1-4 into 18 Occupational Exposure Scenarios (OES). In this section, EPA describes its approach and methodology to estimating occupational exposures and provides a summary of results by OES for inhalation and dermal exposure, and also the number of workers and occupational non-users (ONUs) potentially exposed (Figure 2-5). ONUs include employees that work at the site where TCE is manufactured, processed, used, recycled, or disposed of,[9] but these employees do not directly handle the chemical and are therefore expected to have lower inhalation exposures and are not expected to have dermal exposures. For detailed occupational exposure results, see Appendix P of this document and the (i) "Exposure Assessment" section for each OES and (ii) "Dermal Exposure Assessment" section in [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500*]. An occupational exposure assessment includes the following components:

- **Inhalation Exposure:** Central tendency and high-end estimates of inhalation exposure to workers and occupational non-users by OES.
- **Dermal Exposure:** Occupational exposure scenarios were grouped into bins based on common characteristics and dermal exposure was estimated for workers for each of these bins
- **Number of Workers and Occupational Non-Users:** An estimate of the number of workers and occupational non-users (ONUs) potentially exposed to the chemical for each OES.

Figure 2-5: Components of an occupational assessment for each OES;[10] please refer to Section 2.2.2.2.2 for additional details on the approach and methodology for estimating number of facilities.



[9] Occupational exposures from distribution are considered within each condition of use.

[10] TRI = Toxics Release Inventory; DMR = Discharge Monitoring Report; NEI = National Emissions Inventory; CDR = Chemical Data Reporting; ELG = Effluent Limitation Guidelines; ESD = Emission Scenario Document; BLS = Bureau of Labor Statistics; NIOSH = National Institute of Occupational Safey and Health; OSHA = Occupational Safety and Health Administration; HSIA = Hallogenated Solvent Industry Alliance; NF/FF = Near-Field/Far-Field; DEVL = Dermal Exposure to Volatile Liquids.

2.3.1.1 Results for Occupational Assessment

In some cases, EPA identified relevant inhalation exposure monitoring data for a given OES. The quality of this monitoring data was assessed and EPA established an overall confidence for the data when integrated into the occupational exposure assessment.

Where monitoring data was reasonably available, EPA used this data to characterize central tendency and high end inhalation exposures. Where no inhalation monitoring data was identified, but inhalation exposure models were reasonably available, EPA estimated central tendency and high end exposures using only modeling approaches. If both, inhalation monitoring data and exposure models were reasonably available, where applicable, EPA presented central tendency and high end exposures using both. EPA did not identify any measured dermal exposure estimates. In all cases, the Dermal Exposure to Volatile Liquids (DEVL) model was used to estimate high-end and central tendency dermal exposures for workers in each OES.

In Table 2-12, EPA provides a summary for each of the 18 occupational exposure scenarios (OESs) by indicating whether monitoring data was reasonably available, how many data points were identified, the quality of the data, EPA's overall confidence in the data, whether the data was used to estimate inhalation exposures for workers and ONUs, and also whether EPA used modeling to estimate inhalation and dermal exposures for workers and ONUs.

In many cases, EPA did not have monitoring data to estimate inhalation exposure for ONUs. In some cases, this was addressed with the use of exposure models. However, approximately 50% of OESs do not contain inhalation exposure estimates for ONUs. In addition, EPA expects ONU exposures to be less than worker exposures. Dermal exposure for ONUs was not evaluated because these employees are not expected to be in direct contact with TCE.

A summary of inhalation exposure results based on monitoring data and exposure modeling for each OES is presented for workers in Table 2-13 and ONUs in Table 2-14. These tables provide a summary of time weighted average (TWA) inhalation exposure estimates as well as Acute Exposure Concentrations (AC), Average Daily Concentrations (ADC), and Lifetime Average Daily Concentrations (LADC). The ADC is used to characterize risks for chronic non-cancer health effects whereas the LADC is used for chronic cancer health effects. Additional details regarding AC, ADC, and LADC calculations are available in section 2.3.1.2.4, while EPA's approach and methodology for modeling inhalation exposure using the Near-Field/Far-Field mass balance model can be found in 2.3.1.2.3.

Table 2-15 includes a summary of central tendency and high-end dermal exposure results based on exposure modeling for workers in each OES. Occluded dermal exposures may occur when liquid becomes trapped between the skin and protective clothing (e.g., gloves). This may result in the liquid being unable to evaporate from the skin surface which may increase the quantity of liquid absorbed. Where applicable, both non-occluded and occluded exposure scenarios are assessed and the impact of various glove protection factors (PFs) are also estimated. EPA estimated the dermal retained dose for workers for each OES. These dose estimates assume one exposure event (applied dose) per work day and that approximately eight to thirteen percent[11] of the applied dose is absorbed through the skin. Central tendency and high-end dermal estimates also factor in ranged values for two variables, the surface area of contact, and the quantity remaining on the skin. Additional information on these variables can be found in section 2.3.1.2.5.

[11] The absorbed fraction is a function of indoor air speed, which differs for industrial and commercial settings.

EPA also estimated central tendency and high-end dermal retained doses for occluded scenarios for OESs where occlusion was reasonably expected to occur. Occluded scenarios are generally expected where workers come into contact with bulk liquid TCE during use in open systems (e.g., during solvent changeout in vapor degreasing) and not expected in closed-type systems (e.g., during connection/ disconnection of hoses used in loading of bulk containers in manufacturing).

Dermal exposure estimates are provided for each OES, where the OESs are "binned" based on the maximum possible exposure concentration (Y_{derm}), the likely level of exposure, and potential for occlusion. The exposure concentration is determined based on EPA's review of currently available products and formulations containing TCE. For example, EPA found that TCE concentration in degreasing formulations such as C-60 Solvent Degreaser can be as high as 100 percent. The calculated absorbed dose is low for all non-occluded scenarios since TCE evaporates quickly after exposure. Dermal exposure to liquid is not expected for occupational non-users, since they do not directly handle TCE. Additional details on EPA's approach and methodology for estimating dermal exposures for workers can be found in section 2.3.1.2.5.

Table 2-16 provides a summary of EPA's estimates for the total exposed workers and ONUs for each OES. In order to prepare these estimates, EPA first attempted to identify North American Industrial Classification (NAICS) codes associated with each OES. For these NAICS codes, EPA then reviewed Standard Occupational Classification (SOC) codes from the Bureau of Labor Statistics (BLS) and classified relevant SOC codes as workers or ONUs. All other SOC codes were assumed to represent occupations where exposure is unlikely.

Based on this combination of NAICS and SOC codes, EPA estimated the total number of workers and ONUs potentially exposed for the various OES. EPA also estimated the total number facilities associated with the NAICS codes previously identified based on data from the U.S. Census Bureau.

EPA then estimated the average number of workers and ONUs potentially exposed per site by dividing the total number of workers and ONUs by the total number of facilities. Finally, using EPA's estimates for the number of facilities using TCE, EPA was able to estimate the total number of workers and ONUs potentially exposed to TCE for reach OES.

Additional details on EPA's approach and methodology for estimating the number of facilities using TCE and the number of workers and ONUs potentially exposed to TCE can be found in sections 2.2.2.2.2 and 2.3.1.2.7, respectively.

Table 2-12: A summary for each of the 18 occupational exposure scenarios (OESs).

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 TCE.]

Occupational Exposure Scenario (OES)	Inhalation Exposure									Dermal Exposure Modeling[c]	
	Monitoring					Modeling		Overall Confidence			
	Monitoring Data	# Data Points	Data Quality Rating	Worker	ONU	Worker	ONU	Worker	ONU	Worker	ONU
Manufacturing	✓	16	H	✓	×	×	×	M to H	L	✓	-
Processing as a Reactant	✓	16	M	✓	×	×	×	L to M	L	✓	-
Formulation of Aerosol and Non-Aerosol Products	✓	33	H	✓	×	×	×	M	L	✓	-
Repackaging	✓	33	H	✓	×	×	×	M to H	L	✓	-
Batch Open-Top Vapor Degreasing	✓	123	M	✓	✓	✓	✓	M	M	✓	-
Batch Closed-Loop Vapor Degreasing	✓	19	H	✓	×	×	×	M to H	L	✓	-
Conveyorized Vapor Degreasing	✓	18	M	✓	×	✓	✓	L to M	L to M	✓	-
Web Vapor Degreasing	×	-	-	×	×	✓	✓	L to M	L to M	✓	-
Cold Cleaning	×	-	-	×	×	✓	✓	L to M	L to M	✓	-
Aerosol Applications[a]	×	-	-	×	×	✓	✓	M	M	✓	-
Metalworking Fluids	✓	3	H	✓	×	✓	×	L to M	L	✓	-
Adhesives, Sealants, Paints, and Coatings	✓	24	M to H; M[b]	✓	✓	×	×	L to M	L to M	✓	-
Other Industrial Uses	✓	16	M	✓	×	×	×	L to M	L	✓	-
Spot Cleaning and Wipe Cleaning	✓	8	M	✓	×	✓	✓	M	M	✓	-
Industrial Processing Aid	✓	34	H	✓	✓	×	×	M to H	L to M	✓	-
Commercial Printing and Copying	✓	20	M	✓	×	×	×	L to M	L	✓	-
Other Commercial Uses	✓	8	M	✓	×	✓	✓	M	M	✓	-
Process Solvent Recycling and Worker Handling of Wastes	✓	33	H	✓	×	×	×	M to H	L	✓	-

a. Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases
b. For Workers, data quality is M to H; For ONUs, data quality is is M.
c. 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.

Table 2-13: Summary of inhalation exposure results for Workers based on monitoring data and exposure modeling for each OES.

Occupational Exposure Scenario (OES)	Inhalation Monitoring (Worker, ppm)								Inhalation Modeling (Worker, ppm)							
	TWA		AC		ADC		LADC		TWA		AC		ADC		LADC	
	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT
Manufacturing	2.6	0.38	0.86	0.13	0.59	8.6E-02	0.30	3.4E-02	-	-	-	-	-	-	-	-
Processing as a Reactant	2.6	0.38	0.86	0.13	0.59	8.6E-02	0.30	3.4E-02	-	-	-	-	-	-	-	-
Formulation of Aerosol and Non-Aerosol Products	1.14	4.9E-04	0.38	1.6E-04	0.26	1.1E-04	0.13	4.5E-05	-	-	-	-	-	-	-	-
Repackaging	1.14	4.9E-04	0.38	1.6E-04	0.26	1.1E-04	0.13	4.5E-05	-	-	-	-	-	-	-	-
Batch Open-Top Vapor Degreasing	77.8	13.8	25.9	4.6	17.8	3.2	9.1	1.3	388.0	34.8	129.3	11.6	88.5	8.0	35.3	3.0
Batch Closed-Loop Vapor Degreasing	1.45	0.46	0.48	0.15	0.33	0.10	0.17	4.2E-02	-	-	-	-	-	-	-	-
Conveyorized Vapor Degreasing	48.3	32.4	16.1	10.8	11.0	7.4	5.7	2.9	3043.0	40.8	1014.3	13.6	694.8	9.3	275.2	5.3
Web Vapor Degreasing	-	-	-	-	-	-	-	-	14.1	5.9	4.7	2.0	3.2	1.4	1.3	0.51
Cold Cleaning	-	-	-	-	-	-	-	-	57.2	3.3	19.1	1.1	13.1	0.76	5.2	0.28
Aerosol Applications[a]	-	-	-	-	-	-	-	-	24.0	7.6	8.0	2.5	5.5	1.7	2.2	0.65
Metalworking Fluids	75.4	69.7	25.1	23.2	17.2	15.9	8.8	6.3	0.26	0.07	0.09	0.02	0.06	0.02	0.03	0.01
Adhesives, Sealants, Paints, and Coatings	39.5	4.6	13.2	1.5	9.0	1.1	4.6	0.42	-	-	-	-	-	-	-	-
Other Industrial Uses	2.6	0.38	0.86	0.13	0.59	0.09	0.30	3.4E-02	-	-	-	-	-	-	-	-
Spot Cleaning and Wipe Cleaning	2.9	0.38	0.95	0.13	0.67	0.09	0.34	3.6E-02	2.8	0.96	0.92	0.32	0.65	0.23	0.26	0.08
Industrial Processing Aid[b]	12.8	4.3	6.4	2.13	4.39	1.5	2.2	0.58	-	-	-	-	-	-	-	-
Commercial Printing and Copying	2.1	8.5E-02	0.70	0.03	0.48	0.02	0.25	7.7E-03	-	-	-	-	-	-	-	-
Other Commercial Uses	2.9	0.38	0.95	0.13	0.67	0.09	0.34	3.6E-02	2.8	0.96	0.92	0.32	0.65	0.23	0.26	8.4E-02
Process Solvent Recycling and Worker Handling of Wastes	1.1	4.9E-04	0.38	1.6E-04	0.26	1.1E-04	0.13	4.5E-05	-	-	-	-	-	-	-	-

a. Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases
b. Exposure for this OES is based on a 12 hr TWA; all other exposures based on 8 hr TWAs

Table 2-14: Summary of inhalation exposure results for ONUs based on monitoring data and exposure modeling for each OES.
[Note: for many cases EPA was not able to estimate inhalation exposure for ONUs, but EPA expects these to be lower than inhalation exposure for Workers.]

Occupational Exposure Scenario (OES)	Inhalation Monitoring (ONU, ppm)								Inhalation Modeling (ONU, ppm)							
	TWA		AC		ADC		LADC		TWA		AC		ADC		LADC	
	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT	HE	CT
Manufacturing	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Processing as a Reactant	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Formulation of Aerosol and Non-Aerosol Products	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Repackaging	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Batch Open-Top Vapor Degreasing	9.1	1.1	3.0	0.37	2.1	0.25	1.06	0.10	237.0	18.1	79.0	6.0	54.0	4.1	21.1	1.5
Batch Closed-Loop Vapor Degreasing	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Conveyorized Vapor Degreasing	-	-	-	-	-	-	-	-	1878.0	23.3	626.0	7.8	428.8	5.3	168.3	3.6
Web Vapor Degreasing	-	-	-	-	-	-	-	-	9.6	3.1	3.2	1.0	2.2	0.71	0.87	0.27
Cold Cleaning	-	-	-	-	-	-	-	-	34.7	1.8	11.6	0.61	7.9	0.42	3.1	0.15
Aerosol Applications[a]	-	-	-	-	-	-	-	-	1.0	0.14	0.35	4.7E-02	0.24	3.2E-02	0.09	1.2E-02
Metalworking Fluids	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Adhesives, Sealants, Paints, and Coatings	1.0	0.94	0.33	0.31	0.23	0.21	0.12	8.5E-02	-	-	-	-	-	-	-	-
Other Industrial Uses	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Spot Cleaning and Wipe Cleaning	-	-	-	-	-	-	-	-	1.8	0.48	0.58	0.16	0.41	0.11	0.16	4.2E-02
Industrial Processing Aid[b]	2.9	1.3	1.5	0.66	0.99	0.45	0.51	0.18	-	-	-	-	-	-	-	-
Commercial Printing and Copying	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other Commercial Uses	-	-	-	-	-	-	-	-	1.8	0.48	0.58	0.16	0.41	0.11	0.16	4.2E-02
Process Solvent Recycling and Worker Handling of Wastes	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

a. Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases
b. Exposure for this OES is based on a 12 hr TWA; all other exposures based on 8 hr TWAs

Table 2-15: A summary of dermal retained dose for Workers based on exposure modeling for each OES

[Note: an explanation of each Bin is provided in Table 2-21; where applicable, both non-occluded and occluded exposure scenarios are assessed and the impact of various glove protection factors (PFs) are also estimated; estimates assume one exposure event per work day and that approximately eight to thirteen percent of the applied dose is absorbed through the skin (see Section 2.3.1.2.5 for additional details).]

Occupational Exposure Scenario (OES)		Bin	Max TCE Weight Fraction (Max Y_{derm})	Non-Occluded Worker Dermal Retained Dose (mg/day)								Occluded Worker Dermal Retained Dose (mg/day)	
				No Gloves (PF = 1)		Protective Gloves (PF = 5)		Protective Gloves (PF = 10)		Protective Gloves (PF = 20)			
				HE	CT	HE	CT	HE	CT	HE	CT	HE	CT
Manufacturing		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Processing as a Reactant		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Formulation of Aerosol and Non-Aerosol Products		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Repackaging		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Batch Open-Top Vapor Degreasing		2	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	2,247	749
Batch Closed-Loop Vapor Degreasing		2	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	2,247	749
Conveyorized Vapor Degreasing		2	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	2,247	749
Web Vapor Degreasing		2	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	2,247	749
Cold Cleaning		2	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	2,247	749
Aerosol Applications[a]		3	1.0	184.36	61.45	36.87	12.29	18.44	6.15	-	-	-	-
Metalworking Fluids		4	0.8	147.49	49.16	29.50	9.83	14.75	4.92	-	-	1,798	599
Adhesives, Sealants, Paints, and Coatings	Industrial	3	0.9	165.92	55.31	33.18	11.06	16.59	5.53	-	-	-	-
	Commercial	3	0.9	260.50	86.83	52.10	17.37	26.05	8.68	-	-	-	-
Other Industrial Uses		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Spot Cleaning and Wipe Cleaning		4	1.0	289.44	96.48	57.89	19.30	28.94	9.65	-	-	2,247	749
Industrial Processing Aid		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-
Commercial Printing and Copying		4	0.35	101.30	33.77	20.26	6.75	10.13	3.38	-	-	786	262
Other Commercial Uses		4	1.0	289.44	96.48	57.89	19.30	28.94	9.65	-	-	2,247	749
Process Solvent Recycling and Worker Handling of Wastes		1	1.0	184.36	61.45	36.87	12.29	18.44	6.15	9.22	3.07	-	-

a. Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases

Table 2-16: Summary of the total number of workers and ONUs potentially exposed to TCE for each OES

[Note: EPA's approach and methodology for estimating the number of facilities using TCE and the number of workers and ONUs potentially exposed to TCE can be found in sections 2.2.2.2.2 and 2.3.1.2.7, respectively.]

Occupational Exposure Scenario (OES)	Total Exposed Workers	Total Exposed ONUs	Total Exposed	Number of Facilities[b]	Notes
Manufacturing	350	170	530	5	
Processing as a Reactant	120 to 6,100	55 to 2,900	180 to 9,000	5 to 440	
Formulation of Aerosol and Non-Aerosol Products	306	99	405	19	
Repackaging	36	12	48	22	
Batch Open-Top Vapor Degreasing	4,922	2,889	7,810	194	
Batch Closed-Loop Vapor Degreasing	50	18	68	4	
Conveyorized Vapor Degreasing	92	32	130	8	
Web Vapor Degreasing	-	-	-	1	EPA does not have data to estimate the total workers and ONUs exposed to TCE.
Cold Cleaning	660	400	1,100	13	
Aerosol Applications[a]	14,200	1,690	15,900	4,366	
Metalworking Fluids	-	-	-	-	Based on ESD on the Use of Metalworking Fluids, EPA estimates 46 Workers and 2 ONUs per site; the number of sites that use TCE-based metalworking fluids is unknown to EPA.
Adhesives, Sealants, Paints, and Coatings	3,000	1,400	4,400	70	
Other Industrial Uses	2,300	1,000	3,300	49	
Spot Cleaning and Wipe Cleaning	244,000	25,300	269,000	63,748	Based on assumption of 100% market penetration.
Industrial Processing Aid	310	140	450	18	
Commercial Printing and Copying	-	-	-	-	Based on NIOSH HHE, EPA estimates 44 Workers and 74 ONUs per site; EPA does not have data to estimate total number of sites
Other Commercial Uses	-	-	-	-	EPA does not have data to estimate the total workers and ONUs exposed to TCE
Process Solvent Recycling and Worker Handling of Wastes	380	140	520	30	

a. Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases

b. Please refer to Table 2-3 for notes related to estimates for Number of Facilities using TCE.

2.3.1.2 Approach and Methodology

2.3.1.2.1 General

EPA provided occupational exposure results representative of central tendency conditions and high-end conditions. A central tendency is assumed to be representative of occupational exposures in the center of the distribution for a given condition of use. For risk evaluation, EPA used the 50th percentile (median), mean (arithmetic or geometric), mode, or midpoint values of a distribution as representative of the central tendency scenario. EPA's preference is to provide the 50th percentile of the distribution. However, if the full distribution is not known, EPA may assume that the mean, mode, or midpoint of the distribution represents the central tendency depending on the statistics available for the distribution.

A high-end is assumed to be representative of occupational exposures that occur at probabilities above the 90th percentile but below the exposure of the individual with the highest exposure (U.S. EPA, 1992). For risk evaluation, EPA provided high-end results at the 95th percentile. If the 95th percentile is not reasonably available, EPA used a different percentile greater than or equal to the 90th percentile but less than or equal to the 99.9th percentile, depending on the statistics available for the distribution. If the full distribution is not known and the preferred statistics are not reasonably available, EPA estimated a maximum or bounding estimate in lieu of the high-end.

For occupational exposures, EPA used measured or estimated air concentrations to calculate exposure concentration metrics required for risk assessment, such as average daily concentration (ADC) and lifetime average daily concentration (LADC). These calculations require additional parameter inputs, such as years of exposure, exposure duration and frequency, and lifetime years. EPA estimated exposure concentrations from monitoring data, modeling, or occupational exposure limits.

For the final exposure result metrics, each of the input parameters (e.g., air concentrations, working years, exposure frequency, lifetime years) may be a point estimate (i.e., a single descriptor or statistic, such as central tendency or high-end) or a full distribution. EPA considered three general approaches for estimating the final exposure result metrics:

- **Deterministic calculations:** EPA used combinations of point estimates of each parameter to estimate a central tendency and high-end for each final exposure metric result.
- **Probabilistic (stochastic) calculations:** EPA used Monte Carlo simulations using the full distribution of each parameter to calculate a full distribution of the final exposure metric results and selecting the 50th and 95th percentiles of this resulting distribution as the central tendency and high-end, respectively.
- **Combination of deterministic and probabilistic calculations:** EPA had full distributions for some parameters but point estimates of the remaining parameters. For example, EPA used Monte Carlo modeling to estimate exposure concentrations, but only had point estimates of exposure duration and frequency, and lifetime years.

EPA follows the following hierarchy in selecting data and approaches for assessing inhalation exposures:

1. Monitoring data:
    a. Personal and directly applicable
    b. Area and directly applicable
    c. Personal and potentially applicable or similar
    d. Area and potentially applicable or similar

2. Modeling approaches:
    a. Surrogate monitoring data
    b. Fundamental modeling approaches
    c. Statistical regression modeling approaches
3. Occupational exposure limits:
    a. Company-specific OELs (for site-specific exposure assessments, e.g., there is only one manufacturer who provides to EPA their internal OEL but does not provide monitoring data)
    b. OSHA PEL
    c. Voluntary limits (ACGIH TLV, NIOSH REL, Occupational Alliance for Risk Science (OARS) workplace environmental exposure level (WEEL) [formerly by AIHA])

EPA assessed TCE occupational exposure of the following two receptor categories: male or female workers who are ≥16 years or older; and, female workers of reproductive age (≥16 years to less than 50 years).

2.3.1.2.2 Inhalation Exposure Monitoring Data

EPA reviewed workplace inhalation monitoring data collected by government agencies such as OSHA and NIOSH, monitoring data found in published literature (i.e., personal exposure monitoring data and area monitoring data), and monitoring data submitted via public comments. Studies were evaluated using the evaluation strategies laid out in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018b).

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

2.3.1.2.3 Inhalation Exposure Modeling

EPA's inhalation exposure modeling is based on a near-field/far-field approach (NF/FF) (Nicas, 2009), where a vapor generation source located inside the near-field diffuses into the surrounding environment. The NF/FF model has been extensively peer-reviewed, it is extensively used, and results of the model have been compared with measured data. The comparison indicated that the model and measured values agreed to within a factor of about three (U.S. EPA, 2014b).

EPA considers workers at the facility who neither directly perform activities near the TCE source area nor regularly handle TCE to be occupational non-users (ONU). Workers that are directly handling TCE and/or perform activities near sources of TCE are in the near field and are called workers throughout this report. The near-field is reported to be conceptualized as a volume of air within one-meter in any direction of the worker's head and the far-field comprised the remainder of the room (Tielemans et al., 2008). The source area/exposure zone could be judged by several factors such as the chemical inventory, ventilation of the facility, vapor pressure and emission potential of the chemical, process temperature, size of the room, job tasks, and modes of chemical dispersal from activities (Leblanc et al., 2018).

Esmen et al. (1979) indicated that the assignment of zones is a professional judgment and not a scientific exercise. Applications of the NF/FF model are illustrated in Figure 2-6.

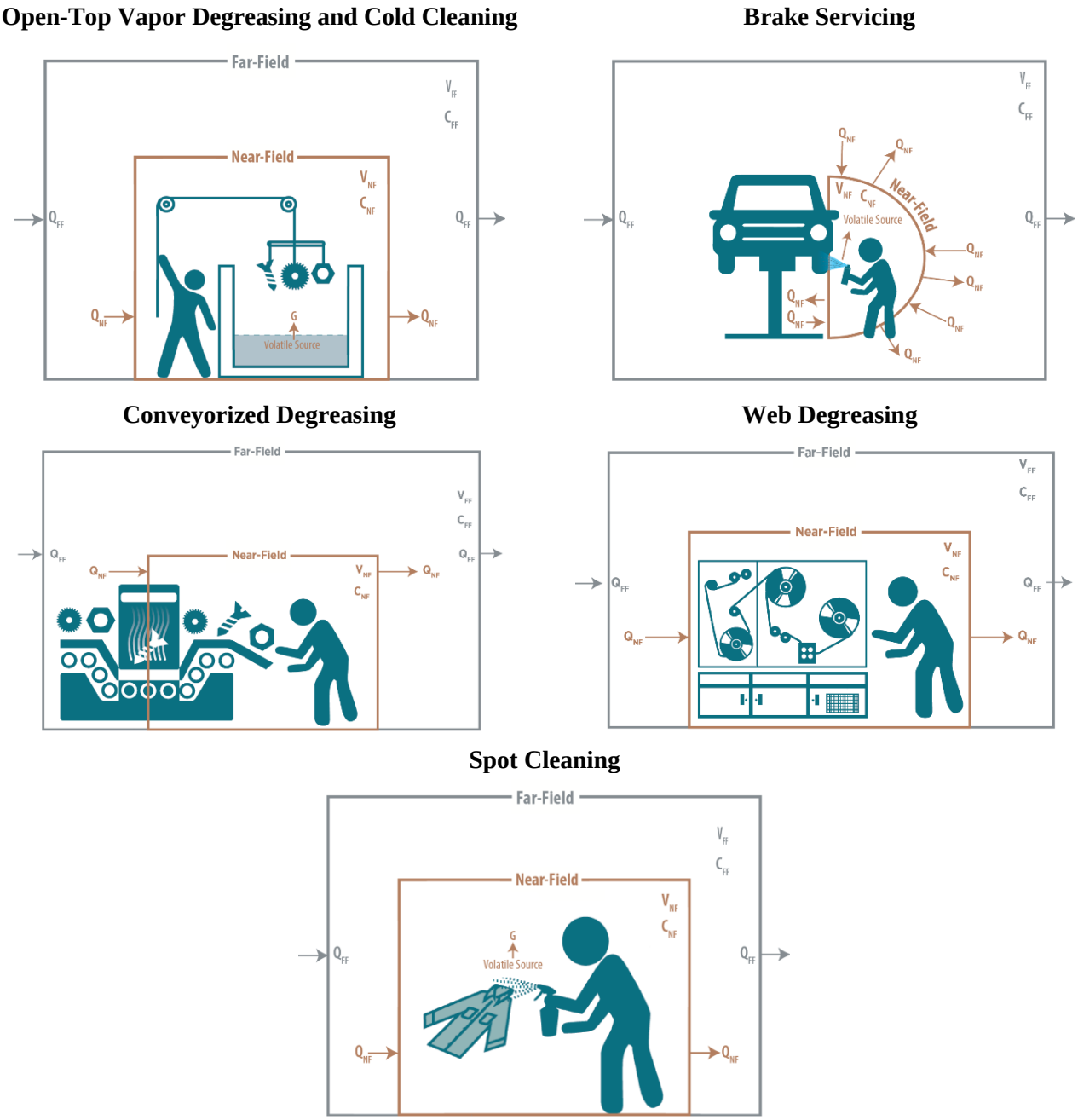


Figure 2-6: Illustrative applications of the NF/FF model to various exposure scenarios.

As the figures show, volatile TCE becomes airborne in the near-field, resulting in worker exposures at a TCE concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, (denoted by G in Figure 2-6), into the near-field, whose volume is denoted by V_{NF}. In the case of brake servicing, there is no evaporation rate. Rather, the aerosol degreaser is assumed to immediately become airborne in the near-field zone upon application, resulting in a sudden rise in the near-field concentration.

The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-

field, resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outside air. The NF/FF model design equations are presented below.

Near-Field Mass Balance

$$V_{NF}\frac{dC_{NF}}{dt} = C_{FF}Q_{NF} - C_{NF}Q_{NF} + G$$

Far-Field Mass Balance

$$V_{FF}\frac{dC_{FF}}{dt} = C_{NF}Q_{NF} - C_{FF}Q_{NF} - C_{FF}Q_{FF}$$

Where:

- V_{NF} = near-field volume;
- V_{FF} = far-field volume;
- Q_{NF} = near-field ventilation rate;
- Q_{FF} = far-field ventilation rate;
- C_{NF} = average near-field concentration;
- C_{FF} = average far-field concentration;
- G = average vapor generation rate; and
- t = elapsed time.

For details on the modeling approach and model equations, please refer to Appendix K; Appendix L; and Appendix M.

2.3.1.2.4 Acute and Chronic Inhalation Exposure Estimates

This report assesses TCE exposures to workers in occupational settings, presented as time weighted average (TWA). The TWA exposures are then used to calculate acute exposure (AC), average daily concentration (ADC) for chronic, non-cancer risks, and lifetime average daily concentration (LADC) for chronic, cancer risks.

Acute workplace exposures are assumed to be equal to the contaminant concentration in air (TWA):

$$AC = \frac{C \times ED}{AT_{acute}}$$

Where:

- AC = acute exposure concentration
- C = contaminant concentration in air (TWA)
- ED = exposure duration (hr/day)
- AT_{acute} = acute averaging time (24 hrs)

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

$$\text{ADC or LADC} = \frac{\text{C} \times \text{ED} \times \text{EF} \times \text{WY}}{\text{AT or AT}_\text{c}}$$

$$AT = WY \times 365 \frac{day}{yr} \times 24 \frac{hr}{day}$$

$$AT_C = LT \times 365 \frac{day}{yr} \times 24 \frac{hr}{day}$$

Where:

ADC = Average daily concentration used for chronic non-cancer risk calculations
LADC = Lifetime average daily concentration used for chronic cancer risk calculations
ED = Exposure duration (hr/day)
EF = Exposure frequency (day/yr)
WY = Working years per lifetime (yr)
AT = Averaging time (hr) for chronic, non-cancer risk
AT_C = Averaging time (hr) for cancer risk
AWD = Annual working days (day/yr)
f = Fractional working days with exposure (unitless)
LT = Lifetime years (yr) for cancer risk

The parameter values in Table 2-17 are used to calculate each of the above acute or chronic exposure estimates. Where exposure is calculated using probabilistic modeling, the AC, ADC, and LADC calculations are integrated into the Monte Carlo simulation. Where multiple values are provided for ED and EF, it indicates that EPA may have used different values for different conditions of use. The rationale for these differences are described below in this section (also see Appendix J for example calculations).

Table 2-17: Parameter Values for Calculating Inhalation Exposure Estimates

Parameter Name	Symbol	Value	Unit
Exposure Duration	ED	8 or 24	hr/day
Exposure Frequency	EF	250	days/yr
Working years	WY	31 (50th percentile) 40 (95th percentile)	years
Lifetime Years, cancer	LT	78	years
Averaging Time, non-cancer	AT	271,560 (central tendency)[a] 350,400 (high-end)[b]	hr
Averaging Time, cancer	AT_c	683,280	hr

[a] Calculated using the 50th percentile value for working years (WY)
[b] Calculated using the 95th percentile value for working years (WY)

Exposure Duration (ED)

EPA generally uses an exposure duration of 8 hours per day for averaging full-shift exposures with an exception of spot-cleaning. Operating hours for spot cleaning were assessed as 2 to 5 hours/day.

Exposure Frequency (EF)

EPA generally uses an exposure frequency of 250 days per year with the following exception: spot

cleaning. EPA assumed spot cleaners may operate between five and six days per week and 50 to 52 weeks per year resulting in a range of 250 to 312 annual working days per year (AWD). Taking into account fractional days exposed (f) resulted in an exposure frequency (EF) of 249 at the 50th percentile and 313 at the 95th percentile.

EF is expressed as the number of days per year a worker is exposed to the chemical being assessed. In some cases, it may be reasonable to assume a worker is exposed to the chemical on each working day. In other cases, it may be more appropriate to estimate a worker's exposure to the chemical occurs during a subset of the worker's annual working days. The relationship between exposure frequency and annual working days can be described mathematically as follows:

$$EF = f \times AWD$$

Where:

EF = exposure frequency, the number of days per year a worker is exposed to the chemical (day/yr)
f = fractional number of annual working days during which a worker is exposed to the chemical (unitless)
AWD = annual working days, the number of days per year a worker works (day/yr)

BLS (2016) provides data on the total number of hours worked and total number of employees by each industry NAICS code. These data are available from the 3- to 6-digit NAICS level (where 3-digit NAICS are less granular and 6-digit NAICS are the most granular). Dividing the total, annual hours worked by the number of employees yields the average number of hours worked per employee per year for each NAICS.

EPA has identified approximately 140 NAICS codes applicable to the multiple conditions of use for the ten chemicals undergoing risk evaluation. For each NAICS code of interest, EPA looked up the average hours worked per employee per year at the most granular NAICS level available (i.e., 4-digit, 5-digit, or 6-digit). EPA converted the working hours per employee to working days per year per employee assuming employees work an average of eight hours per day. The average number of days per year worked, or AWD, ranges from 169 to 282 days per year, with a 50th percentile value of 250 days per year. EPA repeated this analysis for all NAICS codes at the 4-digit level. The average AWD for all 4-digit NAICS codes ranges from 111 to 282 days per year, with a 50th percentile value of 228 days per year. 250 days per year is approximately the 75th percentile. In the absence of industry- and TCE-specific data, EPA assumes the parameter *f* is equal to one for all conditions of use.

Working Years (WY)

EPA has developed a triangular distribution for working years. EPA has defined the parameters of the triangular distribution as follows:

- Minimum value: BLS CPS tenure data with current employer as a low-end estimate of the number of lifetime working years: 10.4 years;
- Mode value: The 50th percentile tenure data with all employers from SIPP as a mode value for the number of lifetime working years: 31 years; and
- Maximum value: The maximum average tenure data with all employers from SIPP as a high-end estimate on the number of lifetime working years: 40 years.

This triangular distribution has a 50th percentile value of 31 years and a 95th percentile value of 40 years.

EPA uses these values for central tendency and high-end ADC and LADC calculations, respectively.

The BLS (U.S. BLS, 2014) provides information on employee tenure with *current employer* obtained from the Current Population Survey (CPS). CPS is a monthly sample survey of about 60,000 households that provides information on the labor force status of the civilian non-institutional population age 16 and over; CPS data are released every two years. The data are available by demographics and by generic industry sectors but are not available by NAICS codes.

The U.S. Census' (U.S. Census Bureau, 2019) Survey of Income and Program Participation (SIPP) provides information on lifetime tenure with all employers. SIPP is a household survey that collects data on income, labor force participation, social program participation and eligibility, and general demographic characteristics through a continuous series of national panel surveys of between 14,000 and 52,000 households (U.S. Census Bureau, 2019). EPA analyzed the 2008 SIPP Panel Wave 1, a panel that began in 2008 and covers the interview months of September 2008 through December 2008 (U.S. Census Bureau, 2019). For this panel, lifetime tenure data are available by Census Industry Codes, which can be cross-walked with NAICS codes.

SIPP data include fields for the industry in which each surveyed, employed individual works (TJBIND1), worker age (TAGE), and years of work experience *with all employers* over the surveyed individual's lifetime.[12] Census household surveys use different industry codes than the NAICS codes used in its firm surveys, so these were converted to NAICS using a published crosswalk (U.S. Census Bureau, 2013). EPA calculated the average tenure for the following age groups: 1) workers age 50 and older; 2) workers age 60 and older; and 3) workers of all ages employed at time of survey. EPA used tenure data for age group "50 and older" to determine the high-end lifetime working years, because the sample size in this age group is often substantially higher than the sample size for age group "60 and older". For some industries, the number of workers surveyed, or the *sample size*, was too small to provide a reliable representation of the worker tenure in that industry. Therefore, EPA excluded data where the sample size is less than five from our analysis.

Table 2-18 summarizes the average tenure for workers age 50 and older from SIPP data. Although the tenure may differ for any given industry sector, there is no significant variability between the 50th and 95th percentile values of average tenure across manufacturing and non-manufacturing sectors.

Table 2-18: Overview of Average Worker Tenure from U.S. Census SIPP (Age Group 50+)

Industry Sectors	Working Years			
	Average	50th Percentile	95th Percentile	Maximum
All industry sectors relevant to the 10 chemicals undergoing risk evaluation	35.9	36	39	44
Manufacturing sectors (NAICS 31-33)	35.7	36	39	40
Non-manufacturing sectors (NAICS 42-81)	36.1	36	39	44

Source: (U.S. Census Bureau, 2019)
Note: Industries where sample size is less than five are excluded from this analysis.

[12] To calculate the number of years of work experience EPA took the difference between the year first worked (TMAKMNYR) and the current data year (i.e., 2008). EPA then subtracted any intervening months when not working (ETIMEOFF).

BLS CPS data provides the median years of tenure that wage and salary workers had been with their current employer. Table 2-19 presents CPS data for all demographics (men and women) by age group from 2008 to 2012. To estimate the low-end value on number of working years, EPA uses the most recent (2014) CPS data for workers age 55 to 64 years, which indicates a median tenure of 10.4 years with their current employer. The use of this low-end value represents a scenario where workers are only exposed to the chemical of interest for a portion of their lifetime working years, as they may change jobs or move from one industry to another throughout their career.

Table 2-19: Median Year of Tenure with Current Employer by Age Group.

Age	January 2008	January 2010	January 2012	January 2014
16 years and over	4.1	4.4	4.6	4.6
16 to 17 years	0.7	0.7	0.7	0.7
18 to 19 years	0.8	1.0	0.8	0.8
20 to 24 years	1.3	1.5	1.3	1.3
25 years and over	5.1	5.2	5.4	5.5
25 to 34 years	2.7	3.1	3.2	3.0
35 to 44 years	4.9	5.1	5.3	5.2
45 to 54 years	7.6	7.8	7.8	7.9
55 to 64 years	9.9	10.0	10.3	10.4
65 years and over	10.2	9.9	10.3	10.3

Source: (U.S. BLS, 2014).

Lifetime Years (LT)

EPA assumes a lifetime of 78 years for all worker demographics.

2.3.1.2.5 Dermal Exposure Modeling

Dermal exposure data was not reasonably available for the OESs in the assessment. Because TCE is a volatile liquid that readily evaporates from the skin, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids (DEVL) Model. See Appendix H of the [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500)*] for the development and underlying research of this 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 TCE based on a theoretical framework provided by Kasting (Kasting and Miller, 2006). The amount of liquid on the skin is adjusted by the weight fraction of TCE in the liquid to which the worker is exposed.

The DEVL is used to assess occupational dermal exposure scenarios because the exposure duration is typically not known across a wide variety of worker activities, and the model's event-based approach allows exposure estimation using the number of exposure events, rather than exposure duration. Further, the model can account for the impact of glove use in occupational settings.

EPA estimated workers' dermal exposure to TCE for the industrial and commercial occupational exposure scenarios (OESs) considering evaporation of liquid from the surface of the hands and use with and without gloves. The OSHA recommends employers utilize the hierarchy of controls for reducing or

removing hazardous exposures. The most effective controls are elimination, substitution, or engineering controls. Gloves are the last course of worker protection in the hierarchy of controls and should only be considered when process design and engineering controls cannot reduce workplace exposure to an acceptable level.

Vapor absorption during dermal exposure requires that TCE be capable of achieving a sufficient concentration in the media at the temperature and atmospheric pressure of the scenario under evaluation to provide a significant driving force for skin penetration. Because TCE is a volatile liquid (VP = 73.46 mmHg and 25°C), the dermal absorption of TCE depends on the type and duration of exposure. Where exposure is not occluded, only a fraction of TCE that comes into contact with the skin will be absorbed as the chemical readily evaporates from the skin. 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 TCE liquids trapped inside the gloves, inhibiting the evaporation of TCE and increasing the exposure duration. EPA collected and reviewed available SDSs (Safety Data Sheets) to inform the evaluation of gloves used with TCE in liquid and aerosol form at varying concentrations.

Trichloroethylene in liquid form at 99-100% concentration is expected to be used in both industrial and commercial settings. For industrial scenarios using this form of TCE, the following OESs are expected; Manufacture of TCE, Processing as a Reactant, Industrial Processing Aid, Formulation of Aerosol and Non Aerosol Products, Repackaging, Process Solvent Recycling, Batch Open Top Vapor Degreasing, Batch Closed-Loop Vapor Degreasing, Conveyorized Vapor Degreasing, and Web Vapor Degreasing.

For trichlorethylene in liquid form at 99-100% concentration an SDS from Mallinckrodt Baker Inc. recommended neoprene gloves and an SDS from Solvents Australia PTY. LTD. recommended the use of gloves made from rubber, PVC, or nitrile (U.S. EPA, 2017c).

Commercial OESs where TCE in liquid form at 99-100% concentration is expected includes Spot Cleaning, Wipe Cleaning, and Carpet Cleaning. An SDS for an R.R. Street & Co. cleaning agent recommended wearing Viton ® [Butyl-rubber], PVA, or Barrier ™ gloves. Two gun wipe cleaning agent manufacturers A.V.W. Inc. and G.B. Distributors recommend Viton or Neoprene gloves and polyethylene, neoprene, or PVA gloves, respectively (U.S. EPA, 2017c).

For Aerosol Degreasing and Aerosol Lubricants applications, TCE is used in a range of concentrations in aerosol form. An SDS for a 90-100% TCE aerosol degreasing agent from Brownells, Inc. recommended using PVA gloves and an SDS for a 45-55% TCE aerosol brake parts cleaner from Zep Manufacturing Co. recommended using Viton® gloves (U.S. EPA, 2017c).

Metalworking Fluids and Adhesives, Sealants, Paints, and Coatings typically contain a maximum TCE concentration of 80-90%. An SDS from LPS Laboratories presented a tap and die fluid at 80-90% TCE concentration and recommended using Viton® [Butyl-rubber], Silver Shield®[PE and EVOH laminate] and PVA gloves. An SDS for a 75-90% TCE adhesive from Rema Tip Top recommended using Neoprene, Butyl-rubber, or nitrile rubber (U.S. EPA, 2017c).

EPA did not find any SDSs with applicable use towards commercial printing and copying applications.

To assess exposure, EPA used the *Dermal Exposure to Volatile Liquids* Model to calculate the dermal retained dose for both non-occluded and occluded scenarios. The equation modifies the *EPA 2-Hand Dermal Exposure to Liquids Model* 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. Default

PF values, which vary depending on the type of glove used and the presence of employee training program, are shown in Table 2-20:

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

Where:

- S is the surface area of contact: 535 cm^2 (central tendency) and 1,070 cm^2 (high end), representing the total surface area of one and two hands, respectively.
- Q_u is the quantity remaining on the skin: 1.4 mg/cm^2-event (central tendency) and 2.1 mg/cm^2-event (high-end). This is the high-end default value used in the EPA dermal models ((U.S. EPA, 2013a).
- 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 (1 event per day)
- f_{abs} is the fraction of applied mass that is absorbed (Default for TCE: 0.08 for industrial facilities and 0.13 for commercial facilities)
- PF is the glove protection factor (Table 2-20)

The steady state fractional absorption (f_{abs}) for TCE is estimated to be 0.08 in industrial facilities with higher indoor wind flows or 0.13 in commercial facilities with lower indoor wind speeds based on a theoretical framework provided by Kasting and Miller (2006) (Kasting and Miller, 2006), meaning approximately 8 or 13 percent of the applied dose is absorbed through the skin following exposure, from industrial and commercial settings, respectively. However, there is a large standard deviation in the experimental measurement, which is indicative of the difficulty in spreading a small, rapidly evaporating dose of TCE evenly over the skin surface.

Table 2-20: 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

Source: (Marquart et al., 2017)

To streamline the dermal exposure assessment, EPA grouped the various OESs based on characteristics known to effect dermal exposure such as the maximum weight fraction of TCE could be present in that scenario, open or closed system use of TCE, and large or small-scale use. Four different groups or "bins" were created based on this analysis (Table 2-21).

1335 **Table 2-21: EPA grouped dermal exposures associated with the various OESs into four bins.**

Bin #	Description
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. EPA assesses the following glove use scenarios for Bin 1 conditions of use: No gloves used: Operators in these industrial uses, while working around closed-system equipment, may not wear gloves or may wear gloves for abrasion protection or gripping that are not chemical resistant. Gloves used with a protection factor of 5, 10, and 20: Operators may wear chemical-resistant gloves when taking quality control samples or when connecting and disconnecting hoses during loading/unloading activities. EPA assumes gloves may offer a range of protection, depending on the type of glove and employee training provided. Scenarios not assessed: EPA does not assess occlusion as workers in these industries are not likely to come into contact with bulk liquid TCE that could lead to chemical permeation under the cuff of the glove or excessive liquid contact time leading to chemical permeation through the glove.
2	**Bin 2** covers industrial degreasing uses, which are not closed systems. For these uses, there is greater opportunity for dermal exposure during activities such as charging and draining degreasing equipment, drumming waste solvent, and removing waste sludge. EPA assesses the following glove use scenarios for Bin 2 conditions of use: No gloves used: Due to the variety of shop types in these uses the actual use of gloves is uncertain. EPA assumes workers may not wear gloves or may wear gloves for abrasion protection or gripping that are not chemical resistant during routine operations such as adding and removing parts from degreasing equipment. Gloves used with a protection factor of 5, 10, and 20: Workers may wear chemical-resistant gloves when charging and draining degreasing equipment, drumming waste solvent, and removing waste sludge. EPA assumes gloves may offer a range of protection, depending on the type of glove and employee training provided. Occluded Exposure: Occlusion may occur when workers are handling bulk liquid TCE when charging and draining degreasing equipment, drumming waste solvent, and removing waste sludge that could lead to chemical permeation under the cuff of the glove or excessive liquid contact time leading to chemical permeation through the glove.
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. EPA assesses the following glove use scenarios for Bin 3 conditions of use: No gloves used: Actual use of gloves in this use is uncertain. EPA assumes workers may not wear gloves or may wear gloves for abrasion protection or gripping that are not chemical resistant during routine aerosol applications. Gloves used with a protection factor of 5 and 10: Workers may wear chemical-resistant gloves when applying aerosol products. EPA assumes the commercial facilities in Bin 3 do not offer activity-specific training on donning and doffing gloves. Scenarios not assessed: EPA does not assess glove use with protection factors of 20 as EPA assumes chemical-resistant gloves used in these industries would either not be accompanied by training or be accompanied by basic employee training, but not activity-specific training. EPA does not assess occlusion for aerosol applications because TCE formulations are often supplied in an aerosol spray can and contact with bulk liquid is unlikely. EPA also does not assess occlusion for non-aerosol niche uses because the potential for occlusion is unknown

4	**Bin 4** covers commercial activities of similar maximum concentration. Most of these uses are uses as spot cleaners or in wipe cleaning, and/or uses expected to have direct dermal contact with bulk liquids. EPA assesses the following glove use scenarios for Bin 4 conditions of use: No gloves used: Actual use of gloves in this use is uncertain. EPA assumes workers may not wear gloves during routine operations (e.g., spot cleaning). Gloves used with a protection factor of 5 and 10: Workers may wear chemical-resistant gloves when charging and draining solvent to/from machines, removing and disposing sludge, and maintaining equipment. EPA assumes the commercial facilities in Bin 4 do not offer activity-specific training on donning and doffing gloves. Occluded Exposure: Occlusion may occur when workers are handling bulk liquid TCE when charging and draining solvent to/from machines, removing and disposing sludge, and maintaining equipment that could lead to chemical permeation under the cuff of the glove or excessive liquid contact time leading to chemical permeation through the glove. Scenarios not assessed: EPA does not assess glove use with protection factors of 20 as EPA assumes chemical-resistant gloves used in these industries would either not be accompanied by training or be accompanied by basic employee training, but not activity-specific training.

2.3.1.2.6 Consideration of Engineering Controls and Personal Protective Equipment

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

Respiratory Protection

OSHA's Respiratory Protection Standard (29 CFR § 1910.134) requires employers in certain industries 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.[13] 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 Table 2-22) and refer to the level of respiratory protection

[13] OSHA does not require controls to be used unless a hazard assessment determines that the hazard is significant enough to require mitigation.

that a respirator or class of respirators is expected to provide to employees when the employer implements a continuing, effective respiratory protection program.

The United States has several regulatory and non-regulatory exposure limits for TCE: an OSHA PEL of 100 ppm 8-hour TWA (OSHA, 2019), a NIOSH Recommended Exposure Limit (REL) of 2 ppm (as a 60-minute ceiling for TCE usage as an anesthetic) and 25 ppm (as a 10-hour TWA for other exposures) (NIOSH, 2019) and an American Conference of Government Industrial Hygienists (ACGIH) 8-hour TLV of 10 ppm and a short-term limit of 25 ppm (ATSDR, 2019). 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 include air-purifying respirators with organic vapor cartridges. Table 2-22 can be used as a guide to show the protectiveness of each category of respirator. Based on the APF, inhalation exposures may be reduced by a factor of 5 to 10,000, when workers and occupational non-users are using respiratory protection.

The respirators should be used when effective engineering controls are not feasible as per OSHA's 29 CFR § 1910.132. The knowledge of the range of respirator APFs is intended to assist employers in selecting the appropriate type of respirator that could provide a level of protection needed for a specific exposure scenario. Table 2-22 lists the range of APFs for respirators. The complexity and burden of wearing respirators increases with increasing APF. The APFs are not to be assumed to be interchangeable for any conditions of use, any workplace, or any worker or ONU.

Table 2-22: 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)

2.3.1.2.7 Number of Workers and Occupational Non-Users Exposed

This section summarizes the methods that EPA used to estimate the number of workers who are potentially exposed to TCE in each of its conditions of use. The method consists of the following steps:

1. Identify the North American Industry Classification System (NAICS) codes for the industry sectors associated with each condition of use.
2. Estimate total employment by industry/occupation combination using the Bureau of Labor Statistics' Occupational Employment Statistics data (U.S. BLS, 2016).
3. Refine the estimates based on BLS Occupational Employment Statistics data where they are not sufficiently granular by using the U.S. Census' (U.S. Census Bureau, 2015) Statistics of U.S. Businesses (SUSB) data on total employment by 6-digit NAICS.
4. Estimate the percentage of employees likely to be using TCE instead of other chemicals (i.e., the market penetration of TCE in the condition of use).
5. Estimate the number of sites and number of potentially exposed employees per site.
6. Estimate the number of potentially exposed employees within the condition of use.

Step 1: Identifying Affected NAICS Codes

As a first step, EPA identified NAICS industry codes associated with each condition of use. EPA generally identified NAICS industry codes for a condition of use by:

- Querying the U.S. Census Bureau's *NAICS Search* tool using keywords associated with each condition of use to identify NAICS codes with descriptions that match the condition of use.
- Referencing EPA Generic Scenarios (GS's) and Organisation for Economic Co-operation and Development (OECD) Emission Scenario Documents (ESDs) for a condition of use to identify NAICS codes cited by the GS or ESD.
- Reviewing Chemical Data Reporting (CDR) data for the chemical, identifying the industrial sector codes reported for downstream industrial uses, and matching those industrial sector codes to NAICS codes using Table D-2 provided in the CDR reporting instructions.

Each condition of use section in the main body of this report identifies the NAICS codes EPA identified for the respective condition of use.

Step 2: Estimating Total Employment by Industry and Occupation

BLS's (U.S. BLS, 2016) Occupational Employement Statistics data provide employment data for workers in specific industries and occupations. The industries are classified by NAICS codes (identified previously), and occupations are classified by Standard Occupational Classification (SOC) codes.

Among the relevant NAICS codes (identified previously), EPA reviewed the occupation description and identified those occupations (SOC codes) where workers are potentially exposed to TCE. Table 2-23 shows the SOC codes EPA classified as occupations potentially exposed to TCE. These occupations are classified into workers (W) and occupational non-users (O). All other SOC codes are assumed to represent occupations where exposure is unlikely.

Table 2-23: SOCs with Worker and ONU Designations for All Conditions of Use Except Dry Cleaning

SOC	Occupation	Designation
11-9020	Construction Managers	O
17-2000	Engineers	O
17-3000	Drafters, Engineering Technicians, and Mapping Technicians	O

19-2031	Chemists	O
19-4000	Life, Physical, and Social Science Technicians	O
47-1000	Supervisors of Construction and Extraction Workers	O
47-2000	Construction Trades Workers	W
49-1000	Supervisors of Installation, Maintenance, and Repair Workers	O
49-2000	Electrical and Electronic Equipment Mechanics, Installers, and Repairers	W
49-3000	Vehicle and Mobile Equipment Mechanics, Installers, and Repairers	W
49-9010	Control and Valve Installers and Repairers	W
49-9020	Heating, Air Conditioning, and Refrigeration Mechanics and Installers	W
49-9040	Industrial Machinery Installation, Repair, and Maintenance Workers	W
49-9060	Precision Instrument and Equipment Repairers	W
49-9070	Maintenance and Repair Workers, General	W
49-9090	Miscellaneous Installation, Maintenance, and Repair Workers	W
51-1000	Supervisors of Production Workers	O
51-2000	Assemblers and Fabricators	W
51-4020	Forming Machine Setters, Operators, and Tenders, Metal and Plastic	W
51-6010	Laundry and Dry-Cleaning Workers	W
51-6020	Pressers, Textile, Garment, and Related Materials	W
51-6030	Sewing Machine Operators	O
51-6040	Shoe and Leather Workers	O
51-6050	Tailors, Dressmakers, and Sewers	O
51-6090	Miscellaneous Textile, Apparel, and Furnishings Workers	O
51-8020	Stationary Engineers and Boiler Operators	W
51-8090	Miscellaneous Plant and System Operators	W
51-9000	Other Production Occupations	W

W = worker designation
O = ONU designation

For dry cleaning facilities, due to the unique nature of work expected at these facilities and that different workers may be expected to share among activities with higher exposure potential (e.g., unloading the dry cleaning machine, pressing/finishing a dry cleaned load), EPA made different SOC code worker and ONU assignments for this condition of use. Table 2-24 summarizes the SOC codes with worker and ONU designations used for dry cleaning facilities.

Table 2-24: SOCs with Worker and ONU Designations for Dry Cleaning Facilities

SOC	Occupation	Designation
41-2000	Retail Sales Workers	O
49-9040	Industrial Machinery Installation, Repair, and Maintenance Workers	W
49-9070	Maintenance and Repair Workers, General	W
49-9090	Miscellaneous Installation, Maintenance, and Repair Workers	W
51-6010	Laundry and Dry-Cleaning Workers	W
51-6020	Pressers, Textile, Garment, and Related Materials	W
51-6030	Sewing Machine Operators	O
51-6040	Shoe and Leather Workers	O
51-6050	Tailors, Dressmakers, and Sewers	O
51-6090	Miscellaneous Textile, Apparel, and Furnishings Workers	O

W = worker designation
O = ONU designation

After identifying relevant NAICS and SOC codes, EPA used BLS data to determine total employment by industry and by occupation based on the NAICS and SOC combinations. For example, there are 110,640 employees associated with 4-digit NAICS 8123 (*Drycleaning and Laundry Services*) and SOC 51-6010 (*Laundry and Dry-Cleaning Workers*).

Using a combination of NAICS and SOC codes to estimate total employment provides more accurate estimates for the number of workers than using NAICS codes alone. Using only NAICS codes to estimate number of workers typically result in an overestimate, because not all workers employed in that industry sector will be exposed. However, in some cases, BLS only provide employment data at the 4-digit or 5-digit NAICS level; therefore, further refinement of this approach may be needed (see next step).

Step 3: Refining Employment Estimates to Account for lack of NAICS Granularity

The third step in EPA's methodology was to further refine the employment estimates by using total employment data in the U.S. Census Bureau's (U.S. Census Bureau, 2015) SUSB. In some cases, BLS OES's occupation-specific data are only available at the 4-digit or 5-digit NAICS level, whereas the SUSB data are available at the 6-digit level (but are not occupation-specific). Identifying specific 6-digit NAICS will ensure that only industries with potential TCE exposure are included. As an example, OES data are available for the 4-digit NAICS 8123 *Drycleaning and Laundry Services*, which includes the following 6-digit NAICS:

- NAICS 812310 Coin-Operated Laundries and Drycleaners;
- NAICS 812320 Drycleaning and Laundry Services (except Coin-Operated);
- NAICS 812331 Linen Supply; and
- NAICS 812332 Industrial Launderers.

In this example, only NAICS 812320 is of interest. The Census data allow EPA to calculate employment in the specific 6-digit NAICS of interest as a percentage of employment in the BLS 4-digit NAICS.

The 6-digit NAICS 812320 comprises 46 percent of total employment under the 4-digit NAICS 8123. This percentage can be multiplied by the occupation-specific employment estimates given in the BLS Occupational Employment Statistics data to further refine our estimates of the number of employees with potential exposure.

Table 2-25 illustrates this granularity adjustment for NAICS 812320.

Table 2-25: Estimated Number of Potentially Exposed Workers and ONUs under NAICS 812320.

NAICS	SOC CODE	SOC Description	Occupation Designation	Employment by SOC at 4-digit NAICS level	% of Total Employment	Estimated Employment by SOC at 6-digit NAICS level
8123	41-2000	Retail Sales Workers	O	44,500	46.0%	20,459
8123	49-9040	Industrial Machinery Installation, Repair, and Maintenance Workers	W	1,790	46.0%	823
8123	49-9070	Maintenance and Repair Workers, General	W	3,260	46.0%	1,499
8123	49-9090	Miscellaneous Installation, Maintenance, and Repair Workers	W	1,080	46.0%	497

8123	51-6010	Laundry and Dry-Cleaning Workers	W	110,640	46.0%	50,867
8123	51-6020	Pressers, Textile, Garment, and Related Materials	W	40,250	46.0%	18,505
8123	51-6030	Sewing Machine Operators	O	1,660	46.0%	763
8123	51-6040	Shoe and Leather Workers	O	Not Reported for this NAICS Code		
8123	51-6050	Tailors, Dressmakers, and Sewers	O	2,890	46.0%	1,329
8123	51-6090	Miscellaneous Textile, Apparel, and Furnishings Workers	O	0	46.0%	0
Total Potentially Exposed Employees				**206,070**		**94,740**
Total Workers						**72,190**
Total Occupational Non-Users						**22,551**

Note: numbers may not sum exactly due to rounding.
W = worker
O = occupational non-user
Source: (U.S. Census Bureau, 2015); (U.S. BLS, 2016)

Step 4: Estimating the Percentage of Workers Using TCE Instead of Other Chemicals

In the final step, EPA accounted for the market share by applying a factor to the number of workers determined in Step 3. This accounts for the fact that TCE may be only one of multiple chemicals used for the applications of interest. EPA did not identify market penetration data any conditions of use. In the absence of market penetration data for a given condition of use, EPA assumed TCE may be used at up to all sites and by up to all workers calculated in this method as a bounding estimate. This assumes a market penetration of 100%. Market penetration is discussed for each condition of use in the main body of this report.

Step 5: Estimating the Number of Workers per Site

EPA calculated the number of workers and occupational non-users in each industry/occupation combination using the formula below (granularity adjustment is only applicable where SOC data are not available at the 6-digit NAICS level):

Number of Workers or ONUs in NAICS/SOC (Step 2) × Granularity Adjustment Percentage (Step 3) = Number of Workers or ONUs in the Industry/Occupation Combination

EPA then estimated the total number of establishments by obtaining the number of establishments reported in the U.S. Census Bureau's SUSB (U.S. Census Bureau, 2015) data at the 6-digit NAICS level.

EPA then summed the number of workers and occupational non-users over all occupations within a NAICS code and divided these sums by the number of establishments in the NAICS code to calculate the average number of workers and occupational non-users per site.

Step 6: Estimating the Number of Workers and Sites for a Condition of Use

EPA estimated the number of workers and occupational non-users potentially exposed to TCE and the number of sites that use TCE in a given condition of use through the following steps:

1. Obtaining the total number of establishments by:

a. Obtaining the number of establishments from SUSB (U.S. Census Bureau, 2015) at the 6-digit NAICS level (Step 5) for each NAICS code in the condition of use and summing these values; or
b. Obtaining the number of establishments from the Toxics Release Inventory (TRI), Discharge Monitoring Report (DMR) data, National Emissions Inventory (NEI), or literature for the condition of use.

2. Estimating the number of establishments that use TCE by taking the total number of establishments from Item 1 and multiplying it by the market penetration factor from Step 4.
3. Estimating the number of workers and occupational non-users potentially exposed to TCE by taking the number of establishments calculated in Item 2 and multiplying it by the average number of workers and occupational non-users per site from Step 5.

2.3.1.3 Assumptions and Key Sources of Uncertainty for Occupational Exposures

2.3.1.3.1 Number of Workers

There are a number of uncertainties surrounding the estimated number of workers potentially exposed to TCE, 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 TCE. Manufacturers and importers are only required to report if they manufactured or imported TCE in excess of 25,000 pounds at a single site during any calendar year; as such, CDR may not capture all sites and workers associated with any given chemical.

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 TCE for the assessed applications. 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 TCE 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 TCE 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.

2.3.1.3.2 Analysis of Exposure Monitoring Data

This report uses existing worker exposure monitoring data to assess exposure to TCE during several conditions of use. To analyze the exposure data, EPA categorized each PBZ data point 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 TCE are categorized as "worker" and samples for employees that are expected to have the lower exposure and do not directly handle TCE 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 TCE 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 be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. Similarly, OSHA CEHD are obtained from OSHA inspections, which may be the result of worker complaints, and may provide exposure results that may generally exceed the industry average.

Some scenarios have limited exposure monitoring data in literature, if any. Where there are few data points available, it is unlikely the results will be representative of worker exposure across the industry. In cases where there was no exposure monitoring data, EPA may have used monitoring data from similar conditions of use as surrogate. While these conditions of use have similar worker activities contributing to exposures, it is unknown that the results will be fully representative of worker exposure across different conditions of use.

Where sufficient data were reasonably available, the 95th and 50th percentile exposure concentrations were calculated using reasonably available data. The 95th percentile exposure concentration is intended to represent a high-end exposure level, while the 50th percentile exposure concentration represents typical exposure level. The underlying distribution of the data, and the representativeness of the reasonably available data, are not known. Where discrete data was not reasonably available, EPA used reported statistics (i.e., median, mean, 90th percentile, etc.). Since EPA could not verify these values, there is an added level of uncertainty.

EPA calculated ADC and LADC values assuming workers and ONUs are regularly exposed during their entire working lifetime, which likely results in an overestimate. Individuals may change jobs during the course of their career such that they are no longer exposed to TCE, and that actual ADC and LADC values become lower than the estimates presented.

2.3.1.3.3 Near-Field/Far-Field Model Framework

The near-field/far-field approach is used as a framework to model inhalation exposure for many conditions of use. The following describe uncertainties and simplifying assumptions generally associated with this modeling approach:

- There is some degree of uncertainty associated with each model input parameter. In general, the model inputs were determined based on review of reasonably available literature. Where the distribution of the input parameter is known, a distribution is assigned to capture uncertainty in the Monte Carlo analysis. Where the distribution is unknown, a uniform distribution is often used. The use of a uniform distribution will capture the low-end and high-end values but may not accurately reflect actual distribution of the input parameters.
- The model assumes the near-field and far-field are well mixed, such that each zone can be approximated by a single, average concentration.

- All emissions from the facility are assumed to enter the near-field. This assumption will overestimate exposures and risks in facilities where some emissions do not enter the airspaces relevant to worker exposure modeling.
- The exposure models estimate airborne concentrations. Exposures are calculated by assuming workers spend the entire activity duration in their respective exposure zones (i.e., the worker in the near-field and the occupational non-user in the far-field). Since vapor degreasing and cold cleaning involve automated processes, a worker may actually walk away from the near-field during part of the process and return when it is time to unload the degreaser. As such, assuming the worker is exposed at the near-field concentration for the entire activity duration may overestimate exposure.
- For certain TCE applications (e.g., vapor degreasing and cold cleaning), TCE 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 TCE condition of use.

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

Vapor Degreasing and Cold Cleaning Models

The OTVD, conveyorized vapor 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 eight 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 TCE 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 degreasing applications involving TCE.
- The TCE Use Dossier (U.S. EPA, 2017c) presented 16 different aerosol degreasing formulations containing TCE. For each Monte Carlo iteration, the model determines the TCE concentration in product by selecting one of 16 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 TCE Use Dossier (U.S. EPA, 2017c) were provided as ranges. For each Monte Carlo iteration the model selects a TCE concentration within

the range of concentrations using a uniform distribution. In reality, the TCE concentration in the formulation may be more consistent than the range provided.

Spot Cleaning Model

The multi-zone spot cleaning model also uses a near-field/far-field approach. Specific uncertainties associated with the spot cleaning scenario are presented below:

- The model assumes a use rate based on estimates of the amount of TCE-based spot cleaner sold in California and the number of textile cleaning facilities in California (IRTA, 2007). It is uncertain if this distribution is representative of other geographic locations in the U.S.
- The model assumes a facility floor area based on data from (CARB, 2006) and King County (Whittaker and Johanson, 2011). It is unknown how representative the area is of "typical" spot cleaning facilities. Therefore, these assumptions may result in an overestimate or underestimate of worker exposure during spot cleaning.
- Many of the model input parameters were obtained from (Von Grote et al., 2003), which is a German study. Aspects of the U.S. spot cleaning facilities may differ from German facilities. However, it is not known whether the use of German data will under- or over-estimate exposure.

2.3.1.3.4 Modeled Dermal Exposures

The *Dermal Exposure to Volatile Liquids Model* is used to estimate dermal exposure to TCE 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. Additionally, the studies used to obtain the underlying values of the quantity remaing on the skin (Q_u) did not take into consideration the fact that liquid retention on the skin may vary with individuals and techniques of application on and removal from the hands. Also the data used were developed from three kinds of oils; therefore, the data may not be applicable to other liquids. Based on the uncertainties described above, EPA has a medium level of confidence in the assessed baseline exposure. See Appendix H of the [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500)*] for the development and underlying research of this model.

2.3.1.3.5 Summary of Overall Confidence in Inhalation Exposure Estimates

Table 2-26 provides a summary of EPA's overall confidence in its inhalation exposure estimates for each of the Occupational Exposure Scenarios assessed.

Table 2-26: Summary of overall confidence in inhalation exposure estimates by OES.

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
Manufacturing	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.
Processing as a Reactant	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Formulation of Aerosol and Non-Aerosol Products	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 33 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium.
Repackaging	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 33 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.
Batch Open-Top Vapor Degreasing	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 123 data points from 16 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium. EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that the underlying methodologies used to estimate these emissions in the 2014 NEI are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Batch Closed-Loop Vapor Degreasing	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 19 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.
Conveyorized Vapor Degreasing	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 18 data points from 2 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for three total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Web Vapor Degreasing	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2011 NEI were only found for one unit, and the underlying methodologies used to estimate the emission is unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Cold Cleaning	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for ten total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Various model parameters were derived from a CARB brake service study and TCE

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	concentration data for 16 products representative of the OES. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium.
Metalworking Fluids	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 3 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include limited dataset (3 data points from 1 site), and the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low. EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. Data from the 2011 Emission Scenario Document on the Use of Metalworking Fluids was used to estimate inhalation exposures. The primary limitations of the exposure outputs from this model include the uncertainty of the representativeness of these data toward the true distribution of inhalation for all TCE uses for the industries and sites covered by this scenario, and the difference between the modeling data and monitoring data. Added uncertainties include that the underlying TCE concentration used in the metalworking fluid was assumed from one metalworking fluid product. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium.
Adhesives, Sealants, Paints, and Coatings	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 22 data points from 2 sources, and the data quality ratings from systematic review for these data were medium to high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to medium to low. For the ONU inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 2 data points from 1 source, and the data quality ratings from systematic review for the data point was high. The primary limitations of this data is the limited dataset (two

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	data points from 1 site), and the uncertainty of the representativeness of this data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Other Industrial Uses	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Spot Cleaning and Wipe Cleaning	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 8 data points from 2 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low. EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Various model parameters were derived from a CARB study. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that the underlying methodologies used to obtain the values in the CARB study, as well as the assumed TCE concentration in the spot cleaning product. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low. Despite these limitations, the modeling and monitoring results match each other very closely. Therefore, the overall confidence is medium.
Industrial Processing Aid	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the

Occupational Exposure Scenario (OES)	Overall Confidence in Inhalation Exposure Estimates
	12-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 30 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 12-hr TWA data in this scenario is medium to high. For the ONU inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 4 data points from 1 source, and the data quality ratings from systematic review for the data point was high. The primary limitations of this single data point include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 12-hr TWA data in this scenario is medium to low.
Commercial Printing and Copying	EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 20 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include a limited dataset, and the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.
Other Commercial Uses	EPA did not identify any inhalation exposure monitoring data related to this OES. EPA assumes the exposure sources, routes, and exposure levels are similar to those for the Spot Cleaning and Wipe Cleaning OES.
Process Solvent Recycling and Worker Handling of Wastes	EPA did not identify any inhalation exposure monitoring data related to waste handling/recycling. EPA assumes the exposure sources, routes, and exposure levels are similar to those for the Repackaging OES.

2.3.2 Consumer Exposures

TCE can be found in consumer and commercial products that are available for purchase at common retailers and can therefore result in exposures to household consumers (i.e., receptors who use a product directly) and bystanders (i.e., receptors who are a non-product users that are incidentally exposed to the product or article) (U.S. EPA, 2017c, h).

2.3.2.1 Consumer Conditions of Use Evaluated

Conditions of use associated with consumer exposure were described in the Problem Formulation (U.S. EPA, 2018d). The availability of TCE in consumer products was determined through the development of EPA's 2017 Market and Use Report (U.S. EPA, 2017h) and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: TCE (U.S. EPA, 2017c). Additional online research was undertaken following Problem Formulation to confirm TCE concentrations and compile a comprehensive list of products that may be available to consumers for household use. These resources were used to select the most appropriate product-specific inputs (e.g., weight fraction and formulation type) associated with each consumer condition of use.

Table 2-27 lays out consumer condition of use categories and associated product subcategories evaluated for TCE. Based on additional research, conditions of use may be described in more detail (e.g., formulation type, specific product type) when compared to the tables presented in the Problem Formulation (U.S. EPA, 2018d). Any differences between the displayed categories and those presented in the Problem Formulation are described in the footnotes.

Table 2-27. Evaluated Consumer Conditions of Use and Products for TCE

Life Cycle Stage	Category	Product Subcategory	Form[1]	No. of Products Utilized in Modeling[1]
Use	Solvents for Cleaning and Degreasing	Brake & Parts Cleaner[2]	Aerosol	4
		Electronic Degreaser/Cleaner[3]	Aerosol	9
		Electronic Degreaser/Cleaner[3]	Liquid	1
		Aerosol Spray Degreaser/Cleaner	Aerosol	8
		Liquid Degreaser/Cleaner[3]	Liquid	2
		Gun Scrubber[4]	Aerosol	2
		Gun Scrubber[4]	Liquid	1
		Mold Release	Aerosol	2
		Tire Cleaner[5]	Aerosol	2
		Tire Cleaner[5]	Liquid	1
	Lubricants and Greases	Tap & Die Fluid	Aerosol	1
		Penetrating Lubricant[6]	Aerosol	5
	Adhesives and Sealants	Solvent-based Adhesive & Sealant	Liquid	3
		Mirror-edge Sealant	Aerosol	1
		Tire Repair Cement/Sealer	Liquid	5
	Cleaning and Furniture Care Products [11]	Carpet Cleaner	Liquid	1
		Spot Remover[7]	Aerosol	1
		Spot Remover[7]	Liquid	4
	Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings[8]	Aerosol	1
	Apparel and Footwear Care Products	Shoe Polish	Aerosol	1
	Other Consumer Uses	Fabric Spray[9]	Aerosol	1
		Film Cleaner	Aerosol	2
		Hoof Polish	Aerosol	1
		Pepper Spray	Aerosol	2
		Toner Aid[10]	Aerosol	1

Life Cycle Stage	Category	Product Subcategory	Form[1]	No. of Products Utilized in Modeling[1]

[1] Form was determined based on the specific products identified as representative of the associated product subcategories. Please see Supplemental File [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*] for the full list of representative products.
[2] The brake cleaner subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the automotive care products category; however, the same brake cleaning conditions of use are now associated with the broader solvents for cleaning and degreasing category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the brake cleaner product(s) and not a broader category of use.
[3] Liquid degreaser/cleaner and electronic degreaser/cleaner (aerosol and liquid) were not specifically named in the Problem Formulation as a potential consumer subcategories. They were added due to product availability based on the additional research noted above that helped to differentiate specific product forms (i.e., liquid or aerosol) and types.
[4] The gun scrubber subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the other consumer uses category; however, the same gun scrubber conditions of use are now associated with the broader solvents for cleaning and degreasing category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the gun scrubber product(s) and not a broader category of use.
[5] Tire cleaner products / subcategories of use were not specifically called out in the Problem Formulation; however, such products were identified in the 2017 Use and Market Report and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: TCE (U.S. EPA, 2017c) and fit within the broader Solvents for Cleaning and Degreasing category.
[6] Based on additional research into the specific product(s) associated with the broader lubricants and greases category, the subcategory name was updated from penetrating lubricant to lubricant.
[7] The spot remover subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the laundry and dishwashing products category; however, the same spot remover conditions of use are now associated with the cleaning and furniture care products category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the spot remover product(s) and not a broader category of use.
[8] Note that this subcategory is referred to as "clear protective coating spray" in U.S. EPA (2014b) and as "spray fixative" in the TCE Significant New Use Rule (80 FR 47441).
[9] Fabric spray (specifically an anti-fray spray) was added following Problem Formulation based on identification in the final 2014 TCE Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).
[10] The toner aid subcategory was listed in Table 2-3 of the Problem Formulation as being associated with the Ink, toner, and colorant products category; however, the toner aid use is not like use of a toner or pigment; therefore, the same toner aid condition of use is now associated with the other consumer use category. This change does not impact evaluated conditions of use, as the evaluated product scenarios are based on the toner aid product(s) and not a broader category of use.
[11] Note that the problem formulation described "cleaning wipes" as a condition of use for this category. However, that referred to the application of a product that is then wiped off, rather than a pre-wet towelette. A number of consumer conditions of use involve wipe cleaning and are described in detail in Section 2.3.2.6.2 as leading to dermal contact with impeded evaporation.

2.3.2.2 Consumer Exposure Routes Evaluated

Inhalation and dermal exposures are evaluated for acute exposure scenarios, i.e., those resulting from short-term or daily exposures. Chronic exposure scenarios resulting from long-term use of household consumer products were not evaluated. In general, the frequency of product use was considered to be too low to create chronic risk concerns. Although high-end frequencies of consumer use are up to 50 times per year, reasonably available toxicological data is based on either single or continuous TCE exposure and it is unknown whether these use patterns are expected to be clustered or intermittent (e.g. one time per week). There is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures. Therefore, EPA cannot fully rule out that consumers at the high-end frequency of use could possibly be at risk for chronic hazard effects, however it is expected to be unlikely.

2.3.2.2.1 Inhalation

The acute exposure via inhalation is the most significant route of exposure for consumer exposure scenarios for users and bystanders. This is in line with EPA's 2014 TSCA Work Plan Chemical Risk Assessment, which evaluated acute inhalation exposure to consumers and bystanders from degreasing and arts & crafts uses (U.S. EPA, 2014b). EPA evaluated inhalation exposures for consumers and bystanders for all consumer conditions of use.

Background levels of TCE in indoor and outdoor air are not assessed in this assessment; therefore, there is a potential for underestimating consumer inhalation exposures, particularly for populations living near a facility emitting TCE or living in a home with other sources of TCE, such as TCE-containing products stored in the home. Similarly, inhalation exposures were evaluated on a product-specific basis and are based on use of a single product type within a day, not multiple products.

2.3.2.2.2 Dermal

EPA assessed dermal exposures to TCE from consumer uses. Instantaneous exposures to skin are expected to evaporate before significant dermal absorption occurs based on TCE's physical chemical properties which include the vapor pressure, water solubility and log $K_{ow.}$ The log K_{ow} estimate is 0.8% absorption and 99.2% volatilization and is derived from IHSkinPerm, a mathematical tool for estimating dermal absorption. Exposure that occurs as a deposition over time or a repeated exposure that maintains a thin layer of liquid TCE had greater absorption based on the estimate from IHSkinPerm for an 8-hr exposure is 1.6% absorption and 98.4% volatilization. Dermal exposures to liquid TCE are expected to be concurrent with inhalation exposures, which are anticipated to reflect the preponderance of overall exposure from a use or activity for most consumer exposure scenarios. This agrees with the NIOSH skin notation profile for TCE, which estimates a low hazard potential by dermal absorption for systemic effects when inhalation and dermal exposures are concurrent (Hudson and Dotson, 2017). There may be certain scenarios with higher dermal exposure potential – where liquid TCE is not able to evaporate readily and volatilization is inhibited. An example of this is a user holding a rag soaked with TCE against their palm during a cleaning activity. Therefore, dermal exposures are quantified and presented for consumer use scenarios that may involve dermal contact with impeded evaporation.

Generally, individuals that have contact with liquid TCE would be users and not bystanders. Therefore, dermal exposures to liquid TCE are not expected and inhalation is the primary route of exposure for bystanders. There is potential for bystanders or users to have indirect dermal contact via contact with a surface upon which TCE has been applied (e.g., counter, floor). Based on the expectation that TCE would evaporate from the surface rapidly, with <1% dermal absorption predicted from instantaneous contact, this route is unlikely to contribute significantly to overall exposure.

2.3.2.3 Potentially Exposed or Susceptible Subpopulations

As part of the Problem Formulation (U.S. EPA, 2018d), EPA identified consumers and bystanders associated with use of TCE-containing consumer products as a potentially exposed and susceptible subpopulation due to greater exposure. Additionally, higher-intensity users (i.e., those using consumer products for longer durations and in greater amounts) were considered and evaluated. Exposures and risks for these subpopulations are considered and evaluated herein. Consumers are considered to include children and adults age 11 and up, but bystanders in the home exposed via inhalation are considered to include any age group, from infant to adult, including pregnant women. Highly exposed (high-intensity users) and potentially exposed or susceptible subpopulations (PESS) within this overall schema as receptor categories overlap, as individuals may belong to multiple receptor groups.

2.3.2.4 Consumer Exposures Approach and Methodology

Modeling was conducted to estimate exposure from the identified consumer conditions of use. Exposures via inhalation and dermal contact to TCE-containing consumer products were estimated using EPA's Consumer Exposure Model (CEM) Version 2.1 (U.S. EPA, 2019a), along with consumer behavioral pattern data (i.e., use patterns) and product-specific characteristics.

Residential indoor air and personal breathing zone data were identified and evaluated during systematic review. However, measured levels are not attributable to specific consumer products or conditions of use and were therefore not compared to modeled estimates. For a summary of these data, see Appendix D.2.

2.3.2.4.1 Modeling Approach

Consumer Exposure Model (CEM) Version 2.1 was selected for the consumer exposure modeling as the most appropriate model to use based on the type of input data available for TCE-containing consumer products. Moreover, EPA did not have the input parameter data (i.e., product-specific chamber emission data) required to run higher-tier indoor air models. The advantages of using CEM to assess exposures to consumers and bystanders are the following:

- CEM model has been peer-reviewed;
- CEM accommodates the distinct inputs available for the products containing TCE; and
- CEM uses the same calculation engine to compute indoor air concentrations from a source as the higher-tier Multi-Chamber Concentration and Exposure Model (MCCEM) but does not require measured chamber emission values.

For a characterization of model sensitivity, see Appendix D.1 .

Modeling Air Concentrations and Inhalation Exposure

CEM predicts indoor air concentrations from consumer product use by implementing a deterministic, mass-balance calculation utilizing an emission profile determined by implementing appropriate emission scenarios. The model uses a two-zone representation of the building of use (e.g., residence, school, office), with Zone 1 representing the room where the consumer product is used (e.g., a utility room) and zone 2 being the remainder of the building. The product user is placed within Zone 1 for the duration of use, while a bystander is placed in Zone 2 during product use. Otherwise, product users and bystanders follow prescribed activity patterns throughout the simulated period. In some instances of product use, a higher concentration of product is expected very near the product user; CEM addresses this by further dividing Zone 1 into near-field, with a default volume of $1 m^3$, and far-field, which reflects the remainder of Zone 1. Each zone is considered well-mixed. Product users are exposed to airborne concentrations estimated within the near-field during the time of use and otherwise follow their prescribed activity pattern. Bystanders follow their prescribed activity pattern and are exposed to far-field concentrations when they are in Zone 1. Background concentrations can be set to a non-zero concentration if desired.

For acute exposure scenarios, emissions from each incidence of product usage are estimated over a period of 72 hours using the following approach that account for how a product is used or applied, the total applied mass of the product, the weight fraction of the chemical in the product, and the molecular weight and vapor pressure of the chemical.

The general steps of the calculation engine within the CEM model include:

- Introduction of the chemical (i.e., TCE) into the room of use (Zone 1) through two possible pathways: (1) overspray of the product or (2) evaporation from a thin film;
- Transfer of the chemical to the rest of the house (Zone 2) due to exchange of air between the different rooms;

- Exchange of the house air with outdoor air; and
- Compilation of estimated air concentrations in each zone as the modeled occupant (i.e., user or bystander) moves about the house per prescribed activity patterns.

As receptors move between zones in the model, the associated zonal air concentrations at each 30-second time step were compiled to reflect the air concentrations a user and bystander would be exposed to throughout the simulation period. Time weighted averages (TWAs) were then computed based on these user and bystander concentration time series per available human health hazard data. For TCE, 3- and 24-hour TWAs were quantified for use in risk evaluation based on alignment relevant acute human health hazard endpoints.

Emission Models

Based on the suite of product scenarios developed to evaluate the TCE consumer conditions of use, the specific emission models applied for the purposes of modeling TCE products include: E1: Emission from Product Applied to a Surface Indoors Incremental Source Model and E3: Emission from Product Sprayed.

E1 assumes a constant application rate over a user-specified duration of use and an emission rate that declines exponentially over time, at a rate that depends on the chemical molecular weight and vapor pressure. This emission model is generally applicable to liquid products applied to surfaces that evaporate from those surfaces, such as cleaners. E1 was applied for all liquid formulations in the modeling of TCE consumer inhalation exposures. E3 assumes a small percentage of product becomes airborne rather than contacting the target surface and therefore immediately available for uptake via inhalation. This is called "overspray" and is not well characterized, though default parameters ranging from 4.5 to 6% overspray are based on a combination of modeled and empirical data from Jayjock (2012) and are said to reflect reasonable worst-case overspray potential (U.S. EPA, 2017b). The remainder of chemical is assumed to contact the target surface and volatilize at a rate that depends on the chemical molecular weight and vapor pressure. The aerosolized portion is treated using a constant emission rate model while the non-aerosolized mass is treated in the same manner as liquid products applied to a surface, combining a constant application rate with an exponentially declining rate. In U.S. EPA (2014b), modeled scenarios were found not to be sensitive to this parameter, with overspray fractions of 1 and 25% producing nearly identical peak concentrations for TCE. Both E1 and E3 have a near-field model option that is selected to capture the higher concentration in the breathing zone of a product user during use.

For additional details on CEM 2.1's underlying emission models, assumptions, and algorithms, please see the User Guide Section 3: Detailed Descriptions of Models within CEM (U.S. EPA, 2019a). The emission models used have been compared to other model results and measured data; see Appendix D: Model Corroboration of the User Guide Appendices for the results of these analyses (U.S. EPA, 2019b).

Modeling Dermal Exposure

CEM also contains a dermal modeling component that estimates absorbed dermal doses resulting from dermal contact with chemicals found in consumer products. Based on the described dermal exposure conditions (i.e., dermal contact with impeded evaporation) and the chemical- and scenario-specific input parameters available for use in modeling (e.g., scenario-specific use duration, measured dermal permeability coefficient), "P_DER2b: Dermal Dose from Product Applied to Skin, Permeability Model" was selected as the most appropriate model to estimate dermal exposures from consumer products containing TCE. P_DER2b estimates dermal flux based on a permeability coefficient (K_p) and is based on the ability of a chemical to penetrate the skin layer once contact occurs. It assumes a constant supply

of chemical directly in contact with the skin throughout the exposure duration. The acute form of the model is given below:

$$ADR = \frac{K_p \times D_{ac} \times \rho \times \frac{SA}{BW} \times FQ_{ac} \times Dil \times WF \times ED_{ac} \times CF_1}{AT_{ac} \times CF_2}$$

Where:

ADR = Potential acute dose rate (mg/kg-day)
K_p = Permeability coefficient (cm/hr)
D_{ac} = Duration of use (min/event)
ρ = Density of formulation (g/cm^3)
SA/BW = Surface area to body weight ratio (cm^2/kg)
FQ_{ac} = Frequency of use (events/day, 1 for acute exposure scenarios)
Dil = Product dilution fraction (unitless, 1 [no dilution] for all TCE scenarios)
WF = Weight fraction of chemical in product (unitless)
ED_{ac} = Exposure duration (days)
CF1 = Conversion factor (1,000 mg/g)
CF2 = Conversion factor (60 min/hr)
AT_{ac} = Averaging time (days, 1 for acute exposure scenarios)

K_p is a measure of the rate of chemical flux through the skin. The parameter can either be specified by the user (if measured data are reasonably available) or be estimated within CEM using a chemical's molecular weight and octanol-water partition coefficient (K_{OW}). Note the permeability model does not inherently account for evaporative losses (unless the available flux or K_p values are based on non-occluded, evaporative conditions), which can be considerable for volatile chemicals in scenarios where evaporation is not impeded. While the permeability model does not explicitly represent exposures involving such impeded evaporation, the model assumptions make it the preferred model for an such a scenario (e.g., a scenario wherein dermal contact involved impeded evaporation, or where there is potential for dermal immersion). Furthermore, it incorporates scenario-specific product use durations and distinct surface area to body weight ratios for various user populations. For additional details on P_DER2b, please see the CEM User Guide Section 3: Detailed Descriptions of Models within CEM (U.S. EPA, 2019a).

For TCE, a measured dermal permeability coefficient (K_p 0.019 cm/hr) is applied, based on findings from Poet (2000), as summarized and presented in the 2017 NIOSH Skin Notation Profile for TCE (Hudson and Dotson, 2017). The permeability coefficient selected was based on a human water-patch test and was within range of the estimated K_p values presented in the 2017 NIOSH Skin Notation Profile (0.01197 cm/hr) (Hudson and Dotson, 2017) and within the CEM model (0.028 cm/hr), both predicted using chemical properties.

Dermal exposure estimates are only quantified and presented for consumer exposure scenarios that could involve such dermal contact with impeded evaporation (e.g., application or cleaning with a rag pressed against user's hand), per the focus described in Section 2.3.2.2.2.

Variation

To capture a range of potential exposure levels associated with consumer conditions of use, three input parameters were varied: mass of product used, weight fraction, and duration of use. Aside from these three parameters, model inputs were held constant across a specific scenario or across all product scenarios. For example, certain inputs such as the room of use (and associated room/Zone 1 volume), overspray fraction, and surface area to body weight ratio exposed in dermal exposure scenarios were

held constant across the multiple iterations of a single product scenario but differed across product scenarios based on their scenario-specific nature. Other parameters such as chemical properties, building volume, air exchange rate, and user and bystander activity patterns (i.e., movements around the home) were held constant across all product scenarios and runs. The majority of the non-varied modeling parameters reflect central tendency inputs (i.e., median or mean values; see Table 2-28); therefore, the combination of high-end inputs for the three varied parameters do not reflect "worst-case" or bounding estimates.

Varied Inputs:

Considering the model sensitivity analysis summarized in Appendix D.1 and the availability of high-quality use-pattern data, EPA varied three input parameters: chemical weight fraction (WF) in a consumer product; mass of product used per use event; and duration of product use per event.

The low-, mid-, and/or high-end weight fractions were selected principally from MSDS/SDS forms. For subcategories where there was only one product with a weight fraction range, only one weight fraction was used for modeling. If there were two or more products with weight fraction ranges, the low-end of lowest non-zero range and high-end of highest range were the bounding weight fractions. For a central tendency weight fraction, the mid-point between bounding weight fractions was calculated. In the case of unknown weight fractions, values were selected from the range of related products. Further detail is provided in the Supplemental File, [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*].

Mass of product used and duration of use selections define user characteristics (e.g., high-intensity user, moderate-intensity user, low-intensity user) and are based on the Household Solvent Products: A National Usage Survey (U.S. EPA, 1987), referred to as the "Westat survey" or "Westat" herein, and described further in section 2.3.2.5. The survey was rated as having "high" quality during the data evaluation phase of systematic review. Weight fraction (i.e., the percentage of TCE in the product formulation) represents the true range in the market based on manufacturer-developed Safety Data Sheets (SDSs).

For each parameter varied, up to three distinct inputs were modeled to address known variability across these three parameters. While this approach resulted in up to 27 distinct exposure results for each product scenario/condition of use, this was a deterministic assessment and results reflect a range based on variation of three key parameters, not a distribution. Unlike inhalation modeling, for dermal modeling, only the weight fraction and duration of product use were varied because mass used is not a parameter in the dermal exposure model P_DER2b.

In the model sensitivity analysis, summarized in Appendix D.1 and shown in the user guide appendices (U.S. EPA, 2019b), additional parameters are identified as highly sensitive, including the air exchange rate and zone volume. However, the central tendency default modeling values were held constant for these inputs. The inputs varied included those that characterize actual users and reflect levels of TCE in actual products.

2.3.2.5 Consumer Exposure Scenarios and Modeling Inputs

Exposure modeling scenarios comprise information that characterizes chemical properties, products, and use patterns, including:

- Formulations (e.g., weight fraction, formulation type [aerosol, liquid]);
- Chemical or product-specific properties (e.g., product density, vapor pressure, molecular weight diffusion coefficient, overspray fraction, transfer coefficients, dilution factor);

- Use patterns (e.g., frequency, duration, and amount used);
- Human exposure factors (e.g., body weight, inhalation rate); and
- Environmental conditions (e.g., air exchange rates and room size).

Consumer exposure modeling scenarios based on the identified conditions of use were built based on identified TCE products that may be available to consumers, including solvents for cleaning and degreasing, lubricants and greases, adhesives and sealants, and other uses. The subcategories of use (i.e., consumer product types) cited in Table 2-27 were used to develop distinct consumer exposure modeling scenarios for use in estimating inhalation and dermal exposure to consumers and bystanders. The availability of TCE in consumer products was determined through the development of EPA's 2017 Market and Use Report and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: TCE. Additional online research was undertaken following Problem Formulation to confirm TCE concentrations and compile a comprehensive list of products that may be available to consumers for household use. Specific product characteristics obtained from manufacturer websites and/or Safety Data Sheets (SDSs) such as form/formulation type, weight fraction and density, were used to select the most appropriate product-specific inputs (e.g., weight fraction and formulation type) associated with each consumer condition of use. Please see Supplemental File [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*]for full product details, including product-specific formulations, weight fractions, and densities.

CEM requires inputs governing chemical properties, product characteristics, use environment, and user patterns (i.e., user behavior). These include inputs such as physical chemical properties, weight fraction, formulation type, duration of product use, mass of product used, and Zone 1 (room of use) volume. To determine relevance and appropriateness of the consumer use pattern parameters, EPA reviewed the consumer product categories available in the Westat Survey (1987). Westat surveyed thousands of American households via questionnaire or telephone from 4,920 respondents across the United States to gather information on consumer behavior (i.e., use patterns) and product characteristics (e.g., product formulation type) related to product categories that may contain halogenated solvents like TCE. The Westat Survey was rated as a high quality study during data evaluation within the systematic review process. It forms the basis for relevant chapters of EPA's Exposure Factors Handbook and was used to derive certain default parameters in EPA's CEM 2.1. Westat (1987) includes survey response data on 30 distinct product categories and reports the following: numbers of respondents; percentage of respondents reporting use; frequency of use; duration of use; time spent in the room of use; brand of product used; form of product used; amount of product used; and room of use.

The room of use selected for this evaluation is based on the room in which the Westat Survey results reported the highest percentage of respondents that last used a product within the room. When the Westat Survey identified the room of use where the highest percentage of respondents last used the product as "other inside room", the utility room was selected within CEM for modeling. The pre-defined product scenarios within CEM were selected based on a cross-walk to similar product categories within the Westat Survey.

In evaluating Westat survey data for appropriateness, EPA considered the similarity of product category, as well as the similarity of reported product formulation type (i.e., aerosol, liquid). When a direct alignment could not be found between the consumer product and Westat product category, EPA used professional judgement in considering other Westat categories with reasonable ranges for use duration and amount of product used. A crosswalk between TCE consumer use scenarios and Westat Product Categories are listed in Table 2-30 and described in more detail in Section 2.3.2.6.2.

2.3.2.5.1 Consumer Exposure Model Inputs

Chemical-specific inputs required to model consumer inhalation and dermal exposure included physical and chemical properties (
Table 1-1), as well as a chemical-specific dermal permeability coefficient (0.019 cm/hr), which were held constant across all modeling scenarios and iterations.

The consumer exposure model requires product-specific data based on product characteristics and use patterns. It also requires fixed inputs to define the exposure zones (e.g., room and building volumes, air exchange rates, interzonal ventilation rates); general use patterns defining the amount of time a receptor is likely to be in the home; receptor characteristics (e.g., age, surface area to body weight ratios); and emission characteristics (e.g., background air concentration, emission factor). These default inputs are held constant for a given scenario but may vary across scenarios based on scenario-specific exposure factors or assumptions. As such, these inputs were not altered to capture within-scenario variation. Table 2-28 shows these default parameters.

Table 2-29 displays TCE consumer product modeling scenarios and associated product-specific inputs that were varied to capture within-scenario variation. These varied inputs include: weight fraction, duration of use, and mass of product used. Westat (1987) is the basis for duration of use and mass of product used and product SDSs are the basis for weight fraction and formulation type.

Table 2-30 presents the consumer product modeling scenarios and associated scenario-specific inputs that were not varied within product modeling scenarios but did vary across scenarios. In modeling exposures within and across all scenarios, parameters displayed in both below tables were utilized, along with the general chemical-specific characteristics and other model defaults. Please see Supplemental File [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*] for a spreadsheet summarizing all of the model inputs and product information.

For all scenarios, the consumer user was assumed to be an adult (age 21+) and two child age groups (16-20 years and 11-15 years), while a non-user bystander can include individuals of any age. For the TCE products identified, younger children would not be expected to be directly using these products. Inhalation exposure results are presented as concentrations encountered by users and non-user bystanders and are independent of age group. EPA presents all three evaluated user age groups for dermal exposures as reported doses are age-group specific.

Table 2-28. Default Modeling Input Parameters

Parameter Type	Modeling Parameter	Default Value Modeled	Value Characterization	Reference
Building Characteristic[1]	Building Volume (m^3)	492	Central Tendency (Mean)	(U.S. EPA, 2011c)
	Air Exchange Rate (hr^{-1})	0.45[2]	Central Tendency (Median)	(U.S. EPA, 2011c)
	Interzonal Ventilation Rate (m^3/hr)[3]	Garage: 109	NA	Default (U.S. EPA, 2019a, b)
		All other rooms modeled: 107		
Emission Characteristics	Background Air Concentration (mg/m^3)	0	Minimum	

	Gas Phase Mass Transfer Coefficient (m/hr)	Based on chemical properties and estimated within CEM		
	Emission Factor (ug/m^2/hr)			
	Saturation Concentration in Air (mg/m^3)	5.18E+05	Based on chemical properties and estimated within CEM	
	Aerosol Fraction (Spray Scenarios Only)	0.06	High-end	
	Product Dilution Fraction	1 (no dilution)	NA	Based on formulation and intended use
Use Patterns and Exposure Factors	Receptor Activity Pattern	Stay at home[4]	NA	Default (U.S. EPA, 2019a, b)
	Use Start Time	9 AM[5]	NA	NA
	Frequency of Use	1 event per day	NA	Default (U.S. EPA, 2019a, b)
	Acute Averaging Time	1 day	NA	
	Surface Area to Body Weight Ratio	Inside of One Hand		
		Adult (21+): 3.10	Central tendency (mean)	
		Children (16-20): 2.90		
		Children (11-15): 3.17		
		10% of Hands		
		Adult (21+): 1.24	Central tendency (mean)	
		Children (16-20): 1.16		
		Children (11-15): 1.27		

[1] An overall residential building volume of 492 m^3 is used to calculate air concentrations in Zone 2 and room volume is used to calculate air concentrations in Zone 1. The volume of the near-field bubble in Zone 1 was assumed to be 1 m^3 in all cases, with the remaining volume of Zone 1 comprising the far-field volume.
[2]Air exchange rates differed for two scenarios: pepper spray and hoof polish (see Table 2-30).
[3] The default interzonal air flows are a function of the overall air exchange rate and volume of the building, as well as the "openness" of the room itself. Kitchens, living rooms, garages, schools, and offices are considered more open to the rest of the home or building of use; bedrooms, bathrooms, laundry rooms, and utility rooms are usually accessed through one door and are considered more closed.
[4] The activity pattern (i.e., zone location throughout the simulated exposure period) for user and bystander was the default "stay-at-home" resident, which assumes the receptors are primarily in the home (in either Zone 1 or 2) throughout the day. These activity patterns in CEM were developed based on Consolidated Human Activity Database (CHAD) data of activity patterns (Isaacs, 2014).
[5] Product use was assumed to start at 9 AM in the morning; as such, the user was assumed to be in the room of use (Zone 1) at that time, regardless of the default activity pattern placement at 9 AM.

2056 **Table 2-29. Consumer Product Modeling Scenarios and Varied Input Parameters**

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Range of Weight Fraction (% TCE)[2]	Weight Fractions Selected for Modeling (% TCE)			Selected Westat Survey Scenario	Duration of Use (min)			Range of Product Density (g/cm^3)[4]	Mass [Volume] of Product Used (g, [oz])		
				Min[2]	Mid	Max		10th %ile[3]	50th %ile	95th %ile		10th %ile	50th %ile	95th %ile
Solvents for Cleaning and Degreasing	Brake & Parts Cleaner	Aerosol (4)	0 - 100	20	60	100	Brake Quieters / Cleaners	1	15	120	1.23-1.62	47.9 [1]	191.6 [4]	766.5 [16]
	Electronic Degreaser/ Cleaner	Aerosol (9)	30 - 100	30	65	100	Specialized Electronics Cleaners (for TV, VCR, Razor, etc.)	0.17	2	30	1.25-1.52	1.8 [0.04]	22.5 [0.5]	337.1 [7.5]
	Electronic Degreaser/ Cleaner	Liquid (1)	100	100			Specialized Electronics Cleaners (for TV, VCR, Razor, etc.)	0.17	2	30	1.46	1.7 [0.04]	21.6 [0.5]	323.8 [7.5]
	Spray Degreaser/ Cleaner	Aerosol (8)	60 - 100	60		100	Engine Degreasing[5]	5	15	120	1.46-1.52	130.8 [2.91]	521.4 [11.6]	2157.4 [48]
	Liquid Degreaser/ Cleaner	Liquid (2)	90 - 100	100			Solvent-Type Cleaning Fluids or Degreasers	2	15	120	1.456	24.1 [0.56]	139.9 [3.25]	1377.7 [32]
	Gun Scrubber	Aerosol (2)	60 - 100[6]	60		100	Solvent-Type Cleaning Fluids or Degreasers[7]	2	15	120	1.36-1.465	NA	0.7 [0.45 mL][8]	NA
	Gun Scrubber	Liquid (1)	100[8]	100			Solvent-Type Cleaning Fluids or Degreasers[7]	2	15	120	1.36	NA	0.6 [0.45 mL][8]	NA

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Range of Weight Fraction (% TCE)[2]	Weight Fractions Selected for Modeling (% TCE)			Selected Westat Survey Scenario	Duration of Use (min)			Range of Product Density (g/cm^3)[4]	Mass [Volume] of Product Used (g, [oz])		
				Min[2]	Mid	Max		10th %ile[3]	50th %ile	95th %ile		10th %ile	50th %ile	95th %ile
	Mold Release	Aerosol (2)	40 - 68.9	40		68.9	Other Lubricants (Excluding Automotive)	0.08	2	30	0.77-1.44	4.3 [0.1]	23.4 [0.55]	212.9 [5]
	Tire Cleaner	Aerosol (2)	70 - 100	70		100	Tire / Hubcap Cleaner	5	15	60	0.67	10.5 [0.53]	52.9 [2.67]	317.0 [16]
	Tire Cleaner	Liquid (1)	80 - 100	100			Tire / Hubcap Cleaner	5	15	60	0.67-1.493	23.4 [0.53]	117.9 [2.67]	706.4 [16]
Lubricants and Greases	Tap & Die Fluid	Aerosol (1)	98	98			Other Lubricants (Excluding Automotive)	0.08	2	30	0.9	2.7 [0.1]	14.8 [0.55]	134.5 [5]
	Penetrating Lubricant	Aerosol (5)	5 - 50	5	27.5	50	Other Lubricants (Excluding Automotive)	0.08	2	30	0.636-1.42	4.2 [0.1]	23.1 [0.55]	209.9 [5]
Adhesives and Sealants	Solvent-based Adhesive & Sealant	Liquid (3)	5 - >90	5	47.5	90	Contact Cement, Super Glues, and Spray Adhesives	0.33	4.25	60	1.33-1.45	1.3 [0.03]	10.7 [0.25]	185.2 [4.32]
	Mirror-edge Sealant	Aerosol (1)	20 - 40	40			Contact Cement, Super Glues, and Spray Adhesives	0.33	4.25	60	0.614	0.5 [0.03]	4.5 [0.25]	78.4 [4.32]
	Tire Repair Cement/ Sealer	Liquid (5)	65 - 95	65	80	95	Contact Cement, Super Glues, and Spray Adhesives	0.33	4.25	60	1.45	1.3 [0.03]	10.7 [0.25]	185.2 [4.32]

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Range of Weight Fraction (% TCE)[2]	Weight Fractions Selected for Modeling (% TCE)			Selected Westat Survey Scenario	Duration of Use (min)			Range of Product Density (g/cm^3)[4]	Mass [Volume] of Product Used (g, [oz])		
				Min[2]	Mid	Max		10th %ile[3]	50th %ile	95th %ile		10th %ile	50th %ile	95th %ile
Cleaning and Furniture Care Products	Carpet Cleaner	Liquid (1)	99	99			Spot Removers	0.25	5	30	1.6	11.8 [0.25]	62.9 [1.33]	526.6 [11.13]
	Spot Remover	Aerosol (1)	20 - 30	30			Spot Removers	0.25	5	30	1.562	11.5 [0.25]	61.4 [1.33]	514.1 [11.13]
	Spot Remover	Liquid (4)	<50 - >75	50		75	Spot Removers	0.25	5	30	1.25-1.45	10.7 [0.25]	57.0 [1.33]	477.2 [11.13]
Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings	Aerosol (1)	20 - 30	30			Aerosol Rust Removers[9]	0.25	5	60	0.704	9.4 [0.45]	45.2 [2.17]	306.0 [14.7]
Apparel and Footwear Care Products	Shoe Polish	Aerosol (1)	10 - 20	20			Spray Shoe Polish	0.5	5	30	0.512	2.9 [0.19]	15.4 [1.02]	151.4 [10]
Other Consumer Uses	Fabric Spray	Aerosol (1)	20 - 40	40			Water Repellents / Protectors (for Suede, Leather, and Cloth)	1.4	10	60	0.614	11.4 [0.63]	49.9 [2.75]	326.8 [18]
	Film Cleaner	Aerosol (2)	80 - 100	100			Aerosol Rust Removers[9]	0.25	5	60	1.45-1.456	19.4 [0.45]	93.4 [2.17]	632.9 [14.7]
	Hoof Polish	Aerosol (1)	30[10]	30			Spray Shoe Polish[11]	0.5	5	30	0.512-0.704	4.0 [0.19]	21.2 [1.02]	208.2 [10]
	Pepper Spray	Aerosol (2)	91.5	91.5			NA[12]	NA	0.08[12]	NA	1.25	NA	4.0 [0.108][12]	NA
	Toner Aid	Aerosol (1)	10 - 20	20			Aerosol Rust Removers[9]	0.25	5	60	1	13.3 [0.45]	64.2 [2.17]	434.7 [14.7]

[1] The number of products identified is based on the product lists in EPA's 2017 Market and Use Report and Preliminary Information on Manufacturing, Processing, Distribution, Use and Disposal: TCE, as well as the 2014 TSCA Work Plan Chemical Risk Assessment for TCE (U.S. EPA, 2017c, h). Please see Supplemental File [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*] for the full product list utilized.

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Range of Weight Fraction (% TCE)[2]	Weight Fractions Selected for Modeling (% TCE)			Selected Westat Survey Scenario	Duration of Use (min)			Range of Product Density (g/cm^3)[4]	Mass [Volume] of Product Used (g, [oz])		
				Min[2]	Mid	Max		10th %ile[3]	50th %ile	95th %ile		10th %ile	50th %ile	95th %ile

[2] Weight fractions were primarily sourced from product Safety Data Sheets (SDSs) or Material Safety Data Sheets (MSDSs), unless otherwise noted. Please see Supplemental File [*Consumer Exposure Assessment Model Input Parameters. Docket: EPA-HQ-OPPT-2019-0500*] for more detailed information on weight fraction sourcing and ranges. If a single weight fraction was used in modeling, it appears in the "Min" weight fraction column, but does not reflect a minimum.

[3] Low-end (10th percentile) durations reported by Westat that are less than 0.5 min (30 sec) are modeled as being equal to 0.5 min (smallest time-step modeled).

[4] Product density ranges reflect identified products containing TCE and were sourced from product SDSs or MSDSs. The high end of the range identified was used to convert reported ounces of product used from Westat (1987) to grams of product used, as required for model input.

[5] Two Westat product categories were considered for use (engine degreasing and solvent-type cleaning fluids or degreasers); however, engine degreasing was selected to source duration of use, room of use, and amount used parameters due to the high percentage of respondents (78.9%) reporting aerosol use.

[6] No weight fraction was reasonably available for the aerosol and liquid gun scrubber formulations, so the weight fractions were based on the ranges reflected by the aerosol and liquid degreasing products.

[7] The solvent-type cleaning fluids or degreasers product category from Westat was used as a surrogate for gun scrubbers for the selection of use durations. Product-specific literature was identified and applied for mass of product used.

[8] Based on EPA/EPAB research and the Eezox Premium Gun Care testing results (ASTM B117-5 Salt Spray Fog Test), 0.42-0.45 mL of the product was used to coat the firearm in a very thin film, which is in-line with use directions.

[9] Three modeling scenarios (film cleaner, spray fixative/coating, and toner aid) had no directly-aligned Westat product categories. Therefore, a number of Westat product categories and use pattern data were considered for appropriateness, with a focus on primary formulation type (aerosol or liquid), duration of use, and amount used. The rust remover product category reflects 98% aerosol products and a lower use duration and amount used than many of the other solvent degreasing-type uses.

[10] Weight fraction and density were not reasonably available, so were based on the ranges reflected by the spray fixative/coating and aerosol shoe polish products.

[11] There were no reasonably available data sources for aerosol hoof polish use patterns; the Westat spray shoe polish product category was used for selection of use duration and amount used.

[12] Based on EPA/EPAB research that found one spray from the most common civilian canister is estimated to be approximately 0.0216-0.108 ounces (based on a pepper spray manufacturer's website). Spraying occurred between 3 and 5 seconds (converted to minutes for use in modeling) before obtaining desired effect (Bertilsson et al., 2017).

2062 **Table 2-30. Consumer Product Modeling Scenarios and Additional Scenario-Specific Input Parameters**

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Zone 1 Room of Use (Volume m^3)[2]	CEM Emission Model Applied[3]	Air Exchange Rate (hr^{-1})	Interzonal Ventilation Rate (m^3/hr)	CEM Dermal Exposure Model Applied[4]	Dermal Surface Area Exposed[5]
Solvents for Cleaning and Degreasing	Brake & Parts Cleaner	Aerosol (4)	Garage (90)	E3	0.45	109	P_DER2b	10% of hands
	Electronic Degreaser/ Cleaner	Aerosol (9)	Utility (20)	E3	0.45	107	NA	10% of hands
	Electronic Degreaser/Cleaner	Liquid (1)	Utility (20)	E1	0.45	107	P_DER2b	Inside of one hand
	Spray Degreaser/Cleaner	Aerosol (8)	Garage (90)	E3	0.45	109	P_DER2b	10% of hands
	Liquid Degreaser/Cleaner	Liquid (2)	Utility (20)	E1	0.45	107	P_DER2b	Inside of one hand
	Gun Scrubber	Aerosol (2)	Utility (20)	E3	0.45	107	NA	10% of hands
	Gun Scrubber	Liquid (1)	Utility (20)	E1	0.45	107	P_DER2b	Inside of one hand
	Mold Release	Aerosol (2)	Utility (20)	E3	0.45	107	NA	10% of hands
	Tire Cleaner	Aerosol (2)	Garage (90)	E3	0.45	109	P_DER2b	10% of hands
	Tire Cleaner	Liquid (1)	Garage (90)	E1	0.45	109	P_DER2b	Inside of one hand
Lubricants and Greases	Tap & Die Fluid	Aerosol (1)	Utility (20)	E3	0.45	107	NA	10% of hands
	Penetrating Lubricant	Aerosol (5)	Utility (20)	E3	0.45	107	NA	10% of hands
Adhesives and Sealants	Solvent-based Adhesive & Sealant	Liquid (3)	Utility (20)	E1	0.45	107	NA	Inside of one hand
	Mirror-edge Sealant	Aerosol (1)	Bathroom (15)	E3	0.45	107	NA	10% of hands
	Tire Repair Cement/ Sealer	Liquid (5)	Garage (90)	E1	0.45	109	NA	Inside of one hand
	Carpet Cleaner	Liquid (1)	Bedroom (36)	E1	0.45	107	P_DER2b	Inside of one hand

Consumer Category	Product Sub-Categories	Form (No. of Pdts)[1]	Zone 1 Room of Use (Volume m^3)[2]	CEM Emission Model Applied[3]	Air Exchange Rate (hr^{-1})	Interzonal Ventilation Rate (m^3/hr)	CEM Dermal Exposure Model Applied[4]	Dermal Surface Area Exposed[5]
Cleaning and Furniture Care Products	Spot Remover	Aerosol (1)	Utility (20)	E3	0.45	107	P_DER2b	10% of hands
	Spot Remover	Liquid (4)	Utility (20)	E1	0.45	107	P_DER2b	Inside of one hand
Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings	Aerosol (1)	Utility (20)	E3	0.45	107	NA	10% of hands
Apparel and Footwear care products	Shoe Polish	Aerosol (1)	Utility (20)	E3	0.45	107	NA	Inside of one hand
Other Consumer Uses	Fabric Spray	Aerosol (1)	Utility (20)	E3	0.45	107	NA	10% of hands
	Film Cleaner	Aerosol (2)	Utility (20)	E3	0.45	107	NA	10% of hands
	Hoof Polish	Aerosol (1)	Barn[6]	E3	4[6]	109	NA	10% of hands
	Pepper Spray	Aerosol (2)	Outside[7]	E3	100[7]	0	NA	10% of hands
	Toner Aid	Aerosol (1)	Utility (20)	E3	0.45	107	NA	10% of hands

[1]The number of products identified is based on the product lists in EPA's 2017 Market and Use Report and Preliminary Information on Manufacturing, Processing, Distribution, Use and Disposal: TCE (U.S. EPA, 2017c, h), as well as the 2014 TSCA Work Plan Chemical Risk Assessment for TCE (U.S. EPA, 2014b). 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 TCE or are no longer reasonably 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. Please see Supplemental File for the full product list utilized.

[2] The use environment (room of use) was generally based on the Westat (1987) survey of consumer behavior patterns, which reported the percentages for the location of last use of product. In cases where the room was identified as "other inside room," the utility room was selected based on professional judgment. Additionally, professional judgment was applied to certain uses, such as those that could reasonably be used in a garage setting.

[3]Emission models used for TCE include E1 – Emission from Product Applied to a Surface Indoors Incremental Source Model and E3 – Emission from Product Sprayed.

[4]All scenarios utilized the P_DER2b model for dermal exposure – Dermal Dose from Product Applied to Skin, Permeability Model

[5]Surface area exposed only applied in dermal scenarios. The indicated surface areas are combined with mean receptor body weights to get surface area to body weight ratios (SA:BW) that are used in estimating dermal dose.

[6]For the purposed of modeling typical aerosol hoof polish consumer exposure, a barn setting was approximated by selecting the garage as the room of use and changing the default air exchange rate from 0.45 to 4 hr^{-1}, which more closely aligns with recommended ventilation levels in a horse barn (Pennsylvania State University, 2016)

[7]The outdoor environment was approximated by selecting the garage as the room of use and increasing the air exchange rate from 0.45 to 100. The "room of use" was also edited to reflect a 16 m3 cloud around user (roughly 6.5-foot dome or cloud surrounding user).

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The 2014 TCE TSCA Work Plan Chemical Risk Assessment included two consumer conditions of use: aerosol degreaser and clear protective coating spray (referred to as "spray fixative" 80 FR 47441) (U.S. EPA, 2014b). The inputs included in the 2014 assessment differed from those used in this assessment for similar conditions of use, either due to updated parameter data (e.g., Zone 2 volume), or professional judgment. The most notable difference between the 2014 scenarios related to the single mass used parameter selected. In the 2014 assessment, aerosol degreaser was modeled assuming 24 g (0.85 oz) and clear protecting coating spray was modeled assuming 11g (0.39 oz). These inputs were not based on user survey data and were described in the 2014 assessment as "potentially on the low end" when compared against the Westat survey data employed in this 2019 risk evaluation.

2.3.2.6 Consumer Exposure Results

Acute inhalation and dermal exposure results are presented below for each consumer condition of use. Dermal exposure results are only presented for those scenarios deemed to have the potential for dermal contact with impeded evaporation per the scope presented in the May 2018 Problem Formulation (U.S. EPA, 2018d). These conditions of use are organized by product subcategories and are also referred to herein as consumer modeling scenarios. Inhalation estimates are presented in terms of acute indoor air concentrations (ppm) resulting from a single consumer use event within a one-day exposure period; they are provided for users and bystanders. Acute dermal exposure estimates are presented as an acute dose (mg/kg/day); they are provided for users only.

2.3.2.6.1 Characterization of Exposure Results

As described in Section 2.3.2.4.1, the consumer exposure modeling approach was deterministic, but a range of exposure results were estimated based on varying three parameters: weight fraction, mass of product used, and duration of use/exposure duration. While the exposure results are not reflective of a probabilistic distribution of all possible exposure levels, the exposure scenarios modeled incorporated low-end (10^{th} percentile), central tendency (50^{th} percentile), and high-end (95^{th} percentile) inputs from Westat (1987) for two of the three varied parameters: mass of product used and exposure duration. Since these inputs primarily reflect user characterization, results are presented for "high-intensity users," "moderate-intensity users," and "low-intensity users." For example, the exposure scenario combining high-end inputs for these three parameters is referred to as a "high-intensity user" scenario. Weight fraction inputs cannot be described in the same terms, as they reflect the range of actual product weight fractions, per associated SDSs, and do not reflect a distribution of user survey data.

Other modeling parameters that were not varied (e.g., room volume, air exchange rate, building volume) reflect central tendency inputs. Therefore, these exposure scenarios and results are not bounding or "worst-case" and may not capture the maximum or minimum of all possible exposure levels.

For TCE, 3- and 24-hr TWA air concentrations are provided for consumers and bystanders. These are based on the relevant human health hazard metrics. The 3-hr TWA air concentrations are higher than the 24-hr air concentrations in all scenarios due to the shorter averaging time surrounding the use event. Likewise, the air concentrations associated with the user are higher than those associated with the bystander in all scenarios due to the higher concentration of chemical expected in the room of use (Zone 1) coupled with the greater amount of time a consumer is assumed to be in the room of use (during and after use event) compared with the bystander. While it is assumed that a bystander of any age, including pregnant women and children, could be exposed to the reported concentrations, the concentrations themselves are not unique for specific subpopulations. The concentrations reported reflect the concentration a consumer or bystander would be exposed to.

Dermal exposure scenarios and results are presented for children and adult age groups, with the children (age 11-15) resulting in the highest estimates dermal exposures due to differences in surface area to body weight ratios in these groups. Results are not presented specifically for pregnant women or women of reproductive age; however, the range of results presented for adults and children age groups are expected to cover dermal exposures for pregnant women as well, with the children (11-15) providing the highest surface area to body weight ratio, thereby providing the highest dermal exposure estimate (see below table for rationale). All values below in Table 2-31 are sourced and/or derived from EPA's 2011 Exposure Factors Handbook (U.S. EPA, 2011c).

Table 2-31. Surface Area and Body Weight Values for Different Consumer and Bystander Subpopulations

Parameter	Adult	Children (16-21)	Children (11-15)	Pregnant Women	Women (21+)	Women (16-21)
10% of Hands Surface Area (cm^2)	99	83	72	89[1]	89[1]	83[2]
Body Weight (kg)	80	71.6	56.8	75[3]	74[4]	65.9[5]
SA:BW	1.24	1.16	1.27	1.19	1.20	1.26

[1]Surface area based on women 21+
[2]Surface area based on combined male/female 16-21
[3]Body weight for all pregnant women
[4]Body weight for females 21+
[5]Body weight for females 16-21

2.3.2.6.2 Consumer Exposure Estimates

Solvents for Cleaning and Degreasing

Brake & Parts Cleaner

Exposure to TCE in brake & parts cleaner products was evaluated based on four aerosol products with weight fractions ranging from 0-20% to 90-100% TCE.

Westat Survey data on brake quieters and cleaners were used as the basis for duration of use and mass of product used. Survey responses indicate that 2.6% of respondents have used products in this category; 65.6% reported use of aerosol formulations. The room of use (Zone 1) was set to the garage (90 m^3) although the Westat survey data for this category indicate primarily outdoor use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-32. Acute Inhalation Exposure Summary: Brake & Parts Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (120)	Max (100)	95th %ile (766.5)	User	3.97E+02	5.76E+01
				Bystander	1.00E+02	1.67E+01

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
Moderate-Intensity User	50th %ile (15)	Mid (60)	50th %ile (191.6)	User	6.60E+01	9.06
				Bystander	1.48E+01	2.26
Low-Intensity User	10th %ile (1)	Min (20)	10th %ile (47.9)	User	5.16	7.09E-01
				Bystander	1.19	1.81E-01

[1]Actual product weight fractions were: 0-20%; 45-55%; 97.5%; 90-100%. 60% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-33. Acute Dermal Exposure Summary: Brake & Parts Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (120)	Max (100)	Adult (≥21 years)	7.63E+01
			Children (16-20 years)	7.14E+01
			Children (11-15 years)	7.80E+01
Central Tendency	50th %ile (15)	Mid (60)	Adult (≥21 years)	5.72
			Children (16-20 years)	5.35
			Children (11-15 years)	5.85
Low-Intensity User	10th %ile (1)	Min (20)	Adult (≥21 years)	1.27E-01
			Children (16-20 years)	1.19E-01
			Children (11-15 years)	1.30E-01

[1]Actual product weight fractions were: 0-20%; 45-55%; 97.5%; 90-100%. 60% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.

Aerosol Electronic Degreaser/Cleaner

Exposure to TCE in aerosol electronic degreasing/cleaning products was evaluated based on nine aerosol products with weight fractions ranging from 30-100% TCE.

Westat Survey data on specialized electronics cleaners were used as the basis for duration of use and mass of product used. Survey responses indicate 13.1% of respondents have used products in this category; 34% reported use of aerosol formulations and 56% reported use of liquid formulations. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the utility room (20 m^3) although the Westat survey data for this category indicate living room and other inside room as the top two locations of reported use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-34. Acute Inhalation Exposure Summary: Aerosol Electronic Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	Max (100)	95th %ile (337.1)	User	2.81E+02	3.76E+01
				Bystander	5.03E+01	7.56
Moderate-Intensity User	50th %ile (2)	Mid (65)	50th %ile (22.5)	User	1.19E+01	1.58
				Bystander	1.96	2.95E-01
Low-Intensity User	10th %ile (0.5)[2]	Min (30)	10th %ile (1.8)	User	4.15E-01	5.55E-02
				Bystander	7.21E-02	1.08E-02

[1]Actual product weight fractions were: 30-50%; 30-60%; 97.2%; 98%; 60-100%; and 90-100%. 65% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Liquid Electronic Degreaser/Cleaner

Exposure to TCE in liquid electronic degreasing/cleaning products was evaluated based on one liquid product with a weight fraction of 100% TCE.

Westat Survey data on specialized electronics cleaners were used as the basis for duration of use and mass of product used. Survey responses indicate 13.1% of respondents have used products in this category; 34% reported use of aerosol formulations and 56% reported use of liquid formulations. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the utility room (20 m^3) although the Westat survey data for this category indicate living room and other inside room as the top two locations of reported use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-35. Acute Inhalation Exposure Summary: Liquid Electronic Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(100)	95th %ile (337.1)	User	2.70E+02	3.61E+01
				Bystander	4.83E+01	7.26
Moderate-Intensity User	50th %ile (2)	(100)	50th %ile (22.5)	User	1.75E+01	2.33
				Bystander	2.90	4.36E-01
Low-Intensity User	10th %ile (0.5)[2]	(100)	10th %ile (1.8)	User	1.30	1.74E-01
				Bystander	2.27E-01	3.41E-02

[1]Single weight fraction of 100% available.
[2]The 10th percentile duration from Westat was < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-36. Acute Dermal Exposure Summary: Liquid Electronic Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (30)	(100)	Adult (≥21 years)	4.30E+01
			Children (16-20 years)	4.03E+01
			Children (11-15 years)	4.39E+01
Moderate-Intensity User	50th %ile (2)	(100)	Adult (≥21 years)	2.88
			Children (16-20 years)	2.68
			Children (11-15 years)	2.92
Low-Intensity User	10th %ile (0.5)[2]	(100)	Adult (≥21 years)	7.15E-01
			Children (16-20 years)	6.70E-01
			Children (11-15 years)	7.31E-01

[1] Single weight fraction of 100% available.
[2] The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Aerosol Spray Degreaser/Cleaner

Exposure to TCE in aerosol spray degreaser/cleaner products was evaluated based on eight aerosol products with weight fractions ranging from 60-100% TCE.

Westat Survey data on engine degreasing were used as the basis for duration of use and mass of product used. Survey responses indicate that 17.2% of respondents have used products in this category; 78.9% reported use of aerosol formulations. The room of use (Zone 1) was set to the garage (90 m^3) although the Westat survey data for this category indicate primarily outdoor use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-37. Acute Inhalation Exposure Summary: Aerosol Spray Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (120)	Max (100)	95th %ile (2157.4)	User	1.12E+03	1.62E+02
				Bystander	2.82E+02	4.71E+01
Moderate-Intensity User	50th %ile (15)	Max (100)	50th %ile (521.4)	User	2.99E+02	4.11E+01
				Bystander	6.70E+01	1.02E+01
Low-Intensity User	10th %ile (5)	Min (60)	10th %ile (130.8)	User	4.54E+01	6.20
				Bystander	9.83	1.50

[1]Actual product weight fractions were: 60-100% and 90-100%.

This condition of use was also assessed in the 2014 TSCA Work Plan Chemical Risk Assessment and refined in the 2016 Supplemental Exposure and Risk Reduction Technical Report in Support of Risk management Options for TCE (TCE) Use in Consumer Aerosol Degreasing. In these prior assessments, different inputs were used for certain modeling parameters including mass used and duration of use. Please see the referenced documents for full details. The amount used (24 g TCE – roughly 27 g product) in the 2014 assessment is much lower than the 10th percentile input obtained from the Westat survey engine degreasing scenario. The lower amount applied in 2014 more closely reflects an aerosol electronic cleaning condition of use, which is characterized by a median mass used of 0.5 oz, or 22.5 g. It is therefore unlikely that the previous assessment captured exposures for consumer involved in larger degreasing efforts such as engine degreasing or brake cleaning.The inputs and associated 24-hr acute air concentrations for users and bystanders from the 2014 assessment are shown below.

2014 Acute Inhalation Exposure Summary: Aerosol Spray Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass Used (g)	Product User or Bystander	24-hr TWA (ppm)
2014 Work Plan Chemical Risk Assessment	60	90	(24)[1]	User	2.9[2]
				Bystander	0.8

[1]Note that this conversion assumes a formulation density of 1. Actual product densities range from 1.46-1.52 g/cm^3. This input is also provided in terms of mass of TCE per use, rather than mass of product per use, which is the actual model input. 24 g of TCE in this 90% formulation would equate to roughly 27 g of product per use.
[2]This user air concentration was shown in the 2014 assessment as 2 ppm; however, in the 2016 supplemental report, it was corrected to 2.9 ppm due to an earlier rounding error or typo.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-38. Acute Dermal Exposure Summary: Aerosol Spray Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (120)	Max (100)	Adult (≥21 years)	7.16E+01
			Children (16-20 years)	6.70E+01
			Children (11-15 years)	7.32E+01
Moderate-Intensity User	50th %ile (15)	Max (100)	Adult (≥21 years)	8.94
			Children (16-20 years)	8.37
			Children (11-15 years)	9.15
Low-Intensity User	10th %ile (5)	Min (60)	Adult (≥21 years)	1.79
			Children (16-20 years)	1.67
			Children (11-15 years)	1.83

[1]Actual product weight fractions were: 60-100% and 90-100%.

Liquid Degreaser/Cleaner

Exposure to TCE in liquid degreasing/cleaning products was evaluated based on two aerosol products with weight fractions ranging from 90-100% TCE.

Westat Survey data on solvent-type cleaning fluids or degreasers were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 28.1% of respondents have used products in this category; 74.4% reported use of liquid formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-39. Acute Inhalation Exposure Summary: Liquid Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (120)	(100)	95th %ile (1337.7)	User	1.05E+03	1.46E+02
				Bystander	2.28E+02	3.61E+01
Moderate-Intensity User	50th %ile (15)	(100)	50th %ile (139.9)	User	1.17E+02	1.56E+01
				Bystander	1.97E+01	2.96
Low-Intensity User	10th %ile (2)	(100)	10th %ile (24.1)	User	1.95E+01	2.60
				Bystander	3.24	4.86E-01

[1]Actual product weight fractions were: 90-100% and 100%.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-40. Acute Dermal Exposure Summary: Liquid Degreaser/Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (120)	(100)	Adult (≥21 years)	1.71E+02
			Children (16-20 years)	1.60E+02
			Children (11-15 years)	1.75E+02
Moderate-Intensity User	50th %ile (15)	(100)	Adult (≥21 years)	2.14E+01
			Children (16-20 years)	2.01E+01
			Children (11-15 years)	2.19E+01
Low-Intensity User	10th %ile (2)	(100)	Adult (≥21 years)	2.85
			Children (16-20 years)	2.68
			Children (11-15 years)	2.92

[1]Actual product weight fractions were: 90-100% and 100%.

Aerosol Gun Scrubber

Exposure to TCE in aerosol gun scrubber/cleaner products was evaluated based on two aerosol products. Only one product had a reported weight fraction (97%), so modeling was based on the full range of aerosol degreasing formulation weight fractions (60-100%).

Westat Survey data on solvent-type cleaning fluids or degreasers were used as the basis for room of use and duration, while manufacturer data on the amount of product required to coat a firearm in a very thin film were used as the basis for the mass of product used. The Westat survey product category selected was not aligned well with this specific use, but the duration data for the selected category was deemed reasonable for use in modeling. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-41. Acute Inhalation Exposure Summary: Aerosol Gun Scrubber

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (120)	Max (100)	(0.7)	User	5.35E-01	7.44E-02
				Bystander	1.16E-01	1.83E-02
Moderate-Intensity User	50th %ile (15)	Max (100)	(0.7)	User	5.87E-01	7.83E-02
				Bystander	9.87E-02	1.48E-02
Low-Intensity User	10th %ile (2)	Min (60)	(0.7)	User	3.41E-01	4.55E-02
				Bystander	5.64E-02	8.47E-03

[1]Only one product had a reported weight fraction (97%), so modeling was based on the full range of aerosol degreasing formulation weight fractions (60-100%).

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-42. Acute Dermal Exposure Summary: Aerosol Gun Scrubber

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (120)	Max (100)	Adult (≥21 years)	6.90E+01
			Children (16-20 years)	6.45E+01
			Children (11-15 years)	7.06E+01
Moderate-Intensity User	50th %ile (15)	Max (100)	Adult (≥21 years)	8.62
			Children (16-20 years)	8.07
			Children (11-15 years)	8.82
Low-Intensity User	10th %ile (2)	Min (60)	Adult (≥21 years)	6.90E-01
			Children (16-20 years)	6.48E-01
			Children (11-15 years)	7.08E-01

[1]Only one product had a reported weight fraction (97%), so modeling was based on the full range of aerosol degreasing formulation weight fractions (60-100%).

Liquid Gun Scrubber

Exposure to TCE in liquid gun scrubber/cleaner products was evaluated based on one liquid product with an unreported weight fraction. Modeling was based on the upper-end of the narrow range of liquid degreasing formulation weight fractions (90-100%).

Westat Survey data on solvent-type cleaning fluids or degreasers were used as the basis for room of use and duration, while manufacturer data on the amount of product required to coat a firearm in a very thin film were used as the basis for the mass of product used. The Westat survey product category selected was not aligned well with this specific use, but the duration data for the selected category was deemed reasonable for use in modeling. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-43. Acute Inhalation Exposure Summary: Liquid Gun Scrubber

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (120)	(100)	(0.7)	User	4.58E-01	6.37E-02
				Bystander	9.94E-02	1.57E-02
Moderate-Intensity User	50th %ile (15)	(100)	(0.7)	User	5.03E-01	6.71E-02
				Bystander	8.46E-02	1.27E-02
Low-Intensity User	10th %ile (2)	(100)	(0.7)	User	4.65E-01	6.22E-02
				Bystander	8.09E-02	1.22E-02

[1]Modeling was based on the upper-end of the narrow range of liquid degreasing formulation weight fractions (90-100%).

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-44. Acute Dermal Exposure Summary: Liquid Gun Scrubber

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (120)	(100)	Adult (≥21 years)	1.60E+02
			Children (16-20 years)	1.50E+02
			Children (11-15 years)	1.63E+02
Moderate-Intensity User	50th %ile (15)	(100)	Adult (≥21 years)	2.00E+01
			Children (16-20 years)	1.87E+01
			Children (11-15 years)	2.04E+01
Low-Intensity User	10th %ile (2)	(100)	Adult (≥21 years)	2.68
			Children (16-20 years)	2.50
			Children (11-15 years)	2.72

[1]Modeling was based on the upper-end of the narrow range of liquid degreasing formulation weight fractions (90-100%).

Mold Release

Exposure to TCE in mold release products was evaluated based on two aerosol products with weight fractions ranging from 40-68.9% TCE.

Westat Survey data on other lubricants (excluding automotive) were used as the basis for room of use, duration of use, and mass of product used. For this product scenario, EPA believes that the selected lubricant Westat scenario, although not a direct match with mold release products, better aligns with the product use pattern when compared against other options, such as solvent-type cleaning fluid, which conveys a much higher use duration and mass used. Survey responses indicate that 34.5% of respondents have used products in this category; 32.5% reported use of aerosol formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-45. Acute Inhalation Exposure Summary: Mold Release

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	Max (68.9)	95th %ile (212.9)	User	1.22E+02	1.64E+01
				Bystander	2.19E+01	3.29
Moderate-Intensity User	50th %ile (2)	Max (68.9)	50th %ile (23.4)	User	1.31E+01	1.75
				Bystander	2.16	3.25E-01
Low-Intensity User	10th %ile (0.5)[2]	Min (40)	10th %ile (4.3)	User	1.32	1.77E-01
				Bystander	2.30E-01	3.45E-02

[1]Actual product weight fractions were: 40-50% and 68.9%.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Aerosol Tire Cleaner

Exposure to TCE in aerosol tire cleaning products was evaluated based on two aerosol products with weight fractions ranging from 70-100% TCE.

Westat Survey data on tire and hubcap cleaners were used as the basis for duration of use and mass of product used. Survey responses indicate that 15.9% of respondents have used products in this category; 29.5% reported use of aerosol formulations and 70.5% reported use of liquid formulations. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the garage (90 m^3) although the Westat survey data for this category indicate primarily outdoor use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer*

Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500] for the full range of results based on all iterations of this modeling scenario.

Table 2-46. Acute Inhalation Exposure Summary: Aerosol Tire Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	Max (100)	95th %ile (317)	User	1.04E+02	1.57E+01
				Bystander	4.39E+01	6.84
Moderate-Intensity User	50th %ile (15)	Max (100)	50th %ile (52.9)	User	3.04E+01	4.17
				Bystander	6.80	1.04
Low-Intensity User	10th %ile (5)	Min (70)	10th %ile (10.5)	User	4.25	5.81E-01
				Bystander	9.21E-01	1.40E-01

[1]Actual product weight fractions were: 70-90% and 80-100%.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-47. Acute Dermal Exposure Summary: Aerosol Tire Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (60)	Max (100)	Adult (≥21 years)	1.58E+01
			Children (16-20 years)	1.48E+01
			Children (11-15 years)	1.61E+01
Moderate-Intensity User	50th %ile (15)	Max (100)	Adult (≥21 years)	3.94
			Children (16-20 years)	3.69
			Children (11-15 years)	4.03
Low-Intensity User	10th %ile (5)	Min (70)	Adult (≥21 years)	9.17E-01
			Children (16-20 years)	8.61E-01
			Children (11-15 years)	9.38E-01

[1]Actual product weight fractions were: 70-90% and 80-100%.

Liquid Tire Cleaner

Exposure to TCE in liquid tire cleaning products was evaluated based on one liquid product with a weight fractions ranging of 80-100% TCE.

Westat Survey data on tire and hubcap cleaners were used as the basis for duration of use and mass of product used. Survey responses indicate that 15.9% of respondents have used products in this category; 29.5% reported use of aerosol formulations and 70.5% reported use of liquid formulations. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the garage (90 m^3) although the Westat survey data for this category indicate primarily outdoor use.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-48. Acute Inhalation Exposure Summary: Liquid Tire Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(100)	95th %ile (706.4)	User	3.33E+02	4.76E+01
				Bystander	9.79E+01	1.52E+01
Moderate-Intensity User	50th %ile (15)	(100)	50th %ile (117.9)	User	6.77E+01	9.28
				Bystander	1.52E+01	2.32
Low-Intensity User	10th %ile (5)	(100)	10th %ile (23.4)	User	1.35E+01	1.85
				Bystander	2.93	4.47E-01

[1]Single weight fraction of 80-100% available.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-49. Acute Dermal Exposure Summary: Liquid Tire Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (60)	(100)	Adult (≥21 years)	8.78E+01
			Children (16-20 years)	8.23E+01
			Children (11-15 years)	8.99E+01
Moderate-Intensity User	50th %ile (15)	(100)	Adult (≥21 years)	2.20E+01
			Children (16-20 years)	2.06E+01
			Children (11-15 years)	2.24E+01
Low-Intensity User	10th %ile (5)	(100)	Adult (≥21 years)	7.33
			Children (16-20 years)	6.85
			Children (11-15 years)	7.49

[1]Single weight fraction of 80-100% available.

Lubricants and Greases

Tap & Die Fluid

Exposure to TCE in tap & die fluid was evaluated based on one aerosol product with a weight fraction of 98% TCE.

Westat Survey data on other lubricants (excluding automotive) were used to select room of use, duration of use, and mass of product used. Survey responses indicated that 34.5% of respondents have used

products in this category; 32.5% reported use of aerosol formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-50. Acute Inhalation Exposure Summary: Tap & Die Fluid

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(98)	95th %ile (134.5)	User	1.10E+02	1.47E+01
				Bystander	1.97E+01	2.95
Moderate-Intensity User	50th %ile (2)	(98)	50th %ile (14.8)	User	1.18E+01	1.57
				Bystander	1.95	2.93E-01
Low-Intensity User	10th %ile (0.5)[2]	(98)	10th %ile (2.7)	User	2.03	2.78E-01
				Bystander	4.96E-01	8.53E-02

[1]Single weight fraction of 98% available.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Penetrating Lubricant

Exposure to TCE in lubricant products was evaluated based on five aerosol products with weight fractions ranging from 5-50 % TCE.

Westat Survey data on other lubricants (excluding automotive) were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 34.5% of respondents have used products in this category; 32.5% reported use of aerosol formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-51. Acute Inhalation Exposure Summary: Penetrating Lubricant

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	Max (50)	95th %ile (209.9)	User	8.74E+01	1.17E+01
				Bystander	1.56E+01	2.35
Moderate-Intensity User	50th %ile (2)	Mid (27.5)	50th %ile (23.1)	User	5.16	6.88E-01
				Bystander	8.53E-01	1.28E-01

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
Low-Intensity User	10th %ile (0.5)[2]	Min (5)	10th %ile (4.2)	User	1.62E-01	2.16E-02
				Bystander	2.80E-02	4.21E-03

[1]Actual product weight fractions were: 5-10%; 10-20%; 30-40%; 48.8%; and 30-50%. 27.5% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Adhesives and Sealants

Solvent-based Adhesive & Sealant

Exposure to TCE in solvent-based adhesive & sealant products was evaluated based on three liquid products with weight fractions ranging from 5->90% TCE.

Westat Survey data on contact cement, superglue, and spray adhesive were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 60.6% of respondents have used products in this category; 97.1% reported use of liquid formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-52. Acute Inhalation Exposure Summary: Solvent-based Adhesive & Sealant

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	Max (90)	95th %ile (185.2)	User	2.46E+02	3.22E+01
				Bystander	2.68E+01	4.06
Moderate-Intensity User	50th %ile (4.25)	Mid (47.5)	50th %ile (10.7)	User	7.76	1.00
				Bystander	6.86E-01	1.03E-01
Low-Intensity User	10th %ile (0.5)[2]	Min (5)	10th %ile (1.3)	User	6.72E-02	8.83E-03
				Bystander	8.68E-03	1.30E-03

[1]Actual product weight fractions were: 5-15%; 40-60; and >90%. 47.5% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Mirror-edge Sealant

Exposure to TCE in mirror-edge sealant products was evaluated based on one aerosol product with a weight fraction of 20-40% TCE.

Westat Survey data on contact cement, superglue, and spray adhesive were used as the basis for duration of use and mass of product used. While there was no Westat scenario that directly aligned with use as a mirror-edge sealant, the selected category is believed to be the best fit based on the associated range of use duration and mass used. Survey responses indicate that 60.6% of respondents have used products in this category; 97.1% reported use of liquid formulations. While the formulation type used by the majority of respondents for this category does not reflect the modeled use, which is an aerosol, it represents the best fit category available. The room of use (Zone 1) was set to the bathroom (15 m^3) based on the product's apparent use on mirror edging.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-53. Acute Inhalation Exposure Summary: Mirror-Edge Sealant

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(40)	95th %ile (78.4)	User	2.45E+01	3.33
				Bystander	5.21	7.84E-01
Moderate-Intensity User	50th %ile (4.25)	(40)	50th %ile (4.5)	User	8.31	1.11
				Bystander	1.34	2.01E-01
Low-Intensity User	10th %ile (0.5)[2]	(40)	10th %ile (0.5)	User	1.68E-01	2.24E-02
				Bystander	2.71E-02	4.07E-03

[1]Single weight fraction of 20-40% available.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Tire Repair Cement/Sealer

Exposure to TCE in tire repair products was evaluated based on five liquid products with weight fractions ranging from 65-95% TCE.

Westat Survey data on contact cement, superglue, and spray adhesive were used as the basis for duration of use and mass of product used. Survey responses indicate that 60.6% of respondents have used products in this category; 97.1% reported use of liquid formulations. The room of use (Zone 1) was set to the garage (90 m^3) based on the product's apparent use on tires.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-54. Acute Inhalation Exposure Summary: Tire Repair cement/Sealer

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	Max (95)	95th %ile (185.2)	User	8.30E+01	1.18E+01
				Bystander	2.44E+01	3.80
Moderate-Intensity User	50th %ile (4.25)	Mid (80)	50th %ile (10.7)	User	4.85	6.64E-01
				Bystander	1.07	1.63E-01
Low-Intensity User	10th %ile (0.5)[2]	Min (65)	10th %ile (1.3)	User	4.32E-01	5.97E-02
				Bystander	1.05E-01	1.59E-02

[1]Actual product weight fractions were: 65-80%; 70-85%; 75-90%; and 80-95%. 80% is a mathematically-derived mid-point (i.e., mean) for use in modeling, based on the minimum and maximum inputs.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Cleaning and Furniture Care Products

Carpet Cleaner

Exposure to TCE in carpet cleaner was evaluated based on a single liquid formulation with a weight fraction of >99% TCE.

Westat Survey data on spot removers were used to select the duration of use and mass of product used. Survey responses indicate that 39.1% of respondents have used products in this category; 43.9% reported use of a liquid formulation. The room of use (Zone 1) was set to the bedroom (36 m^3) based on professional judgement. There are no data in the Westat Survey exactly matching a use as a carpet cleaner; therefore, data reflecting spot cleaners were applied.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-55. Acute Inhalation Exposure Summary: Carpet Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(99)	95th %ile (526.6)	User	3.90E+02	5.26E+01
				Bystander	7.65E+01	1.15E+01
Moderate-Intensity User	50th %ile (5)	(99)	50th %ile (62.9)	User	4.75E+01	6.36
				Bystander	8.39	1.26
Low-Intensity User	10th %ile (0.5)[2]	(99)	10th %ile (11.8)	User	8.14	1.10
				Bystander	1.55	2.33E-01

[1]Single weight fraction of >99% available.

[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-56. Acute Dermal Exposure Summary: Carpet Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (30)	(99)	Adult (≥21 years)	4.65E+01
			Children (16-20 years)	4.36E+01
			Children (11-15 years)	4.77E+01
Central-Tendency	50th %ile (5)	(99)	Adult (≥21 years)	7.77
			Children (16-20 years)	7.28
			Children (11-15 years)	7.93
Low-Intensity User	10th %ile (0.5)[2]	(99)	Adult (≥21 years)	3.89E-01
			Children (16-20 years)	3.64E-01
			Children (11-15 years)	3.98E-01

[1] Single weight fraction of >99% available.
[2] The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Aerosol Spot Remover

Exposure to TCE in aerosol spot remover products was evaluated based on one aerosol product with a weight fraction of 20-30% TCE.

Westat Survey data on spot removers were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 39.1% of respondents have used products in this category; 43.9% reported use of a liquid formulation and 56.1% reported use of an aerosol formulation. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-57. Acute Inhalation Exposure Summary: Aerosol Spot Remover

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(30)	95th %ile (514.1)	User	2.50E+02	3.24E+01
				Bystander	2.28E+01	3.43

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
Moderate-Intensity User	50th %ile (5)	(30)	50th %ile (61.4)	User	2.93E+01	3.78
				Bystander	2.49	3.75E-01
Low-Intensity User	10th %ile (0.5)[2]	(30)	10th %ile (11.15)	User	4.34	5.65E-01
				Bystander	4.59E-01	6.90E-02

[1]Single weight fraction of 20-30% available.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-58. Acute Dermal Exposure Summary: Aerosol Spot Remover

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (30)	(30)	Adult (≥21 years)	5.52
			Children (16-20 years)	5.16
			Children (11-15 years)	5.64
Moderate-Intensity User	50th %ile (5)	(30)	Adult (≥21 years)	9.18E-01
			Children (16-20 years)	8.61E-01
			Children (11-15 years)	9.42E-01
Low-Intensity User	10th %ile (0.5)[2]	(30)	Adult (≥21 years)	9.18E-02
			Children (16-20 years)	8.61E-02
			Children (11-15 years)	9.42E-02

[1] Single weight fraction of 20-30% available.
[2] The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Liquid Spot Remover

Exposure to TCE in liquid spot remover products was evaluated based on four liquid products with weight fractions ranging from 50-75%.

Westat Survey data on spot removers were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 39.1% of respondents have used products in this category; 43.9% reported use of a liquid formulation and 56.1% reported use of an aerosol formulation. Therefore, these Westat data were applied to both aerosol and liquid product scenarios. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-59. Acute Inhalation Exposure Summary: Liquid Spot Remover

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	Max (75)	95th %ile (477.2)	User	2.98E+02	3.99E+01
				Bystander	5.34E+01	8.02
Moderate-Intensity User	50th %ile (5)	Max (75)	50th %ile (57)	User	3.55E+01	4.73
				Bystander	5.80	8.72E-01
Low-Intensity User	10th %ile (0.5)[2]	Min (50)	10th %ile (10.7)	User	4.09	5.47E-01
				Bystander	7.14E-01	1.07E-01

[1]Actual product weight fractions were: <50%; <75%; and >75%.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-60. Acute Dermal Exposure Summary: Liquid Spot Remover

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (30)	Max (75)	Adult (≥21 years)	3.21E+01
			Children (16-20 years)	3.00E+01
			Children (11-15 years)	3.28E+01
Moderate-Intensity User	50th %ile (5)	Max (75)	Adult (≥21 years)	5.33
			Children (16-20 years)	4.99
			Children (11-15 years)	5.45
Low-Intensity User	10th %ile (0.5)[2]	Min (50)	Adult (≥21 years)	3.55E-01
			Children (16-20 years)	3.33E-01
			Children (11-15 years)	3.63E-01

[1] Actual product weight fractions were: <50%; <75%; and >75%.
[2] The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

Arts, Crafts, and Hobby Materials

Fixatives & Finishing Spray Coating

Exposure to TCE in fixatives & finishing spray coating products was evaluated based on one aerosol product with a weight fraction of 20-30% TCE.

Westat Survey data on aerosol rust removers were used as the basis for duration of use and mass of product used. This Westat category was selected as a surrogate, as there were no well-aligned product categories for this use. However, survey responses for the selected surrogate category reported 98.3% use of aerosol formulations, which is supportive of its application to the modeled product scenario. Duration of use and mass of product data were also reviewed for reasonableness and were considered

more reasonable (i.e., lower) than the higher use patterns associated with most of the solvent degreasing or cleaning categories. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-61. Acute Inhalation Exposure Summary: Fixatives & Finishing Spray Coatings

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(30)	95th %ile (306)	User	6.83E+01	9.31
				Bystander	1.51E+01	2.28
Moderate-Intensity User	50th %ile (5)	(30)	50th %ile (45.2)	User	1.13E+01	1.50
				Bystander	1.84	2.77E-01
Low-Intensity User	10th %ile (0.5)[2]	(30)	10th %ile (9.4)	User	2.17	2.90E-01
				Bystander	3.76E-01	5.66E-02

[1]Single product weight fraction of 20-30% available.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

This condition of use was also assessed in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). In the prior assessment, different inputs were used for certain modeling parameters including mass used and duration of use. The amount of TCE used (11 g – roughly 37 g of product) in the 2014 assessment is roughly equivalent to the 50th percentile input obtained from the Westat survey rust remover surrogate scenario applied in this latest evaluation. These inputs and associated 24-hr acute air concentrations for users and bystandersare included below.

2014 Acute Inhalation Exposure Summary: Fixatives & Finishing Spray Coatings

Scenario Description	Duration of Use (min)	Weight Fraction (%)	Mass Used (g)	Product User or Bystander	24-hr TWA (ppm)
2014 Chemical Work Plan Risk Assessment	30	30	11[1]	User	0.4
				Bystander	0.1

[1]Note that this conversion assumes a formulation density of 1. Actual product densities range from 1.46-1.52 g/cm^3. This input is also provided in terms of mass of TCE per use, rather than mass of product per use, which is the actual model input. 11 g of TCE in this 30% formulation would equate to roughly 37 g of product per use, which is similar to the central tendency input used in the current evaluation.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Apparel and Footwear care Products

Shoe Polish

Exposure to TCE in shoe polish products was evaluated based on one aerosol product with a weight fraction of 10-20% TCE.

Westat Survey data on spray shoe polish were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 11.7% of respondents have used products in this

category; 97.7% reported use of aerosol formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-62. Acute Inhalation Exposure Summary: Shoe Polish

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(20)	95th %ile (151.4)	User	2.52E+01	3.38
				Bystander	4.52	6.79E-01
Moderate-Intensity User	50th %ile (5)	(20)	50th %ile (15.4)	User	2.56	3.41E-01
				Bystander	4.18E-01	6.28E-02
Low-Intensity User	10th %ile (0.5)	(20)	10th %ile (2.9)	User	4.46E-01	5.96E-02
				Bystander	7.74E-02	1.16E-02

[1]Single weight fraction of 10-20% available.

Dermal exposures are also presented for this scenario, as it is assumed that the product could be applied in a manner leading to dermal contact with impeded evaporation, thereby increasing the likelihood and/or duration of dermal absorption. There is uncertainty surrounding the duration of dermal contact with impeded evaporation. The exposure durations modeled could exceed the duration of such dermal contact; therefore, the higher-end durations may result in an overestimation of dermal exposure.

Table 2-63. Acute Dermal Exposure Summary: Shoe Polish

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Receptor	Acute ADR (mg/kg/day)
High-Intensity User	95th %ile (30)	(20)	Adult (≥21 years)	3.02
			Children (16-20 years)	2.82
			Children (11-15 years)	3.08
Moderate-Intensity User	50th %ile (5)	(20)	Adult (≥21 years)	5.00E-01
			Children (16-20 years)	4.70E-01
			Children (11-15 years)	5.14E-01
Low-Intensity User	10th %ile (0.5)	(20)	Adult (≥21 years)	5.00E-02
			Children (16-20 years)	4.70E-02
			Children (11-15 years)	5.14E-02

[1]Single weight fraction of 10-20% available.

Other Consumer Uses

Fabric Spray

Exposure to TCE in fabric spray products was evaluated based on one aerosol product with a weight fraction of 20-40% TCE. This use (i.e., no-fray fabric spray) was originally identified in the 2014 TSCA Work Plan Chemical Risk Assessment of TCE (U.S. EPA, 2014b).

Westat Survey data on water repellents/protectors for suede, leather, and cloth were used as the basis for room of use, duration of use, and mass of product used. Survey responses indicate that 35.5% of respondents have used products in this category; 72.1% reported use of aerosol formulations. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-64. Acute Inhalation Exposure Summary: Fabric Spray

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(40)	95th %ile (326.8)	User	1.93E+02	2.53E+01
				Bystander	2.10E+01	3.18
Moderate-Intensity User	50th %ile (10)	(40)	50th %ile (49.9)	User	3.24E+01	4.18
				Bystander	2.75	4.13E-01
Low-Intensity User	10th %ile (1.4)	(40)	10th %ile (11.4)	User	5.64	7.35E-01
				Bystander	6.09E-01	9.15E-02

[1]Single product weight fraction of 20-40% available.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Film Cleaner

Exposure to TCE in film cleaner products was evaluated based on two aerosol products with weight fractions ranging 80-100% TCE.

Westat Survey data on aerosol rust removers were used as the basis for duration of use and mass of product used. This Westat category was selected as a surrogate, as there were no well-aligned product categories for this use. However, survey responses for the selected surrogate category reported 98.3% use of aerosol formulations, which is supportive of its application to the modeled product scenario. Duration of use and mass of product data were also reviewed for reasonableness and were considered more reasonable (i.e., lower) than the higher use patterns associated with most of the solvent degreasing or cleaning categories. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-65. Acute Inhalation Exposure Summary: Film Cleaner

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(100)	95th %ile (632.9)	User	4.71E+02	6.42E+01
				Bystander	1.04E+02	1.57E+01
Moderate-Intensity User	50th %ile	(100)	50th %ile	User	7.77E+01	1.03E+01

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
	(5)		(93.4)	Bystander	1.27E+01	1.91
Low-Intensity User	10th %ile (0.5)[2]	(100)	10th %ile (19.4)	User	1.49E+01	1.99
				Bystander	2.59	3.89E-01

[1]Actual product weight fractions were: 80-100% and 95%.
[2]The 10th percentile duration from Westat is < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Hoof Polish

Exposure to TCE in hoof polish products was evaluated based on one aerosol product with an unreported weight fraction. Modeling was based on the upper-end of the narrow range of shoe polish and spray fixative/coating formulation weight fractions (20-30%).

Westat Survey data on spray shoe polish were used as the basis for duration of use and mass of product used. This Westat category was selected as a surrogate, as there were no well-aligned product categories for this use. Survey data indicate that 11.7% of respondents used products in this category; 97.7% reported use of aerosol formulations. The room of use (Zone 1) was set to approximate a barn environment. This was done by using a garage (90 m^3) but increasing the default air exchange rate of a residential room from 0.45 to 4 air exchanged per hour, which was based on recommended ventilation rates for a horse stable (Pennsylvania State University, 2016).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-66. Acute Inhalation Exposure Summary: Hoof Polish

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (30)	(30)	95th %ile (208.2)	User	1.76E+01	2.21
				Bystander	8.83E-02	1.10E-02
Moderate-Intensity User	50th %ile (5)	(30)	50th %ile (21.2)	User	1.73	2.16E-01
				Bystander	3.81E-03	4.76E-04
Low-Intensity User	10th %ile (0.5)	(30)	10th %ile (4)	User	2.46E-01	3.08E-02
				Bystander	6.23E-04	7.79E-05

[1]Actual weight fraction is not reported; modeling was based on the upper-end of the narrow range of shoe polish and spray fixative/coating formulation weight fractions (20-30%).

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Pepper Spray

Exposure to TCE in pepper spray products was evaluated based on two aerosol products with a single reported weight fraction of 91.5% TCE.

Internal research was the basis for duration of use and mass of product used. One spray from the most common civilian canister is estimated to be approximately 0.0216-0.108 ounces, based on information on a pepper spray manufacturer's website. Spraying occurred between 3 and 5 seconds (0.05-0.08 min) before obtaining desired effect (Bertilsson et al., 2017). The room of use (Zone 1) was set to approximate a "cloud" around the user (16 m^3) in an outdoor environment. This was done by increasing the default air exchange rate of a residential room from 0.45 to 100 air exchanges per hour. Since the interzonal ventilation rate for this "outdoor" scenario is held at 0, there are no bystander exposures estimated. Based on the limited parameter data for this scenario, no inputs were varied.

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-67. Acute Inhalation Exposure Summary: Pepper Spray

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
Single Scenario	(0.5)[2]	(91.5)	(4)	User	1.42E-01	1.77E-02
				Bystander	1.42E-01	1.77E-02

[1]Single weight fraction of 91.5% available.
[2]The selected < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.
[3]Bystander in the home not modeled due to simulated outdoor scenario - can be considered equal to user.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

Toner Aid

Exposure to TCE in toner aid products was evaluated based on one aerosol product with a weight fraction of 10-20% TCE.

Westat Survey data on aerosol rust removers were used as the basis for duration of use and mass of product used. This Westat category was selected as a surrogate, as there were no well-aligned product categories for this use. However, survey responses for the selected surrogate category reported 98.3% use of aerosol formulations, which is supportive of its application to the modeled product scenario. Duration of use and mass of product data were also reviewed for reasonableness and were considered more reasonable (i.e., lower) than the higher use patterns associated with most of the solvent degreasing or cleaning categories. The room of use (Zone 1) was set to the utility room (20 m^3).

Inhalation exposures for users and bystanders are presented below reflecting high-, moderate-, and low-intensity user scenarios. See Supplemental File [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures. Docket: EPA-HQ-OPPT-2019-0500*] for the full range of results based on all iterations of this modeling scenario.

Table 2-68. Acute Inhalation Exposure Summary: Toner Aid

Scenario Description	Duration of Use (min)	Weight Fraction[1] (%)	Mass Used (g)	Product User or Bystander	3-hr Max TWA (ppm)	24-hr Max TWA (ppm)
High-Intensity User	95th %ile (60)	(20)	95th %ile (434.7)	User	6.47E+01	8.82
				Bystander	1.43E+01	2.16
Moderate-Intensity User	50th %ile (5)	(20)	50th %ile (64.2)	User	1.07E+01	1.42
				Bystander	1.74	2.62E-01
Low-Intensity User	10th %ile (0.5)[2]	(20)	10th %ile (13.3)	User	2.05	2.73E-01
				Bystander	3.55E-01	5.34E-02

[1]Single weight fraction of 10-20% available.
[2]The selected < 0.5 minutes, but 0.5 minutes was used in the model, as it reflects the smallest timestep in the model run.

There are no dermal exposures quantified for this scenario, as this use pattern is not expected to involve dermal contact with impeded evaporation.

2.3.2.6.3 Summary of Consumer Exposure Assessment

Table 2-69 displays the consumer conditions of use evaluated for acute inhalation and/or dermal exposures.

Table 2-69. Evaluated Pathways for Consumer Conditions of Use

Life Cycle Stage	Categories	Product Subcategories	Form	Acute Inhalation Exposure	Acute Dermal Exposure
Use	Solvents for Cleaning and Degreasing	Brake & Parts Cleaner	Aerosol	✓	✓
		Electronic Degreaser/Cleaner	Aerosol	✓	
		Electronic Degreaser/Cleaner	Liquid	✓	✓
		Aerosol Spray Degreaser/Cleaner	Aerosol	✓	✓
		Liquid Degreaser/Cleaner	Liquid	✓	✓
		Gun Scrubber	Aerosol	✓	✓
		Gun Scrubber	Liquid	✓	✓
		Mold Release	Aerosol	✓	
		Tire Cleaner	Aerosol	✓	✓
		Tire Cleaner	Liquid	✓	✓
	Lubricants and Greases	Tap & Die Fluid	Aerosol	✓	
		Penetrating Lubricant	Aerosol	✓	
	Adhesives and Sealants	Solvent-based Adhesive & Sealant	Liquid	✓	
		Mirror-edge Sealant	Aerosol	✓	
		Tire Repair Cement/Sealer	Liquid	✓	
	Cleaning and Furniture Care Products	Carpet Cleaner	Liquid	✓	✓
		Spot Remover	Aerosol	✓	✓
		Spot Remover	Liquid	✓	✓
	Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings	Aerosol	✓	
	Apparel and Footwear Care Products	Shoe Polish	Aerosol	✓	✓

Life Cycle Stage	Categories	Product Subcategories	Form	Acute Inhalation Exposure	Acute Dermal Exposure
	Other Consumer Uses	Fabric Spray	Aerosol	✓	
		Film Cleaner	Aerosol	✓	
		Hoof Polish	Aerosol	✓	
		Pepper Spray	Aerosol	✓	
		Toner Aid	Aerosol	✓	

A range in acute inhalation and acute dermal exposures is provided in Table 2-70, summarized by the consumer category. Ranges provided are based on the presented user scenario descriptions (high-, moderate-, and low-intensity) and may not reflect overall minimum and maximum exposure levels from all iterations of the modeling scenario, which can be seen in the Supplemental Files [*Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures* and *Risk Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures. Docket: EPA-HQ-OPPT-2019-0500*].

Table 2-70. Summary of Consumer Exposure Levels by Category

Consumer Category	Acute Inhalation 24-hr TWA[1] (ppm)		Acute Dermal ADR[2] (mg/kg/d)
Solvents for Cleaning and Degreasing	User	4.55E-02 – 1.62E+02	1.19E-01 – 1.75E+02
	Bystander	8.47E-03 – 4.71E+01	
Lubricants and Greases	User	2.16E-02 – 1.47E+01	NA
	Bystander	4.21E-03 – 2.95	
Adhesives and Sealants	User	8.83E-03 – 3.22E+01	NA
	Bystander	1.30E-03 – 4.06	
Cleaning and Furniture Care Products	User	5.47E-01 – 5.26E+01	8.61E-02 – 4.77E+01
	Bystander	6.90E-02 – 1.15E+01	
Arts, Crafts, and Hobby Materials	User	2.90E-01 – 9.31	NA
	Bystander	5.66E-02 – 2.28	
Apparel and Footwear Care Products	User	5.96E-02 – 3.38	4.70E-02 – 3.08
	Bystander	1.16E-02 – 6.79E-01	
Other Consumer Uses	User	1.77E-02 – 6.42E+01	NA
	Bystander	7.79E-05 – 1.57E+01	

[1]The level of variation displayed in the ranges of consumer categories reflect multiple, specific consumer conditions of use / subcategories and do not reflect the degree of variation present within scenario-specific results. The displayed category ranges therefore reflect a much broader spread of exposure estimates.
[2]The range in acute dermal ADRs reflect all age groups modeled (children and adult).

2.3.2.7 Assumptions and Key Sources of Uncertainty for Consumer Exposures

EPA's approach recognizes the need to include uncertainty analysis. One important distinction for such an analysis is variability versus uncertainty – both aspects need to be addressed. Variability refers to the inherent heterogeneity or diversity of data in an assessment. It is a quantitative description of the range or spread of a set of values and is often expressed through statistical metrics, such as variance or standard deviation, that reflect the underlying variability of the data. Uncertainty refers to a lack of data or an incomplete understanding of the context of the risk evaluation decision.

Variability cannot be reduced, but it can be better characterized. Uncertainty can be reduced by collecting more or better data. Quantitative methods to address uncertainty include non-probabilistic approaches such as sensitivity analysis and probabilistic or stochastic methods. Uncertainty can also be addressed qualitatively, by including a discussion of factors such as data gaps and subjective decisions or instances where professional judgment was used.

Uncertainties associated with approaches and data used in the evaluation of consumer exposures are described below.

2.3.2.7.1 Modeling Approach Uncertainties

Deterministic vs. Stochastic

With deterministic approaches like the one applied in this evaluation of consumer exposure, the output of the model is fully determined by the choices of parameter values and initial conditions. Stochastic approaches feature inherent randomness, such that a given set of parameter values and initial conditions can lead to an ensemble of different model outputs. The overall approach to the CEM modeling is intended to capture a range of low- to high-intensity User exposure estimates by varying only a limited number of key parameters that represent the range of consumer product and use patterns for each scenario. As previously mentioned the parameters selected were chemical weight fraction, product mass, and duration of use. All other parameters remained constant between model runs. Since not all parameters were varied, there is uncertainty regarding the full range of possible exposure estimates. Although these estimates are thought to reflect the range in exposure estimates for the suite of possible exposures based on the three varied parameters, the scenarios presented are not considered bounding or "worst-case," as there are unvaried parameters that are also identified as sensitive inputs held constant at a central tendency value. These include the room of use volume, residential building volume, and air exchange rate. Because EPA's largely deterministic approach involves choices regarding highly influential factors such as mass of product used and weight fraction, it likely captures the range of potential exposure levels although it does not necessarily enable characterization of the full probabilistic distribution of all possible outcomes.

Aggregate Exposure

Background levels of TCE in indoor and outdoor air are not considered or aggregated in this assessment; therefore, there is a potential for underestimating consumer inhalation exposures, particularly for populations living near a facility emitting TCE or living in a home with other sources of TCE, such as TCE-containing products stored in the home. For example, the indoor air and personal breathing zone monitoring values presented in Appendix D.2 were not considered for aggregation with modeled, use-specific acute air concentrations. Similarly, inhalation exposures were evaluated on a product-specific basis and are based on use of a single product type within a day, not multiple products.

Acute Exposure

EPA assumes that a consumer product would be used only once per day. This is a reasonable assumption for most scenarios, but a Do-It-Yourself- (DIY-) type user could potentially use the same product multiple times in one day. Additionally, based on human health hazard considerations and typical use patterns, chronic exposures were not evaluated for TCE-containing consumer products. However, it is possible that there would be concern for chronic exposure effects for use frequencies greater than intermittent. For example, daily or DIY-type uses of consumer products could constitute a short-term chronic exposure scenario or repeated-acute exposure scenario that is not captured in this evaluation. Identified chronic non-cancer and cancer hazard endpoints (Section 3.2) are unlikely to present for these populations based on reasonably available information, however the possibility cannot be ruled out. For the vast majority of the consumer population which are only exposed through short-term, occasional use of TCE products, only acute exposure is applicable.

Dermal Exposure Approach

Dermal exposures are quantified and presented for scenarios that may involve dermal contact with impeded evaporation based on professional considerations of the formulation type and likely use pattern. However, there is uncertainty surrounding the assumption that such dermal contact with impeded evaporation would occur for those scenarios. For example, for aerosol formulations, it is possible that aerosol degreasing or cleaning products may be sprayed and left to drip or dry from the target surface. It is also possible users would follow spraying with wiping, which could lead to some duration of dermal contact with impeded evaporation.

There is related uncertainty surrounding the application of exposure durations for such scenarios. The exposure durations modeled are based on reported durations of product use and may not reflect reasonable durations of such dermal contact with impeded evaporation. In many cases, the exposure duration modeled could exceed a reasonable duration of such dermal contact with a wet rag, for example. Therefore, dermal exposure results based on the higher-end durations (i.e., those associated with the moderate- and high-intensity user scenarios) may overestimate dermal exposure. Another source of potential overestimation is the application of a single formulation density to scenarios covering a range of specific TCE-containing products with a range of formulation densities. For such scenarios, a single (highest) density was chosen to convert the mass used input obtained from the Westat (1987) survey from ounces of product to grams of product. For some scenarios, this may have driven up the mass used, though the degree of this impact is dependent on he broadness of the density range for that condition of use.

In the evaluation of consumer dermal exposure, P_DER2b utilizes a measured dermal permeability coefficient (K_p). EPA selected a K_p of 0.019 cm/hr from Poet (2000) obtained from a water patch test on human skin using TCE in aqueous solution. While it is within range of other, predicted K_p values – CEM predicts a K_p of 0.028 cm/hr and the NIOSH Skin Notation Profile for TCE calculates a K_p of 0.01197 cm/hr (Hudson and Dotson, 2017) – it is a key parameter and there is some uncertainty surrounding the impact of applying an aqueous K_p for the prediction of dermal flux for formulations of TCE-containing consumer products, some of which contain nearly 100% TCE. While neat TCE would be estimated to have a lower K_p based on relatively low water solubility (
Table 1-1) compared to its density, TCE is an irritant that would be expected to disrupt the stratum corneum and lead to greatly increased absorption over time.

Inhalation Modeling for Outdoor Scenarios

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 pepper spray, 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.

2.3.2.7.2 Data Uncertainties

Product Data

The products and articles assessed in this risk evaluation are largely based on EPA's 2017 Use and Market Profile for TCE, as well as EPA's Use Report and Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: TCE, which provide information on commercial and consumer products available in the US marketplace at that time (U.S. EPA, 2017c, h). While it is possible that some products may have changed since 2017, EPA believes that the timeframe is recent enough to represent the ongoing and reasonably foreseen consumer uses. Additional sources of product information were evaluated, including the NIH Household Product Survey and EPA's Chemical and Products Database (CPDat), as well as available product labels and safety data sheets (SDSs). However, it is possible that the entire universe of products may not have been identified, or that certain changes in the universe of products may not have been captured, due to market changes or research limitations.

Use Patterns

A comprehensive survey of consumer use patterns in the Westat Survey, was used to parameterize critical consumer modeling inputs, based on applicable product and use categories. This large survey of over 4,920 completed questionnaires, obtained through a randomized sampling technique, is highly relevant because the primary purpose was to provide statistics on the use of solvent-containing consumer products for the calculation of exposure estimates. The survey focused on 32 different common household product categories, generally associated with cleaning, painting, lubricating, and automotive care. Although there is uncertainty due to the age of the use pattern data, as specific products in the household product categories have likely changed over time, EPA believes that the use pattern data presented in the Westat survey reflect reasonable estimates for current use patterns of similar product types.

A crosswalk was completed to select the most appropriate Westat survey category for each consumer conditions of use in the current risk evaluation. Although detailed product descriptions were not provided in the Westat survey, a list of product brands and formulation type in each category was useful in pairing the Westat product categories to the scenarios being assessed. In most cases, the product categories in the Westat survey aligned reasonably well with the products being assessed. Where Westat survey product categories did not align well with consumer conditions of use, professional judgment was used to select the most appropriate Westat category. This involved considering the reasonableness of the duration and mass used, as well as comparing the primary formulation type. For a limited number of scenarios, technical fact sheets or labels with information on product use amounts were available, and this information was used in the assessment as needed.

Westat's overall respondent pool of the survey was large, but the number of users in each product category was varied, with some product categories having a much smaller pool of respondents than others. Product categories such as spot removers, cleaning fluids, glues and adhesives, lubricants, paints,

paint strippers, fabric water repellents, wood stains, tire cleaners, engine degreasers, carburetor cleaners, and specialized electronic cleaners had sample sizes ranging from roughly 500 to 2,000 users; whereas, categories such as shoe polish, adhesive removers, rust removers, primers, outdoor water repellents, gasket removers and brake cleaners had sample sizes of fewer than 500 users.

Emission Rate

The higher-tier Multi-Chamber Concentration and Exposure Model (MCCEM) was considered by EPA for use in estimating inhalation exposures from consumer conditions of use; however, key data (i.e., chamber emission data) were not reasonably available. Therefore, the model used (CEM 2.1) estimates emission rate based on chemical properties and emission profiles matching a spray or liquid application.

2.3.2.8 Confidence in Consumer Exposure Scenarios

The considerations and confidence ratings for the acute inhalation consumer exposure scenarios are displayed in Table 2-71 with detailed explanations of rationale for the parameters in the footnotes. Overall, there is moderate to high or high confidence in the consumer inhalation exposure modeling approach and results. This is based on strength of the model employed, as well as the quality and relevance of the default and user-selected/varied modeling inputs. CEM 2.1 is peer reviewed, publicly available, and was designed to estimate inhalation and dermal exposures from household uses of products and articles. CEM 2.1 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 used, use duration). These defaults are sourced from EPA's exposure factors handbook (U.S. EPA, 2011c). The one default value with a high-end input is the overspray fraction, which is used in the aerosol or spray scenarios. It assumes a certain percentage is immediately available for inhalation. However, due to TCE's physical chemical properties, this is a not a sensitive parameter. In the 2014 TCE Risk Assessment, this parameter was varied from 1% to 25% and resulted in almost no difference in the modeled peak air concentration (U.S. EPA, 2014b). The default emission rate from a thin film is estimated within the model based on TCE's molecular weight and vapor pressure, as described in the Chinn equation[14] and is deemed appropriate given the lack of consumer product chamber emission data. The confidence in the user-selected varied inputs (i.e., mass used, use duration, and weight fraction) are moderate to high, depending on the condition of use; the sources of these data include the Westat Survey (U.S. EPA, 1987) and company-generated safety data sheets (SDSs). The representativeness of the consumer use patterns (duration of use, amount used, room of use, etc.) described in the Westat Survey (U.S. EPA, 1987) is believed to remain strong when compared to present day consumer use patterns even though some aspects of the use may have changed. However, ease of access to products on-line or in big box stores (like home improvement stores), readily accessible how-to videos, and a consumer movement toward more do-it-yourself projects with products containing the chemical of concern could impact the representativeness of the consumer use patterns described within the Westat Survey and may lead to an underestimate of overall consumer exposure. There is a medium uncertainty associated with the representativeness of the consumer use patterns described within the Westat Survey and present day consumer use patterns. In some cases, professional judgment was used in selection of room of use, which sets the volume for modeling zone 1.

The considerations and confidence ratings for the acute dermal consumer exposure scenarios are displayed in Table 2-72 with detailed explanations of rationale for the parameters in the footnotes.

[14] The value of k is determined from an empirical relationship, developed by (Chinn, 1981), between the time required for 90% of a pure chemical film to evaporate ($EvapTime$) and the chemical's molecular weight (MW) and vapor pressure (VP): EvapTime = 145 / (MW x VP) 0.9546, k = ln(10) / (EvapTime x 60), where k = first-order rate constant for emission decline (min-1), MW = molecular weight, VP = vapor pressure.

Overall, there is a low to moderate confidence in the consumer dermal exposure modeling approach and results. The same model is employed to estimate dermal exposures; however, there is greater uncertainty related to the potential for dermal contact with impeded evaporation (i.e., dermal exposure scenarios wherein volatilization from the skin surface is inhibited); this contributes to the lower overall confidence in the dermal results. The dermal permeability approach was selected for modeling instead of the fraction absorbed method. Based on rationale provided in the problem formulation, EPA determined that only dermal exposures with impeded evaporation would be evaluated for consumer conditions of use. This is based on the expectation that, if not inhibited from volatilizing, inhalation exposure would account for the preponderance of exposure from consumer uses. An example of dermal contact with impeded evaporation for consumer applications would be having a TCE-soaked rag pressed firmly against a user's fingers or hands for a period of time. Therefore, the permeability approach was deemed more reflective of this type of dermal exposure scenario, as it does not account for losses due to volatilization and assumes a constant flux of TCE for the duration of the use event. In modeling these scenarios, the same use durations sourced from the Westat survey (U.S. EPA, 1987) are applied; however, in doing so, the model assumes that there are no losses throughout the entire use duration. It is unlikely that dermal contact would involve impeded evaporation for the entire use duration, particularly for central-tendency and high-end use durations. It is more likely that such contact would be intermittent throughout longer use durations and not constant. This leads to an overall low confidence in that input; however, there would be greater confidence in the results obtained from the low-end use duration inputs for any weight fraction modeled.

An additional point of confidence in the consumer modeling approach related to capturing variation and estimating results for a range of exposure levels. Although a probabilistic assessment was not employed, EPA did use up to three inputs for three key modeling parameters: mass used, use duration, and weight fraction. The first two parameters are based on the Westat survey data, which presented a distribution of responses. For these parameters, a low-end (10^{th} percentile), central tendency (50^{th} percentile), and high-end (95^{th} percentile) was used in modeling. Weight fraction inputs were based on product SDSs, so the full range of reported weight fractions was reflected in the modeling inputs using either minimum and maximum weight fractions or using minimum and maximum weight fractions along with a mid-point weight fraction. For subcategories with only one product, only one weight fraction was used in the modeling. Otherwise, these varied parameters were varied in all possible combinations, resulting in up to 27 iterations for a given modeling scenario.

Consumer exposure monitoring studies associated with conditions of use are not reasonably available for direct comparison with modeled results. Indoor air monitoring data are available but are not associated with specific conditions of use or TCE-containing consumer products and are therefore only relevant for considerations of background levels of TCE in homes.

While there were certain scenarios that have moderate confidence ratings rather than high confidence for user-selected varied inputs, there are not reasonably available alternative inputs that would serve to increase confidence in the modeling estimates. For example, in modeling film cleaner, the alternative to applying mass used and use duration from the rust remover Westat survey scenario is professional judgment, which is unlikely to decrease uncertainty.

Table 2-71. Confidence Ratings for Acute Inhalation Consumer Exposure Modeling Scenarios

Consumer Condition of User			Confidence in Model Used[1]	Confidence in Model Default Values[2]	Confidence in User-Selected Varied Inputs[3]				Overall Confidence
Category	Subcategory	Form			Mass Used[4]	Use Duration[5]	Weight Fraction[6]	Room of Use[7]	
Solvents for Cleaning and Degreasing	Brake & Parts Cleaner	Aerosol	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Electronic Degreaser/ Cleaner	Aerosol	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Electronic Degreaser/ Cleaner	Liquid	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Spray Degreaser/ Cleaner	Aerosol	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Liquid Degreaser/ Cleaner	Liquid	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Gun Scrubber	Aerosol	High	High	High	Moderate	High	Moderate	Moderate to High
Solvents for Cleaning and Degreasing	Gun Scrubber	Liquid	High	High	High	Moderate	High	Moderate	Moderate to High
Solvents for Cleaning and Degreasing	Mold Release	Aerosol	High	High	Moderate	High	High	High	Moderate to High
Solvents for Cleaning and Degreasing	Tire Cleaner	Aerosol	High	High	High	High	High	High	High
Solvents for Cleaning and Degreasing	Tire Cleaner	Liquid	High	High	High	High	High	High	High
Lubricants and Greases	Tap & Die Fluid	Aerosol	High	High	High	High	High	High	High
Lubricants and Greases	Penetrating Lubricant	Aerosol	High	High	High	High	High	High	High
Adhesives and Sealants	Solvent-based Adhesive & Sealant	Liquid	High	High	High	High	High	High	High
Adhesives and Sealants	Mirror-edge Sealant	Aerosol	High	High	Moderate	Moderate	High	High	High

Consumer Condition of User			Confidence in Model Used[1]	Confidence in Model Default Values[2]	Confidence in User-Selected Varied Inputs[3]				Overall Confidence
Category	Subcategory	Form			Mass Used[4]	Use Duration[5]	Weight Fraction[6]	Room of Use[7]	
Adhesives and Sealants	Tire Repair Cement/ Sealer	Liquid	High	High	High	High	High	High	High
Cleaning and Furniture Care Products	Carpet Cleaner	Liquid	High	High	Moderate	Moderate	High	Moderate	Moderate to High
Cleaning and Furniture Care Products	Spot Remover	Aerosol	High	High	High	High	High	High	High
Cleaning and Furniture Care Products	Spot Remover	Liquid	High	High	High	High	High	High	High
Arts, Crafts, and Hobby Materials	Fixatives & Finishing Spray Coatings	Aerosol	High	High	Moderate	Moderate	High	Moderate	Moderate to High
Apparel and Footwear Care Products	Shoe Polish	Aerosol	High	High	High	High	High	High	High
Other Consumer Uses	Fabric Spray	Aerosol	High	High	High	High	High	High	High
Other Consumer Uses	Film Cleaner	Aerosol	High	High	Moderate	Moderate	High	Moderate	Moderate to High
Other Consumer Uses	Hoof Polish	Aerosol	High	NA	Moderate	Moderate	High	High	Moderate to High
Other Consumer Uses	Pepper Spray	Aerosol	High	NA	High	High	High	Moderate	Moderate to High
Other Consumer Uses	Toner Aid	Aerosol	High	High	Moderate	Moderate	High	Moderate	Moderate to High

[1]Confidence in Model Used considers whether model has been peer reviewed, as well as whether it is being applied in a manner appropriate to its design and objective. The model used (CEM 2.1) has been peer reviewed, is publicly available, and has been applied in a manner intended – to exposures associated with uses of household products and/or articles.
[2]Confidence in Model Default Values considers default value data source(s) such as building and room volumes, interzonal ventilation rates, and air exchange rates. These default values are all central tendency values (i.e., mean or median values) sourced from EPA's Exposure Factors Handbook (U.S. EPA, 2011c). The one default value with a high-end input is the overspray fraction, which is used in the aerosol or spray scenarios. It assumes a certain percentage is immediately available for inhalation. However, due to TCE's physical chemical properties, this is a not a sensitive parameter. In the 2014 TSCA Work Plan Chemical Risk Assessment for TCE (U.S. EPA, 2014b), this parameter was varied from 1% to 25% and resulted in almost no difference in the modeled peak air concentration.
[3]Confidence in User-Selected Varied Inputs considers the quality of their data sources, as well as relevance of the inputs for the selected consumer condition of use.

Consumer Condition of User			Confidence in Model Used[1]	Confidence in Model Default Values[2]	Confidence in User-Selected Varied Inputs[3]				Overall Confidence
Category	Subcategory	Form			Mass Used[4]	Use Duration[5]	Weight Fraction[6]	Room of Use[7]	

[4]Mass Used is primarily sourced from the Westat (1987) survey, which received a high-quality rating during data evaluation and has been applied in previous agency assessments. Two conditions of use had product information that was used instead of Westat (gun scrubber and pepper spray).
[5]Use Duration is primarily sourced from the Westat (1987) survey, which received a high-quality rating during data evaluation and has been applied in previous agency assessments. One condition of use had product information that was used instead of Westat (pepper spray). Relevance of these inputs from the Westat survey to the specific consumer condition of use they were applied to is considered in the reported confidence ratings.
[6]Weight fraction of TCE in products is sourced from product Safety Data Sheets (SDSs), which were not reviewed as part of systematic review but were taken as authoritative sources on a product's ingredients.
[7]Room of use (zone 1 in modeling) is informed by responses in the Westat (1987) survey, which received a high-quality rating during data evaluation, although professional judgment is also applied for some scenarios. The reasonableness of these judgements is considered in the reported confidence ratings.

Table 2-72. Confidence Ratings for Acute Dermal Consumer Exposure Modeling Scenarios

Consumer Condition of User			Confidence in Model Used[1]	Confidence in Model Default Values[2]	Confidence in Assumption of Dermal Contact with Impeded Evaporation[3]	Confidence in User-Selected Varied Inputs[4]			Overall Confidence
Category	Subcategory	Form				K_p[5]	Use Duration[6]	Weight Fraction[7]	
Solvents for Cleaning and Degreasing	Brake & Parts Cleaner	Aerosol	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Electronic Degreaser/ Cleaner	Liquid	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Spray Degreaser/ Cleaner	Aerosol	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Liquid Degreaser/ Cleaner	Liquid	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Gun Scrubber	Aerosol	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Gun Scrubber	Liquid	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Tire Cleaner	Aerosol	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate
Solvents for Cleaning and Degreasing	Tire Cleaner	Liquid	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Cleaning and Furniture Care Products	Carpet Cleaner	Liquid	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Cleaning and Furniture Care Products	Spot Remover	Aerosol	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate

Consumer Condition of User			Confidence in Model Used[1]	Confidence in Model Default Values[2]	Confidence in Assumption of Dermal Contact with Impeded Evaporation[3]	Confidence in User-Selected Varied Inputs[4]			Overall Confidence
Category	Subcategory	Form				K_p[5]	Use Duration[6]	Weight Fraction[7]	
Cleaning and Furniture Care Products	Spot Remover	Liquid	Low to Moderate	High	Moderate	Moderate	Low	High	Low to Moderate
Apparel and Footwear Care Products	Shoe Polish	Aerosol	Low to Moderate	High	Low	Moderate	Low	High	Low to Moderate

[1]Confidence in Model Used considers whether model has been peer reviewed, as well as whether it is being applied in a manner appropriate to its design and objective. The model used (CEM 2.1) has been peer reviewed, is publicly available, and has been applied in a manner intended – to estimate exposures associated with uses of household products and/or articles. For the purposes of dermal exposure, this confidence rating also considers the appropriateness of the dermal permeability model within CEM 2.1 for estimating dermal exposures with impeded evaporation and known sources of uncertainty.
[2]Confidence in Model Default Values considers default value data source(s) such as surface area to body weight ratios for the dermal contact area. These default values are all central tendency values (i.e., mean or median values) sourced from EPA's Exposure Factors Handbook (U.S. EPA, 2011c).
[3]Confidence in Assumption of Dermal Contact with Impeded Evaporation characterizes the uncertainty surrounding whether or not occluded contact is even possible or likely. Certain conditions of use have greater uncertainty over whether or not any occluded contact is expected, i.e., the spray scenarios. The liquid formulations are likely to result in some dermal contact with a rag; however, there remains uncertainty related to the degree to which such contact would be occluded.
[4]Confidence in User-Selected Varied Inputs considers the quality of their data sources, as well as relevance of the inputs for the selected consumer condition of use.
[5]The dermal permeability coefficient (K_p) used (0.019 cm/hr) from Poet (2000) came from a water patch test on human skin using TCE in an aqueous solution. While it is within range of other, predicted K_p values (CEM 2.1 predicts 0.028 cm/hr and NIOSH calculates 0.01197 cm/hr), it is a key parameter and there is uncertainty surrounding the impact of applying an aqueous K_p for prediction of dermal flux for formulations of TCE-containing consumer products with nearly 100% TCE.
[6]Use Duration is primarily sourced from the Westat (1987) survey, which received a high-quality rating during data evaluation and has been applied in previous agency assessments. The dermal modeling receives a "low" confidence for this criterion due to the uncertainty associated with the period of time during which a dermal exposure duration is likely to be occluded, not due to relevance or data source.
[7]Weight fraction of TCE in products is sourced from product Safety Data Sheets (SDSs) and were taken as authoritative sources on a product's ingredients.

2.3.3 Potentially Exposed or Susceptible Subpopulations

TSCA requires that a risk evaluation "determine whether at chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." 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 TCE. Exposures of TCE would be expected to be higher amongst groups living near industrial facilities, groups with TCE containing products in their homes, workers who use TCE 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 TCE 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 TCE). Exposure estimates were developed for users (males and female workers of reproductive age) exposed to TCE as well as non-users or workers exposed to TCE 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. TCE has been identified as being used in products available to consumers. Sections 2.3.2.1 and 2.3.2.2 provide an overview of exposure pathways considered for the consumer assessment. Furthermore, EPA identified consumers and bystanders associated with use of TCE-containing consumer products as a potentially exposed and susceptible subpopulation due to greater exposure as described in Section 2.3.2.3. 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 2.3.2.6 (Table 2-32 through Table 2-68).

In developing dermal exposure scenarios, EPA quantified age and gender-specific differences. For TCE, 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, 2011c) to inform body weights, intake rates, and body surface areas for children and adults. Distinct dermal exposure estimates are provided for for adults (including women of reproductive age) and children (Section 2.3.2.6.1).

For occupational exposures, EPA assessed exposures to workers and ONUs from all TCE conditions of use. Table 2-73 presents the percentage of employed workers and ONUs whom may experience either greater exposure or biological susceptibility within select industry sectors relevant to TCE 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 2-73, 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 2-74 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 TCE.

Table 2-73. 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 (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%

Source: (U.S. BLS, 2017). While statistics on pregnant women are not reasonably available, CPS 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. Percentage calculated using CPS Table 14, "Employed persons in nonagricultural industries by age, sex, race, and Hispanic or Latino ethnicity."

Table 2-74. 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. TCE 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 TCE, 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 TCE 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 TCE or products and formulations containing TCE. 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 TCE.
- Other services – This sector comprises establishments engaged in providing services not specifically provided for elsewhere in the classification system. For TCE, this sector covers the vast majority of commercial repair and maintenance facilities that are likely to use TCE for Aerosol Applications (spray degreasing). The sector also covers the use of TCE in spot cleaning.

3 HAZARDS

3.1 Environmental Hazards

3.1.1 Approach and Methodology

During scoping and problem formulation (U.S. EPA, 2018d), EPA reviewed potential environmental health hazards associated with TCE. EPA identified the following sources of environmental hazard data: European Chemicals Agency (ECHA) Database (ECHA, 2017), European Union (EU) environmental risk assessment on TCE (ECHA, 2004) EPA Chemical Test Rule Data (U.S. EPA, 2017a) Environment and Climate Change Canada (ECCC) Risk Assessment for Trichloroethylene (Environment Canada and Health Canada, 1993) and Ecological Hazard Literature Search Results in Trichloroethylene (CASRN 79-01-6) Bibliography: Supplemental File for the TSCA Scope Document (U.S. EPA, 2017i).

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). Studies were rated high, medium, or low for quality. The data quality evaluation results are outlined in the [*Data Quality Evaluation of Environmental Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500*] and indicate that most of the acceptable studies for TCE were rated high and moderate for quality. With the reasonably available data, EPA used studies rated high or medium for quantitative analysis during data integration, and used studies rated low qualitatively to characterize the environmental hazards of trichloroethylene. Any study assigned an overall quality level of unacceptable was not used for data integration. Mechanistsic studies were used qualitatively, because toxicity values measuring a population-level effect (e.g. mortality, development, growth) were available to use quantitatively.

3.1.2 Hazard Identification

Toxicity to Aquatic Organisms

EPA identified 25 acceptable studies that contained aquatic toxicity data, including data for fish, amphibians, aquatic invertebrates, and algae. Aquatic toxicity studies considered in this assessment are summarized in the text below, and the data EPA used quantitatively are displayed in Table 3-1. As stated in Section 2.1, TCE is not expected to accumulate in aquatic organisms due to low measured BCFs and an estimated BAF.

Fish Toxicity

Acute fish data for TCE were identified in six acceptable studies representing four different species, including fresh and saltwater species (fathead minnows [*Pimephales promelas*], American flagfish [*Jordanella floridae*], bluegill [*Lepomis macrochirus*], and sheepshead minnow [*Cyprinodon variegatus*]). In these studies, all used quantitatively in this assessment, the lethal concentrations at which 50% of test organisms die (LC_{50}s) ranged from 28.28 mg/L to 66.8 mg/L (Geiger et al., 1985); (Broderius et al., 2005; Smith et al., 1991; Ward et al., 1986; Buccafusco et al., 1981; Alexander et al., 1978). Ward et al. (1986) tested a saltwater species, sheepshead minnow, and derived an LC_{50} of 52 mg/L. Because this value is within the of the range of values for freshwater species, and because baseline narcosis is the expected mode of action for TCE in both freshwater and saltwater fish (Alexander et al., 1978); (Ward et al., 1986); (Broderius et al., 2005), freshwater and saltwater LC_{50} values were assessed together during data integration. EPA calculated a geometric mean of 42 mg/L using LC_{50}s from high and medium quality studies. Acute fish data for TCE also included a 96-hour EC_{50} (the concentration at which 50% of test organisms exhibit an effect) of 21.9 mg/L for loss of

equilibrium in a freshwater species, fathead minnows (Alexander et al., 1978). This study was rated high for quality.

Subchronic fish data were also identified in two acceptable studies representing two species. Smith et al. (1991) established a 10-day NOEC of 5.758 mg/L and a LOEC of 21.233 mg/L resulting in a chronic value (ChV) of 11 mg/L for fry survival in American flagfish (*Jordanella floridae*). Schell (1987) established a 10-day LC_{50} of 82 mg/L in Japanese medaka (*Oryzias latipes*) embryos. The author found that lethality occurred at every stage of development for embryos. Schell also observed lesion development in the embryos after exposure in a dose-dependent pattern, with higher test concentrations resulting in earlier formation of lesions. Both abovementioned sub-chronic studies received a high rating for quality during data evaluation, and EPA used the data quanitatively.

Chronic fish data for TCE were identified in two acceptable studies representing two freshwater species, American flagfish (*Jordanella floridae*) and fathead minnows (*Pimephales promelas*). In addition to the subchronic value mentioned above, Smith et al. (1991) established a 28-day NOEC of 10.568 mg/L and a LOEC of 20.915 mg/L for fry survival in American flagfish. This allowed the authors to establish a 28-day ChV of 14.85 for fry survival. Broderius et al. (2005) established an EC_{50} for growth of 11.8 mg/L and an EC_{20} for growth of 7.88 mg/L in a 32-day fathead minnow study. Both studies were rated high for quality during data evaluation. EPA used the chronic data in these studies quantitiatively.

Broderius et al. (2005) reported baseline narcosis as TCE's expected mode of action in fish. This is corroborated by other studies, including Ward, et al. (1986), which observed signs of narcosis in sheepshead minnows, a saltwater species, with observations of fish spinning at 357 mg/L. EPA used this information qualitatilvey in this assessment. Alexander et al. (1978) reported signs of narcosis in fathead minnows, a freshwater species, with a 96-hour EC_{10} of 13.7 mg/L, EC_{50} of 21.9 mg/L, and EC_{90} of 34.9 mg/L. The effect reported was loss of equilibrium. EPA used the 96-hour EC_{50} from Alexander et al. (1978) quantitatively in this assessment.

Two mechanistic studies were also available for fish. Hayashi et al. (1998) examined genotoxicity in rose bitterling (*Rhodeus ocellatus*) embryos using a new assay developed by the authors. The authors found an increase in structural chromosomal aberrations and micronuclei in cells from embryos, establishing a NOEC of 300 mg/L and a LOEC of 3,000 mg/L. The authors noted the low sensitivity of the assay and suggested using more embryos in the future. This study was rated medium for quality. Another *in vitro* study, rated low for quality, derived an EC_{50} of 11.6 mg/L for the inhibition of total protein content in a fathead minnow cell line (Dierickx, 1993). Because this cellular effect is not directly tied to a population effect, and because of the low-quality rating, this study was not used with the other acute data to calculate a geometric mean of EC_{50}s during data integration; however, the results contribute to the qualitative description of mechanistic effects of TCE exposure in fish.

Amphibian Toxicity

For amphibians, acute data were available from three acceptable studies, representing one species, African clawed frogs (*Xenopus laevis*). All three studies were rated either high or medium for quality during data evaluation. The studies included 96-hour LC_{50} values ranging from 412.0 mg/L to 490.0 mg/L (McDaniel et al., 2004; Fort et al., 2001; Fort et al., 1993; Fort et al., 1991). EPA used these studies quantitatively, and during data integration, a geometric mean of all LC_{50}s was calculated at 438 mg/L.

Sub-chronic data were also available for amphibians, from four acceptable studies representing five different species (green frog [*Rana clamitans*], wood frog [*Rana sylvatica*], African clawed frogs

[*Xenopus laevis*], American toad [*Bufo americanus*], and spotted salamander [*Ambystoma maculatum*]). These studies reported 96-hr EC_{50} values for developmental effects ranging from 22 mg/L to > 85 mg/L (McDaniel et al., 2004; Fort et al., 2001; Fort et al., 1993; Fort et al., 1991). EPA used these data quantitatively, and during data integration, a geometric mean of all definitive EC_{50}s for developmental effects was calculated at 34 mg/L. These developmental effects are irreversible and would result in effects that last throughout the animals' lifetime. Developmental effects described included gut miscoiling and microphthalmia, muscular kinking, incomplete development of the mouth, and severe hypognathia in African clawed frogs, and edema and dorsal flexure of the tail and notochord in tadpoles of green frogs, wood frogs, American toads, and spotted salamanders (McDaniel et al., 2004; Fort et al., 1993; Fort et al., 1991). As stated previously, McDaniel et al. (2004) reported signs of narcosis in green and wood frog tadpoles.

Limited chronic data were also available for amphibians. McDaniel et al., (2004) included a chronic toxicity test for amphibians on American toad tadpoles. However, chronic toxicity values for deformities were not established, because more than 25% of control animals exhibited deformities. Mortality, however, was below 25% in controls, and authors saw no significant difference in mortality between test concentrations (4 mg/L and 1 mg/L) and controls. This suggests that survival rates for American toad tadpoles would not be affected by 4 mg/L of TCE. It should be noted that acute exposure data show American toads are less sensitive to TCE than other amphibian species, so they may also be less sensitive to chronic exposures. EPA used this information qualitatively.

McDaniel et al. (2004) reported signs of narcosis in green and wood frog tadpoles.

Aquatic Invertebrate Toxicity

For aquatic invertebrates, acute data were found in seven acceptable studies representing five different species, including fresh and saltwater species. Five of these studies included LC_{50} or EC_{50} values rated high or medium for quality; these values ranged from 7.75 mg/L to 43.14 mg/L for *Daphnia magna*, *Ceriodaphnia dubia*, and *Mysidopsis bahia* (Dobaradaran et al., 2012; Niederlehner et al., 1998; Abernethy et al., 1986; Ward et al., 1986; LeBlanc, 1980). The only saltwater species tested, *Mysidopsis bahia*, had an LC_{50} of 14 mg/L, which is within the of the range of values for freshwater species. EPA used these data quantitatively. Additionally, Ward et al. (1986) and Niederlehner et al. (1998) reported baseline narcosis as the mode of action for TCE in freshwater and saltwater invertebrates. Therefore, freshwater and saltwater values were integrated together. The geometric mean of the EC_{50} and LC_{50}s from high and medium quality studies is 16 mg/L. EPA used these data quantitatively. Another study, Sánchez-Fortún et al. (1997), rated low for quality, established LC_{50}s in *Artemia salina* larvae at three different ages; however, this study was not used quantitatively during data integration, given that medium and high-quality studies were available for invertebrates.

One subchronic study found an LC_{50} of 1.7 mg/L in planarian (*Dugesia japonica*) over 7 days (Yoshioka et al., 1986). This study was rated low for quality. Because other higher quality studies were available for aquatic invertebrates, this study was not used quantitatively during data integration.

Chronic data for aquatic invertebrates were identified in two acceptable studies, both rated high for quality. One study established toxicity values for reproduction, an effect that is relevant at the population level. Niederlehner et al. (1998) established a NOEC of 7.1 mg/L and a LOEC of 12 mg/L for reproduction in *Ceriodaphnia dubia*, resulting in a ChV of 9.2 mg/L. Niederlehner et al. (1998) established a 7-day reproductive inhibitory concentration (IC_{50}) of 11 mg/L, the concentration at which the mean number of young decreased by 50%. EPA used these data quantitatively.

Two studies reported baseline narcosis as the mode of action for TCE in invertebrates. Ward et al. (1986) observed mild intoxication in *Mysidopsis bahia*, a saltwater species, and Niederlehner et al. (1998) observed behavioral changes, including narcosis and abnormal movement in *Ceriodaphnia dubia*, a freshwater species. EPA used this information qualitatively.

Two studies provided mechanistic data for invertebrates. Vidal et al. (2001), rated high for quality, examined mechanistic effects of an acute exposure to a freshwater clam species, *Corbicula fluminea*. A one-time exposure over five days resulted a significant change in protein activity related to phase I metabolism. Results indicated a NOEC of 1.2 mg/L and a LOEC of 3.6 mg/L for significantly increasing cytochrome P-450 levels, and a NOEC of 3.6 mg/L and LOEC of 14 mg/L for significantly decreasing NADPH cytochrome C reductase activity (Vidal et al., 2001). Houde et al. (2015), also rated high for quality, examined the effects of TCE on *Daphnia magna* at the cellular and life-stage levels. The authors found a significant increase in chitinase production over 10 days, with a NOEC of 0.001 mg/L and a LOEC of 0.01 mg/L. Chitinase is an enzyme involved in molting and therefore development in *Daphnia magna*. While the study did not find a significant change in the total number of molts for the concentrations tested, the results were very close to significant with a $p = 0.051$ (assuming significance at $p \leq 0.05$), suggesting more tests are necessary to determine the impact of increased chitinase at the life-stage level. Because this mechanistic data is not directly linked to a population-level response, this data was used qualitatively rather than quantitiatively.

Aquatic Plant Toxicity

For aquatic plants hazard studies, algae are the common test species. Algae are cellular organisms which will cycle through several generations in hours to days; therefore the data for algae was assessed together regardless of duration rather than being categorized as acute or chronic.

There were six acceptable studies reported data on 11 species of algae, including fresh and saltwater species, and cyanobacteria and eukaryotes. There was a wide range of toxicity values reported in the literature for algae exposed to TCE. EC_{50}s measuring growth represent nine species and range from 26.24 mg/L to 820 mg/L (Lukavsky et al., 2011; Labra et al., 2010; Tsai and Chen, 2007; Ando et al., 2003; Brack and Rottler, 1994; Ward et al., 1986). Ward et al. (1986) reported results on the only saltwater species found in the acceptable studies, *Skeletonema costatum*, with an EC_{50} of 95 mg/L. This value is within the of the range of values for freshwater species, so saltwater and freshwater species were integrated together. EPA derived a geometric mean of 242 mg/L from the high and medium quality EC_{50}s. A 72-hour EC_{10} of 12.3 mg/L was also established by Brack and Rottler (1994) measuring biomass (a measure of growth) in *Chlamydomonas reinbardtii*, a freshwater eukaryotic green algae. Additionally, several NOECs and LOECs were established. Labra et al. (2010) found a 72-hour NOEC of 0.02 mg/L and a LOEC of 0.05 mg/L for cell count (a measure of growth) in *Raphidocelis subcapitata*. This study also assessed the integrity of algal cell membranes and found a dose-dependent increase in membrane damage starting at 0.05 mg/L. EPA used the abovementioned algae data quantitatively.

Ando et al. (2003) measured relative absorbance of chlorophyll *a* (an indirect measure of algal growth) in three species of algae, *Selenastrum capricornutum*, *Chlorella vulgaris*, and *Volvulina steinii*. They found no significant change in the relative absorbance of chlorophyll *a* for *S. capricornutum* or *C. vulgaris* during the 10-day test; however, they established a 10-day LOEC of 0.003 mg/L for *V. steinii*, a flagellar algae. The authors attributed the variation in algal species sensitivity to methylene chloride to *V. steinii*'s high metabolism. For several reasons explained in Section 3.1.4 Weight of the Scientific Evidence, these data were considered less biologically relevant than values from other studies and were not used quantitatively during data integration.

192
193 **Table 3-1 Ecological Hazard Data used Quantitatively to Characterize TCE Hazard for Aquatic**
194 **Organisms**

Duration	Test organism	Endpoint	Hazard value (mg/L)[1]	Geometric Mean[2] (mg/L)	Effect Endpoint	Citation (Study Quality)
Acute[3]	Fish	LC_{50} (freshwater)	28.28 – 66.8	42	Mortality	(Geiger et al., 1985) (high); (Alexander et al., 1978) (high); (Smith et al., 1991) (high); (Broderius et al., 2005) (high); (Buccafusco et al., 1981) (medium)
		LC_{50} (saltwater)	52			(Ward et al., 1986) (medium)
		EC_{50} (freshwater)	21.9		Immobilization	(Alexander et al., 1978) (high)
	Amphibian	LC_{50}	412.0 – 490.0	436	Mortality	(Fort et al., 2001) (medium); (Fort et al., 1991) (medium); (Fort et al., 1993) (high)
	Aquatic Invertebrates	EC_{50}/LC_{50} (freshwater)	7.8 – 33.85	**16**	Mortality and Immobilization	(LeBlanc, 1980) (high); (Niederlehner et al., 1998) (high); (Abernethy et al., 1986) (medium); (Dobaradaran et al., 2012) (medium)
		LC_{50} (saltwater)	14			(Ward et al., 1986) (medium)
Subchronic /Chronic[3]	Fish	EC_{20}	**7.88**		Growth	(Broderius et al., 2005) (high)
		EC_{50}	11.8		Growth	
		NOEC LOEC ChV	10.568 20.915 14.87		Fry Survival	(Smith et al., 1991) (high)
		NOEC LOEC ChV (subchronic)	5.758 21.233 11		Fry Survival	
		LC_{50} (subchronic)	82		Mortality	(Schell, 1987) (high)
	Amphibians	NOEC	4		Tadpole Survival	(McDaniel et al., 2004) (medium)
		EC_{50} (subchronic)	22 – >85	34	Deformities	(Fort et al., 2001) (medium); (Fort et al., 1991) (medium); (Fort et al., 1993) (high); (McDaniel et al., 2004) (high and medium)
	Aquatic invertebrates	NOEC LOEC ChV	7.1 12 9.2		Reproduction	(Niederlehner et al., 1998) (high)
		IC_{50}	11			
Algae[4]		EC_{50} (freshwater)	26.24 – 820	242	Growth	(Brack and Rottler, 1994) (high); (Tsai and Chen, 2007) (high); (Labra et al., 2010) (medium); (Ando et al., 2003) (medium); (Lukavsky et al., 2011) (medium)

	EC50 (saltwater)	95			(Ward et al., 1986) (medium)
	EC_{10}	12.3		Growth	(Brack and Rottler, 1994) (high)
	NOEC LOEC ChV	0.02 0.05 **0.03**		Growth	(Labra et al., 2010) (medium)

[1]Values in the table are presented in the number of significant figures reported by the study authors.
[2] Geometric mean of definitive values only (i.e., > 85 mg/L was not used in the calculation).
[3] Acute and chronic hazard data include fish, invertebrates, or amphibian data
[4] Because algae can cycle through several generations in hours to days, the data for algae was assessed together regardless of duration (i.e., 48-hrs to 96-hrs).
Note: Values in **bold** were used to derive Concentrations of Concern (COC) as described in Section 3.1.5 of this document. All values are listed individually with study quality in [*Data Quality Evaluation of Environmental Hazard Studies* and *Data Extraction for Environmental Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500*].

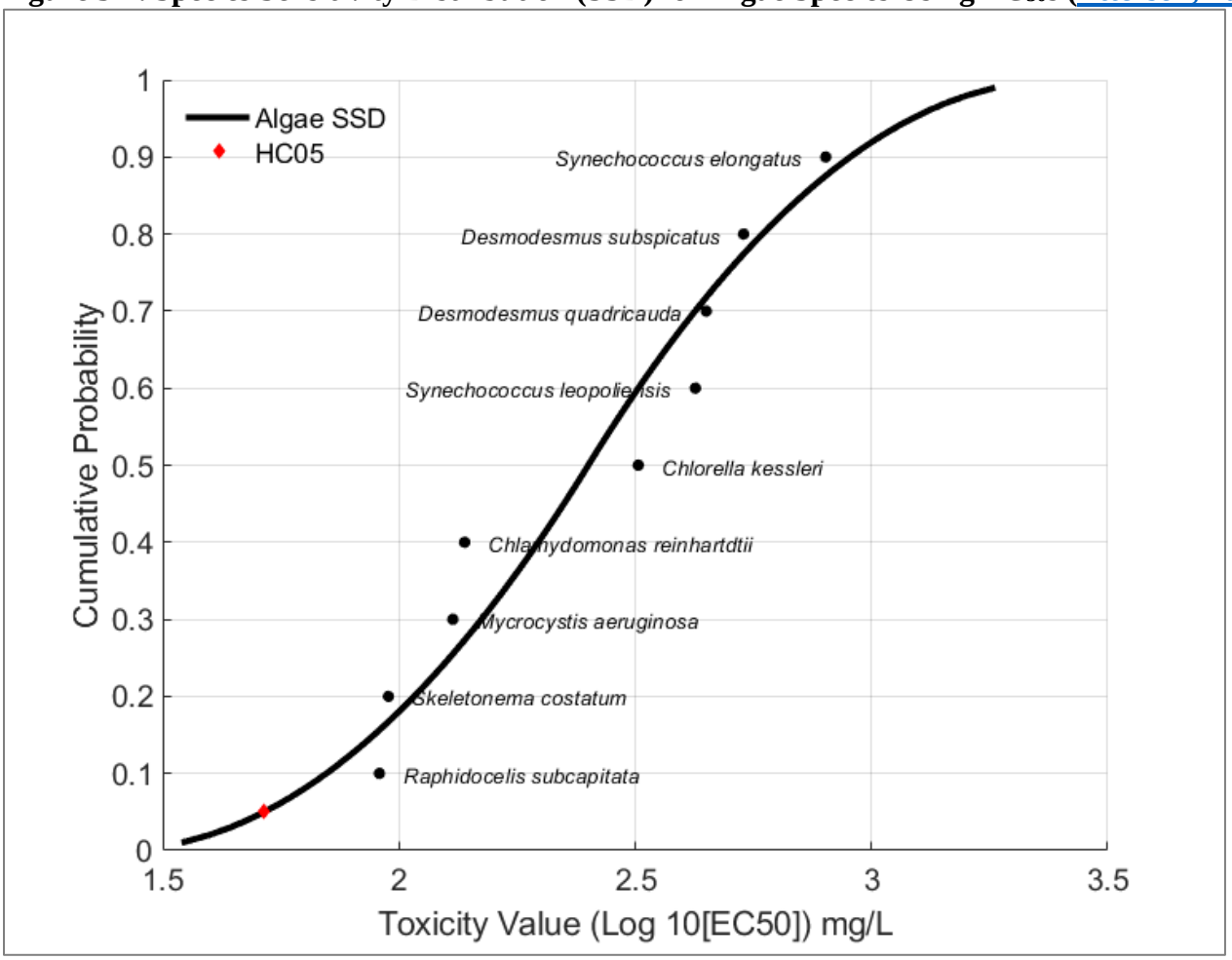
3.1.3 Species Sensitivity Distributions (SSDs)

A Species Sensitivity Distribution (SSD) is a type of probability distribution of toxicity values from multiple species. It can be used to visualize which species are most sensitive to a toxic chemical exposure, and to predict a concentration of a toxic chemical that is hazardous to a percentage of species. This hazardous concentration is represented as an HC_p, where p is the percent of species.

As stated previously, there were a wide range of toxicity values reported in the literature for algae exposed to TCE. EC_{50}s were as low as 26.24 mg/L and as high as 820 mg/L, representing nine different species. With such a wide range of sensitivities, it is helpful to show how TCE could be affecting algae species as a whole. Therefore, EPA generated an SSD to help interpret the data. Figure 3-1 shows the SSD for algae created using EPA's SSD Toolbox (Etterson, 2019). The data used in the SSD includes EC_{50}s measuring growth from freshwater species, a saltwater species, cyanobacteria, eukaryotes, a diatom, and a colonizing species. As stated in Section 3.1.2, saltwater and freshwater species were assessed together, because the only saltwater species, *Skeletonema costatum*, had an EC_{50} within that of the range of values for freshwater species.

An HC_{05} (Hazardous Concentration threshold for 5% of species) for algae of 52 mg/L was derived from this SSD.

Figure 3-1. Species Sensitivity Distribution (SSD) for Algae Species Using EC_{50}s (Etterson, 2019)



Note: The data in this figure includes EC_{50}s measuring growth from medium- or high-quality studies. A black dot indicates the toxicity value used for that species. The red diamond indicates an HC_{05}. The SSD was created using a triangular distribution and fit using graphical methods (Appendix E.1).

Given these data, certain algae species may be more sensitive than others; however, there is not enough data to make definitive conclusions. The three cyanobacteria, *Mycrocystis aeruginosa*, *Synechococcus leopoliensis*, *and Synechococcus elongatus*, are distributed throughout the curve and as a group do not appear to be more or less sensitive than the eukaryotic species. The saltwater species, *Skeletonema costatum*, also the only diatom, is one of the more sensitive species on the distribution. The species that organizes into colonies, *Mycrocystis aeruginosa*, is also one of the more sensitive species represented on the curve. However, with only one saltwater species, diatom, and colonizing species represented, generalizations about the sensitivity of these types of algae could not be made.

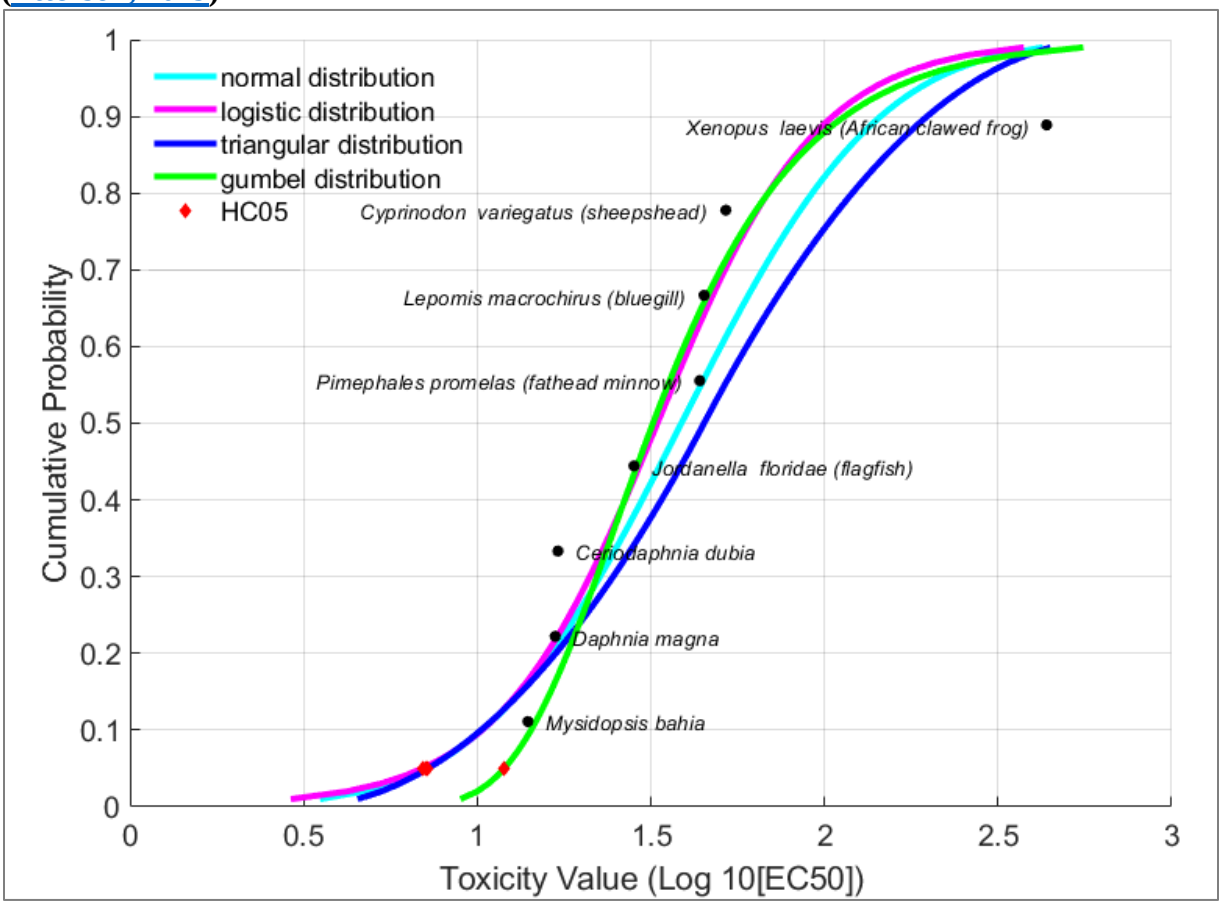
It is important note that, for consistency, this distribution only includes EC_{50}s to compare between studies and species. Therefore, it does not capture some of the lowest toxicity values reported, including LOECs and NOECs. For example, the ChV of 0.03 mg/L for algae derived from Labra et al. (2010) is not included in the algae SSD.

An SSD was also created using the acute hazard data, including LC_{50} and EC_{50} data for fish, amphibians, and invertebrates (Figure 3-2) (Etterson, 2019). The input data for Figure 3-2 included EC_{50}s and LC_{50}s available in the literature representing four species of fish (LC_{50}s), one species of amphibian (LC_{50}s), and three species of invertebrates (LC_{50}s/EC_{50}s). As stated previously, freshwater and saltwater species were assessed together, because the saltwater values were within the of the range freshwater species in

the same taxonomic group. Additionally, for fish and invertebrates, the mode of action for freshwater and saltwater species expected to be the same (Broderius et al., 2005; Ward et al., 1986; Alexander et al., 1978).

For the HC_{05} for acute hazard data, EPA used a model average of the Gumbel, triangular, normal, and logistic distributions (Figure 3-2). The model-averaged HC_{05} from all three distributions was 9.9 mg/L, which estimates a concentration that is hazardous for 5% of aquatic species. The SSDs showed aquatic invertebrates were the most sensitive species.

Figure 3-2. Species Sensitivity Distributions (SSDs) for Acute Hazard Data Using LC_{50}s or EC_{50}s (Etterson, 2019)



Note: The data in this figure includes LC_{50}s and EC_{50}s measuring mortality and immobilization from medium- or high-quality studies. A black dot indicates the toxicity value used for that species. The red diamonds indicate HC_{05}s for the normal, logistic, triangular, and Gumble distributions using the maximum likelihood fitting method (Appendix E.1).

This SSD shows that generally, invertebrates are the most sensitive taxonomic group to short-term (48-96 hour) exposure to TCE. Amphibians and fish were distributed throughout the center of the distribution, with the two frog species being the most sensitive amphibians, and American flagfish (*Jordanella floridae*) the most sensitive fish.

A chronic SSD for aquatic species was not created due to insufficient data.

3.1.4 Weight of the Scientific Evidence

During the data integration stage of systematic review EPA analyzed, synthesized, and integrated the data/information. This involved weighing the scientific evidence for quality and relevance, using a weight-of-evidence approach (U.S. EPA, 2018b).

During data evaluation, EPA assigned studies an overall quality level of high, medium, or low for quality 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 TCE, EPA gave more weight to relevant data/information rated high or medium for quality than to data/information rated low. Only data/information rated as high, medium, or low for quality was considered for the environmental risk assessment. Any information rated as unacceptable was not considered. EPA also considered relevance in selecting data/information for this risk evaluation, specifically 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 region of concern. (U.S. EPA, 1998)

EPA used this weight-of-evidence approach to assess hazard data and develop concentrations of concern (COCs) and HC_{05}s. Given the reasonably available data, EPA only used studies assigned an overall quality level of high or medium to derive COCs or HC_{05}s for each taxonomic group. 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). To calculate HC_{05}s, EPA created SSDs for algae species using comparable data (e.g., EC_{50}s measuring growth) and for all species (e.g., EC_{50}s and LC_{50}s measuring population effect measures, like growth, mortality, immobilization, and deformities). Non-definitive toxicity values (e.g., EC_{50} >85 mg/L) were not used to derive geometric means or HC_{05}s.

To assess aquatic toxicity from acute exposures, data for three taxonomic groups were reasonably available: fish, amphibians, 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 for aquatic invertebrates, 16 mg/L, represented the lowest toxicity value derived from each of the four taxonomic groups. The SSD in Figure 3-2 shows that the three most sensitive species in the distribution are aquatic invertebrates, further substantiating that this is the most sensitive taxonomic group to acute exposures.

To assess aquatic toxicity from chronic exposures, data for three taxonomic groups were described in the acceptable literature: fish, amphibians, and aquatic invertebrates. However, for amphibians, only a NOEC was established. Therefore, the endpoints for fish and aquatic invertebrates (ChVs, an EC_{20}, and an EC_{50}) were more biologically relevant, because they measured a toxic effect. Of these values, the most sensitive was the EC_{20} measuring growth in fish at 7.88 mg/L.

To assess the toxicity of TCE to algae, data for 11 species were reasonably available from studies rated high and medium for quality. The most sensitive endpoint reported for algae was a 10-day LOEC of 0.003 mg/L from Ando et al. (2003), rated medium for quality. However, the study did not include critical details, such as analytical measurement of test concentrations, or chemical substance source or purity, and the authors were not able to establish a NOEC. Therefore, these data were considered less

biologically relevant than values from other studies, and not used quantitatively during data integration. The ChV of 0.03 from Labra et al. (2010) was the most sensitive endpoint from the more relevant studies. Labra et al. (2010) was rated medium for quality. An EC_{10} of 12.3 mg/L from a high-quality study, Brack et al. (1994), was also available; however, taking biological relevance into consideration, EPA used the ChV derived from Labra et al. (2010), because there was a wide range in toxicity values reported in the literature between algae species. Therefore, EPA used the value from *Raphidocelis subcapitata* (formerly known as *Pseudokirchneriella subcapitata*) from Labra et al. (2010) to represent the more sensitive algae species in the COCs. (According to the algae SSD, *Raphidocelis subcapitata* is generally more sensitive to TCE exposure than *Chlamydomonas reinhartdtii*, the species used in Brack et al. (1994).) In addition to this ChV, EPA considered the results from the SSD for algae in assessing toxicity to algae. The SSD represented toxicity values for nine species of algae and provided an additional line of evidence for how TCE exposure could affect this taxonomic group.

3.1.5 Concentrations of Concern

The concentrations of concern (COCs) for aquatic species were calculated based on the environmental hazard data for TCE, using the weight of evidence approach described above and EPA methods (U.S. EPA, 2016i, 2012c). For TCE, EPA derived an acute COC, a chronic COC, and an algal COC. 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 evidence and selecting the appropriate toxicity values from the integrated data to calculate an acute, chronic, and algal COC, an assessment factor (AF) is applied according to EPA methods (U.S. EPA, 2016i, 2012c). 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 also account for differences in inter- and intra-species variability, as well as laboratory-to-field variability. These AFs are dependent on the availability of datasets that can be used to characterize relative sensitivities across multiple species within a given taxa or species group. However, they are often standardized in risk assessments conducted under TSCA, since the data reasonably 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 (U.S. EPA, 2012c).

To derive an acute COC for TCE, EPA used the geometric mean of the EC_{50} and LC_{50}s for aquatic invertebrates from five different studies, all rated high or medium for quality (Dobaradaran et al., 2012; Niederlehner et al., 1998; Abernethy et al., 1986; Ward et al., 1986; LeBlanc, 1980). The geometric mean for aquatic invertebrates represented the lowest acute value from all four taxonomic groups of aquatic species from the integrated data for TCE. The data used to calculate the geometric mean represent toxicity data for three species, *Daphnia magna*, *Ceriodaphnia dubia*, and *Mysidopsis bahia*. To calculate an acute COC, the geometric mean, 16 mg/L, was divided by the AF of 5 for aquatic invertebrates and multiplied by 1,000 to convert mg/L to µg/L (or ppb).

Therefore, the acute COC = (16 mg/L) / AF of 5 = 3.2 x 1,000 = 3,200 µg/L or ppb.

The acute COC for TCE is 3,200 ppb.

To derive a chronic COC, EPA used the lowest chronic toxicity value from the integrated data, an EC_{20} for growth in fish (fathead minnows) from a study rated high for quality (Broderius et al., 2005). This value, 7.88 mg/L was divided by an assessment factor of 10, and then multiplied by 1,000 to convert from mg/L to µg/L (or ppb).

Therefore, the chronic COC = (7.88 mg/L) / AF of 10 = 0.788 x 1,000 = 788 µg/L or ppb.

The chronic COC for TCE is 788 ppb.

To derive an algal COC, EPA used a geometric mean of a LOEC and a NOEC for growth in *Raphidocelis subcapitata* (Labra et al., 2010). This value, 0.03 mg/L was divided by an assessment factor of 10, and then multiplied by 1,000 to convert mg/L to µg/L (or ppb).

Therefore, the algal COC = (0.03 mg/L) / AF of 10 = 0.003 x 1,000 = 3 µg/L or ppb.

The algal COC for TCE is 3 ppb.

Additionally, EPA used algae data representing nine species to produce an SSD, which was used to calculate an HC_{05} of 52 mg/L (or 52,000 ppb). As stated previously, this HC_{05} estimates a concentration that is hazardous for 5% of species. The HC_{05} can be used in addition to the COC for algae, estimating the concentration of TCE that is expected to protect 95% of algae species.

The algal HC_{05} for TCE is 52,000 ppb.

3.1.6 Summary of Environmental Hazard

The reasonably available environmental hazard data indicate that TCE presents hazard to aquatic organisms. For acute exposures to invertebrates, toxicity values ranged from 7.8 to 33.85 mg/L (integrated into a geometric mean of 16 mg/L). For chronic exposures, toxicity values for fish and aquatic invertebrates were as low as 7.88 mg/L and 9.2 mg/L, respectively. The data also indicated that TCE presents hazard for aquatic plants, with toxicity values in algae as low as 0.03 mg/L (geometric mean between a NOEC and a LOEC), and a wide range in toxicity between algae species (EC_{50}s ranging from 26.24 – 820 mg/L).

The COCs derived for aquatic organisms are summarized in Table 3-2. EPA calculated the acute COC for TCE at 3,200 ppb, based on the geometric mean of LC_{50}s and EC_{50}s for aquatic invertebrates, from five studies rated either high or medium for quality (Dobaradaran et al., 2012; Niederlehner et al., 1998; Abernethy et al., 1986; Ward et al., 1986; LeBlanc, 1980). EPA calculated the chronic COC for TCE at 788 ppb, based on an EC_{20} for fathead minnows from Broderius et al. (2005), rated high for quality.

As stated previously, algae were assessed separately from other aquatic organisms, because durations normally considered acute for other species (e.g., 96 hours) can encompass several generations of algae. EPA calculated an algal COC for TCE at 3 ppb, based on a geometric mean of a LOEC and NOEC for growth in *Raphidocelis subcapitata* from Labra et al. (2010), a study rated medium for quality. EPA also calculated an HC_{05} of 52,000 ppb for algae based on the EC_{50}s for nine species, from studies rated medium and high for quality.

Table 3-2 Concentrations of Concern (COCs) for Environmental Toxicity

Environmental Aquatic Toxicity	Concentration of Concern
Toxicity from Acute Exposure	3,200 ppb
Toxicity from Chronic Exposure	788 ppb
Toxicity for Algae: COC based on the lowest toxicity value	3 ppb
HC_{05} based on EC_{50}s	52,000 ppb

3.1.7 Assumptions and Key Uncertainities for Environmental Hazard Data

While EPA determined that there was sufficient environmental hazard data to characterize environmental hazards of TCE, there are uncertainties. First, assessment factors (AFs) were used to calculate the acute and chronic concentrations of concern for TCE. As described in Section 3.1.5, AFs account for differences in inter- and intra-species variability, as well as laboratory-to-field variability and are routinely used within TSCA for assessing the hazard of new industrial chemicals. Some uncertainty may be associated with the use of the specific AFs used in the hazard assessment.

Second, there was more acute duration data reasonably available in the literature than chronic duration data. Therefore, EPA is less certain of chronic hazard values, which are based on one fish species, than the acute hazard values, which are based on data from multiple species of aquatic invertebrates. However, a few lines of evidence mitigate the uncertainty in the chronic data. For example, the fish toxicity value on which the chronic COC is based, is from a high-quality, relevant study. Additionally, the acute data show aquatic invertebrates are the most sensitive taxonomic group, and they are represented in chronic duration data. Also, the other chronic fish toxicity values as well as the chronic aquatic invertebrate values were very close to the fish value used to derive the chronic COC. Therefore, some of the uncertainties EPA had around the chronic COC were mitigated.

Third, while the toxicity values for fish, amphibians, and invertebrates are relatively consistent, there was wide variation in the toxicity values for different species of algae. One study, Lukavsky et al. (2011) examined several species of algae using standardized methods within the same lab to determine whether the variation seen in the literature was due to differences in laboratory practices, methodology used, or species studied. They found that conducting the tests with standard methods in the same lab reduced the variation seen in toxicity levels between species; however, EC_{50}s were still as low as 130 mg/L and as high as 820 mg/L for the eight species of algae tested (compared to a range of 26.24 – 820 mg/L from the entire body of literature), indicating there is in fact a wide range in species sensitivities. Taking this range of sensitivies into consideration, EPA used two approaches to characterize hazard in algae. EPA developed an algae COC, using a toxicity value of 0.03 mg/L, which represents one species. The data show that there are other species that are less sensitive to TCE exposure. To provide more context for this taxonomic group, EPA also used algae data from nine species to create an SSD and derive an HC_{05}. EPA considered the HC_{05} analogous to a COC. However there are pros and cons to each approach. For example, the COC incorporates the most sensitive endpoint in a geometric mean of a NOEC and LOEC for growth, while the HC_{05} does not consider the most sensitive endpoints reported in the data. However, the HC_{05} is derived using data from nine species rather than just one, and is therefore representative of a larger portion species in the environment.

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 TCE's human health hazard and dose-response information.

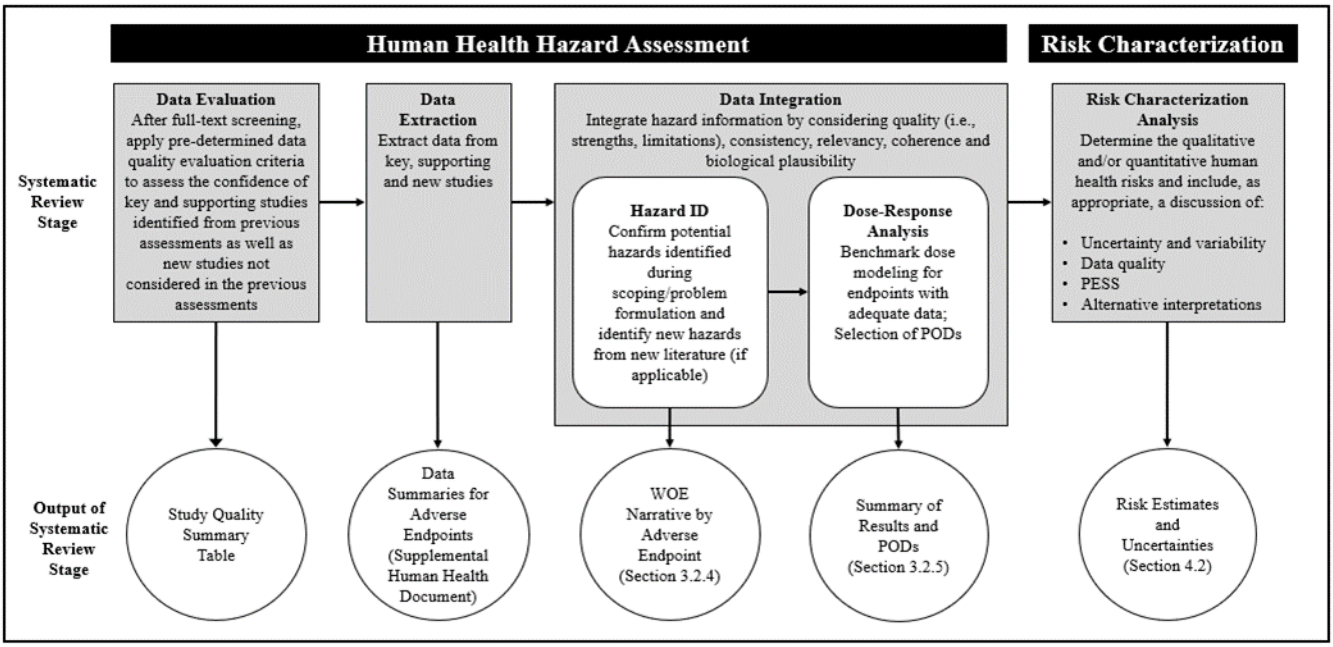


Figure 3-3. EPA Approach to Hazard Identification, Data Integration, and Dose-Response Analysis for TCE

Specifically, EPA reviewed key and supporting information from previous hazard assessments as well as the existing body of knowledge on TCE's human health hazards. These data sources included an EPA IRIS Assessment (U.S. EPA, 2011e) and an ATSDR Toxicological Profile ((ATSDR, 2019), data sources originally obtained from the 2014 Draft Toxicological Profile); hence, many of the hazards of TCE have been previously compiled and systematically reviewed. Furthermore, EPA previously reviewed data/information on health effects endpoints, identified hazards and conducted dose-response analysis in the 2014 TSCA Work Plan Chemical Risk Assessment for TCE (U.S. EPA, 2014b) but did not exclusively rely on this assessment.

All health hazards of TCE previously identified in these reviews were described and reviewed in this risk evaluation, including: acute overt toxicity, liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity (including sensitization), reproductive toxicity, developmental toxicity, 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 TCE hazard and dose-response assessments considered EPA and National Research Council (NRC) risk assessment guidance.

The new literature was screened against inclusion criteria in the PECO statement and the relevant studies (e.g., useful for dose-response)[15] were further evaluated using the data quality criteria for human, animal, and *in vitro* studies described in the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018b) (see Section 1.5). EPA skipped the screening step (for relevance to TCE) 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.

EPA considered studies of low, medium, or high confidence 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 3-3. The weight of evidence 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.

Tables summarizing all studies considered for this assessment, including the reported no-observed- or lowest-observed-adverse-effect levels (NOAEL and LOAEL) for non-cancer health endpoints by target organ/system and the incidence for cancer endpoints, along with the results of the data quality evaluation, are provided in [*Data Quality Evaluation of Human Health Hazard Studies* and *Data Extraction for Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]*.

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).[16] PODs were adjusted as appropriate to conform to the specific exposure scenarios evaluated.

[15] 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.

[16] 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.

Human equivalent concentrations (HECs) and human equivalent doses (HEDs) were obtained via EPA's previously published and peer-reviewed Physiologically-Based Pharmacokinetic (PBPK) model (U.S. EPA, 2011e), which accounts for both extrapolation from rodents to humans and human variability (see Section 3.2.2.1 and [*PBPK Model and ReadMe (zipped). Docket: EPA-HQ-OPPT-2019-0500]*). The PBPK model also allows data-based route-to-route extrapolation between oral and inhalation studies. For HEC calculations, these values were adjusted based on 24-hr exposure durations unless otherwise noted. Limited toxicological data are reasonably available by the dermal route for TCE and a PBPK model that would facilitate route-to-route extrapolation has not been developed for the dermal exposure route. Therefore, oral HEDs were also utilized for risk estimation following dermal exposure, consistent with the analysis plan as described in the Problem Formulation (U.S. EPA, 2018d).

Section 3.2.5 describes the dose-response assessment guiding the selection of PODs for non-cancer endpoints. The BMD modeling results for pulmonary immunotoxicity (Selgrade and Gilmour, 2010), which was not included in the 2014 TCE Risk Assessment (U.S. EPA, 2014b), are presented in Appendix F. The full description of the PBPK and BMD model outputs for all other endpoints can be found in (U.S. EPA, 2011e).

3.2.2 Toxicokinetics

The toxicokinetics and PBPK modeling of TCE were thoroughly discussed in the 2014 Risk Assessment (U.S. EPA, 2014b). This discussion is summarized below.

TCE is fat soluble (lipophilic) and easily crosses biological membranes. Though there are quantitative differences across species and routes, TCE is readily absorbed into the body following oral, dermal, or inhalation exposure. Because of its lipophilicity, TCE can cross the placenta and also passes into breast milk (U.S. EPA, 2011e).

Absorption following inhalation of TCE is rapid and the inhaled absorbed dose is proportional to the exposure concentration, duration of exposure, and lung ventilation rate. Therefore, for this risk evaluation absorption of TCE is assumed to be 100% via inhalation. Likewise, TCE is rapidly absorbed from the gastrointestinal tract into the systemic circulation (i.e., blood) following oral ingestion. Oral absorption of TCE has been shown to be influenced by dose of the chemical, the dosing vehicle and stomach contents. Absorbed TCE is first transported to the liver where it is metabolized for eventual elimination (i.e., "first-pass effect") (U.S. EPA, 2011e).

Rapid absorption through the skin has been shown by both vapor and liquid TCE contact with the skin. In several human volunteer studies, both TCE liquid and vapors were shown to be well absorbed in humans via the dermal route. Dermal absorption was rapid following exposures of between 20 and 30 minutes, with peak TCE levels in expired air occurring within 15 minutes (liquid) and 30 minutes (vapor) (U.S. EPA, 2011e). Dermal exposure to TCE disrupts the stratum corneum, impacting the barrier function of skin and promoting its own absorption. Therefore, absorption may increase at a greater than linear rate due to increasing epidermal disruption over time (ATSDR, 2019). Based on this information, this risk evaluation assumes that TCE dermal absorption under occluded (or impeded evaporation) scenarios is 100%. Dermal absorption under non-occluded occupational exposure scenarios was evaluated by the Dermal Exposure to Volatile Liquids Model in order to account for evaporation of TCE deposited on skin (Section 2.3.1). Consumer exposure was only evaluated for scenarios that may involve dermal contact with impeded evaporation using a skin permeability model with a dermal permeability coefficient of 0.019 cm/hr (Section 2.3.2.4.1).

Regardless of the route of exposure, TCE is widely distributed throughout the body. TCE levels can be found in many different human and rodent tissues including: brain, muscle, heart,

kidney, lung, liver, and adipose tissues. It can also be found in human maternal and fetal blood and in the breast milk of lactating women (U.S. EPA, 2011e).

The metabolism of TCE has been extensively studied in humans and rodents (U.S. EPA, 2011e). Animals and humans metabolize TCE to metabolites to varying degrees. These metabolites are known to play a key role in causing TCE-associated toxic effects. TCE metabolites are known to target the liver and kidney. The two major metabolic pathways are (1) oxidative metabolism via the cytochrome P450 (CYP) mixed function oxidase system and (2) glutathione (GSH) conjugation followed by further biotransformations and processing with other enzymes. The liver is the major tissue for the oxidative and GSH conjugation metabolic pathways. Both pathways are saturable, and above the saturable concentration/dose, TCE is excreted unchanged in expired air. Table 3-3 presents the important metabolites formed following both the CYP (oxidation) and GSH (conjugation) pathways in humans and animals. The amount and types of metabolites formed are important for understanding the toxicity of TCE in both animals and humans.

These major TCE metabolites as well as a number of minor metabolites are also observed in the metabolic pathway of TCE-related compounds (Table 3-4). This may be important in determining exposures because people may be co-exposed to many of these solvents at the same time. Concomitant exposures to TCE and its related compounds can affect TCE's metabolism and increase toxicity by generating higher internal metabolite concentrations than those resulting from TCE exposure only (U.S. EPA, 2011e).

Table 3-3 TCE Metabolites Identified by Pathway

Oxidative Metabolites	GSH Conjugation Metabolites
Chloral ***(metabolized to TCOH[a])***	DCVG[e] (*metabolized to DCVC[f] isomers*)
Trichloroethylene oxide ***(re-arranged to DCAC[b])***	
Trichloroethanol or TCOH ***(metabolized to TCOG[c])***	
Trichloroacetic acid or TCA ***(may lead to DCA[d])***	

Abbreviations: [a] TCOH = trichloroethanol; [b] DCAC= dichloroacetyl chloride; [c] TCOG= trichloroethanol, glucuronide conjugate; [d] DCA=dichloroacetic acid; [e] DCVG= S-dichlorovinyl-glutathione (collectively, the 1,2- and 2,2- isomers); [f] DCVC= S-dichlorovinyl-L-cysteine (collectively, the 1,2- and 2,2- isomers)

A review of *in vitro* metabolism data in the liver suggested that rodents (i.e., especially mice) have greater capacity to metabolize TCE via the oxidation pathway (U.S. EPA, 2011e). *In vitro* data have also reported modest sex- and age-dependent differences in the oxidative TCE metabolism in humans and animals. Significant variability may exist in human susceptibility to TCE toxicity given the existence of CYP isoforms and the variability in CYP-mediated TCE oxidation (U.S. EPA, 2011e).

Table 3-4 Common Metabolites of TCE and Related Compounds

Parent ↓ / →	Tetrachloro-ethylene	1,1,2,2,-	TCE	1,1,1-	1,2,-	1,2,-

Metabolites		Tetrachloro-ethane		Trichloro-ethane	Dichloro-ethylene	Dichloro-ethane
Oxalic acid		X	X		X	
Chloral	X		X			
Chloral hydrate (CH)	X		X			
Monochloroacetic acid	X	X	X	X	X	X
Dichloroacetic acid (DCA)	X	X	X			X
Dichloroacetic acid (TCA)	X	X	X	X		
Trichloroethanol (TCOH)	X	X	X	X		
Trichloroethanol-glucuronide	X	X	X	X		

Note: Table is the same as Table 2-21 in (U.S. EPA, 2014b).

Conjugation is a process that generally leads to detoxification. However, this is not the case for TCE and many other halogenated alkanes and alkenes because they are biotransformed into reactive metabolites. The eventual metabolite(s) of concern for TCE are formed several steps from the initial GSH conjugate formed in the liver, which ultimately results in toxicity or carcinogenicity in the kidney (U.S. EPA, 2011e).

Compared to the CYP oxidation pathway, there appear to be more significant sex and species differences in TCE metabolism via the GSH pathway (U.S. EPA, 2011e). Animal data show that rates of TCE GSH conjugation in male rats/mice are higher than females. According to some *in vitro* data, the rates of DCVG production in liver/kidney cytosol are highest in humans, followed by mice, and then rats. *In vitro* data also suggest that γ-glutamyl transpeptidase (i.e., GGT, an enzyme involved in DCVC production) activity in kidneys seems to be highest in rats, then humans, and then mice (U.S. EPA, 2011e). Furthermore, species-dependent enzymatic activities have been reported for the β-lyase and FMO3 enzymes (U.S. EPA, 2011e).

The majority of TCE absorbed into the body is eliminated by the metabolic pathways discussed above. With the exception of unchanged TCE and CO_2, which are excreted by exhalation, most TCE metabolites (i.e., TCA, TCOH, GSH metabolites) are primarily excreted in urine and feces. Elimination of TCE metabolites can also occur through the sweat and saliva, but these excretion routes are likely to be relatively minor (U.S. EPA, 2011e).

Varying rates of TCE pulmonary excretion in humans have been observed in different studies (Chiu et al., 2007; Opdam, 1989; Sato et al., 1977). The relatively long terminal half-lives observed (up to 44 hours) suggest that the lungs require considerable time to completely eliminate TCE, primarily due to high partitioning to adipose tissues (U.S. EPA, 2011e). Various laboratories have studied the urinary elimination kinetics of TCE and its major metabolites in humans and rodents. Animal studies have shown that rodents exhibit faster urinary elimination kinetics than humans, with demonstrated

elimination half-lives of just over 50 hours in humans and only approximately 16 hours in rats (Ikeda and Imamura, 1973).

3.2.2.1 Physiologically-Based Pharmacokinetic (PBPK) Modeling Approach

Given the complicated metabolic profile of TCE, understanding the relationship between the external dose/concentration (*i.e.*, exposure) and internal dose at the target organ of interest is critical to quantifying potential risk(s) because internal dose is more closely associated with toxicity at the target tissue (U.S. EPA, 2006). Predictions of internal dose in chemical risk assessments are achieved by employing PBPK modeling.

PBPK models use a series of mathematical representations to describe the absorption, distribution, metabolism and excretion of a chemical and its metabolites. Because PBPK modeling assumes that the toxic effects in the target tissue are closely related to the internal dose of the biologically active form of the chemical, knowledge about the chemical's mode of action guides the selection of the appropriate dose metric. Traditional risk estimates based on applied dose carry higher uncertainties than those based on PBPK-derived internal dose metrics. This reduction in uncertainty and the versatility of PBPK approaches have resulted in a growing interest to use these models in risk assessment products (U.S. EPA, 2006).

U.S. EPA developed a peer-reviewed comprehensive Bayesian PBPK model-based analysis of TCE and its metabolites in mice, rats and humans (U.S. EPA, 2011e). This model is briefly discussed below to provide clarity on how the PBPK modeling was used to estimate the PBPK-derived HECs. For all PBPK model files, including inputs and outputs of all model runs, see [*PBPK Model and ReadMe (zipped). Docket: EPA-HQ-OPPT-2019-0500]*.

Physiological, chemical, *in vitro* and *in vivo* data were considered when building the PBPK model, including many studies in animals and humans that quantified TCE levels in various tissues following oral and inhalation exposures. Some of these studies provided key data/ parameters for the calibration of the PBPK model used in the IRIS assessment (U.S. EPA, 2011e). All of this information was used to build a model that was able to predict different dose metrics as measures of potential TCE toxicity. Each dose-metric was developed to evaluate a different metabolic pathway/target organ effect based on the dose-response analysis and understanding of metabolism (Table 3-5 and Figure 3-4).

In general, an attempt was made to use tissue-specific dose-metrics representing particular pathways or metabolites identified from reasonably available data on the role of metabolism in toxicity for each endpoint (discussed in more detail below). The selection was limited to dose metrics for which uncertainty and variability could be adequately characterized by the PBPK model. For most endpoints, sufficient information on the role of metabolites or mode of action was not available to identify likely relevant dose metrics, and more upstream metrics representing either parent compound or total metabolism had to be used.

Table 3-5 List of All of the PBPK-Modeled Dose Metrics Used in the TCE IRIS Assessment

Dose-Metric Identifier	*Dose-Metric Definition*
ABioactDCVCBW34	Amount of DCVC bioactivated in the kidney per unit adjusted body weight
ABioactDCVCKid	Amount of DCVC bioactivated in the kidney per unit kidney mass
AMetGSHBW34	Amount of TCE conjugated with GSH per unit adjusted body weight
AMetLiv1BW34	Amount of TCE oxidized in liver per unit adjusted body weight
AMetLivOtherBW34	Amount of TCE oxidized to metabolites other than TCA or TCOH per unit adjusted body weight

AMetLivOtherLiv	Amount of TCE oxidized to metabolites other than TCA or TCOH per unit liver weight
AMetLngBW34	Amount of TCE oxidized in respiratory tract per unit adjusted body weight
AMetLngResp	Amount of TCE oxidized in respiratory tract per unit respiratory tract tissue
AUCCBld	Area under the curve of venous blood concentration of TCE
AUCCTCOH	Area under the curve of blood concentration of TCOH
AUCLivTCA	Area under the curve of the liver concentration of TCA
TotMetabBW34	Total amount of TCE metabolized per unit adjusted body weight
TotOxMetabBW34	Total amount of TCE oxidized per unit adjusted body weight
TotTCAInBW	Total amount of TCA produced

For developmental toxicity endpoints, the TCE PBPK model did not incorporate a pregnancy model to estimate the internal dose of TCE in the developing fetus. In this case, the maternal dose-metric was used as the surrogate measure of target tissue dose in the developing fetus. A complete description of the TCE PBPK model, including the rationale for parameter choices in animals and humans, choice of dose metric, and experimental information used to calibrate and optimize the model is found in the TCE IRIS assessment (U.S. EPA, 2011e).

As shown in Figure 3-4 and Figure 3-5, several steps were needed to derive the PBPK-derived HECs used in this assessment. First, the rodent PBPK model was run to estimate rodent internal dose Points of Departure (idPODs) for the applied dose PODs (i.e., LOAEL, NOAEL, or BMDL) that were identified in the TCE IRIS assessment. Separately, the human PBPK model was run for a range of continuous exposures from 0.1 to 2,000 ppm or 0.1 to 2,000 mg/kg-bw/day to establish the relationship between human exposure air levels and internal dose for the same dose-metric evaluated in the rodent PBPK model. This relationship was used to derive Human Equivalent Concentrations (HECs) and Human Equivalent Doses (HEDs) corresponding to the idPOD by interpolation (U.S. EPA, 2011e).

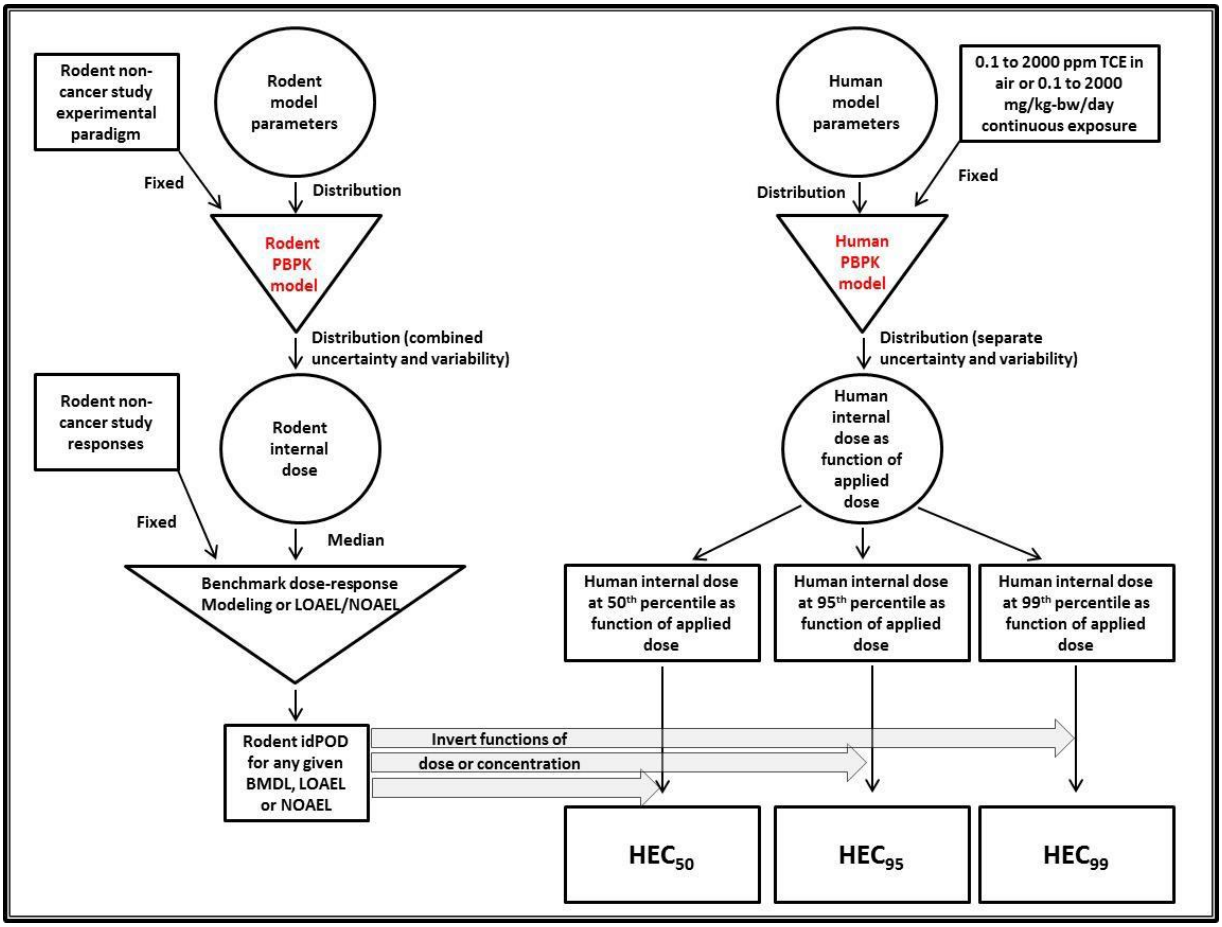


Figure 3-4 Dose-Response Analyses of Rodent Non-Cancer Effects Using the Rodent and Human PBPK Models

Notes: Figure adapted from Figure 5-2 (Chapter 5, TCE IRIS assessment) (U.S. EPA, 2011e). Square nodes indicate point values, circle nodes indicate distributions and the inverted triangle indicates a (deterministic) functional relationship.

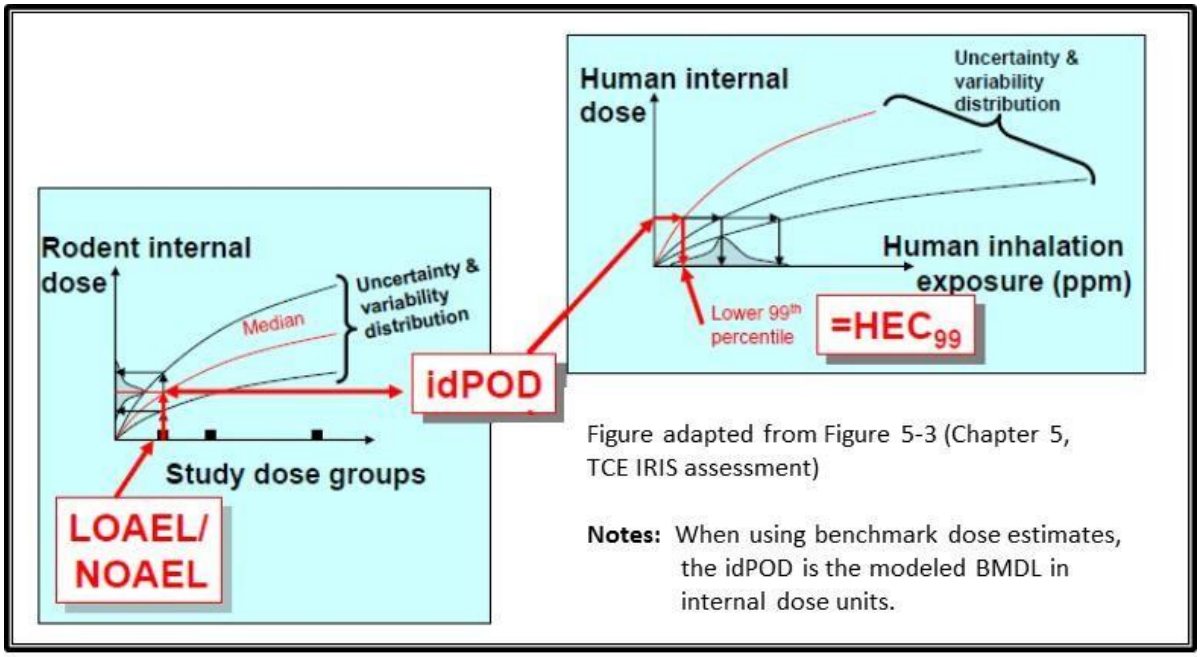


Figure 3-5 Example of HEC99 Estimation through Interpecies, Intraspecies and Route-to- Route Extrapolation from a Rodent Study LOAEL/NOAEL

The rodent population model was designed to characterize study-to-study variation and used median values of dose-metrics to generate idPODs. The rodent PBPK model did not characterize variation within studies and assumed that the rodent idPODs were for pharmacokinetically identical animals. The basis of that assumption was that animals with the same sex/species/strain combination were considered pharmacokinetically identical and represented by the group average. In practice, the use of median or mean internal doses for rodents did not make much difference except when the uncertainty in the rodent dose-metric was high (U.S. EPA, 2011e).

On the other hand, the human population model characterizes toxicokinetic uncertainty and individual-to-individual variation and used median, 95th and 99th percentile values of dose- metrics to general human idPODs. The 50th, 95th, or 99th percentile of the combined uncertainty and variability distribution of human internal doses was used to derive the HEC/HED$_{50}$, HEC/HED$_{95}$ or HEC/HED$_{99}$ estimates, respectively. The HEC$_{95}$ and HEC$_{99}$ were interpreted as being the concentrations of TCE in air for which there is 95% and 99% likelihood, respectively, that a randomly selected individual will have an internal dose less than or equal to the idPOD derived from the rodent study. HED values represent the same likelihood for given administered doses of TCE. This risk evaluation presents both HEC/HED$_{50}$ and HEC/HED$_{99}$ POD values.

3.2.3 Hazard Identification

3.2.3.1 Non-Cancer Hazards

EPA previously identified human health hazard for the below endpoints in (U.S. EPA, 2011e) and (U.S. EPA, 2014b). Key and supporting studies from those publications that were used for derivation of tissue-specific PODs were reviewed along with any newer studies identified through EPA's updated literature search beginning with studies published after the TCE IRIS assessment (U.S. EPA, 2011e). A short summary of the overall database and short details on any older key studies or relevant new studies are provided here; details on all reviewed studies can be found in [*Data Extraction for Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500*].

3.2.3.1.1 Liver toxicity

Animals and humans exposed to TCE consistently experience liver toxicity. Specific effects include the following structural changes: increased liver weight, increase in deoxyribonucleic acid (DNA) synthesis (transient), enlarged hepatocytes, enlarged nuclei, and peroxisome proliferation.

The role of metabolites is important but not well understood. Many investigators have dosed animals with TCE, as well as with many of its metabolites to determine the role and potency of each in terms of target organ toxicity. It appears that the oxidation pathway is important for the development of liver toxicity, but the specific role of each metabolite (i.e., that of TCA, DCA, and chloral hydrate), as well as the parent TCE, is unclear.

EPA did not identify any new repeat-dose experimental studies in animals or human epidemiological studies that would contribute significant additional hazard information for this endpoint. Therefore, EPA relied primarily on conclusions from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).

Human Data

Several human studies (including those in TCE degreaser operations) reported an association between TCE exposure and significant changes in serum liver function tests used in diagnosing liver disease, or changes in plasma or serum bile acids. There was also human evidence for hepatitis accompanying immune-related generalized skin diseases, jaundice, hepatomegaly, hepatosplenomegaly, and liver failure in TCE-exposed workers (U.S. EPA, 2011e). Cohort studies examining cirrhosis and either TCE exposure or solvent exposure did not generally identify a statistically significant association, but due to limitations in this database these studies do not rule out an association between TCE and liver disorders/toxicity (U.S. EPA, 2011e). A case study published after the 2011 IRIS Assessment reported TCE hypersensitivity-induced liver damage (Jung et al., 2012).

Animal Data

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) reviewed many oral and inhalation studies in rats and mice. Studies in animals exposed to TCE reported increased liver weight, a small, transient increase in DNA synthesis, enlarged hepatocytes, increased size of nuclei of liver cells, and proliferation of peroxisomes (U.S. EPA, 2011e). Dose-responsive increases in relative liver weight (compared to body weight) were observed both following administration of TCE for 6 weeks via gavage (Buben and O'Flaherty, 1985) and for up to 120 days via inhalation (Woolhiser et al., 2006; Kjellstrand et al., 1983). Hypertrophy, histopathology, cytotoxicity, and altered serum biochemistry were also observed in mice in (Buben and O'Flaherty, 1985) and (Kjellstrand et al., 1983). Increased liver weight was additionally observed in (Boverhof et al., 2013), identified in the EPA literature

search, following 6hr/day inhalation exposure to a single concentration level (1000ppm) of TCE for 4 weeks.

3.2.3.1.2 Kidney toxicity

Studies in both humans and animals have shown changes in the proximal tubules of the kidney following exposure to TCE. DCVC (and to a lesser extent other metabolites) appears to be responsible for kidney damage and kidney cancer following TCE exposure (U.S. EPA, 2011e). Toxicokinetic data suggest that the TCE metabolites derived from GSH conjugation (in particular DCVC) can be systemically delivered or formed in the kidney. Importantly, DCVC-treated animals showed the same type of kidney damage as those treated with TCE (U.S. EPA, 2011e).

EPA did not identify new any repeat-dose experimental studies in animals or human epidemiological studies that would contribute significant additional hazard information for this endpoint. Therefore, EPA relied primarily on conclusions from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).

Human Data

Occupational studies showed increased levels of kidney damage (proximal tubules) and end-stage renal disease in TCE-exposed workers. Human studies reported increased excretion of urinary proteins among TCE-exposed workers when compared to unexposed controls. While some of these studies included subjects previously diagnosed with kidney cancer, other studies report similar results in subjects who are disease free (U.S. EPA, 2011e).

Animal Data

In animal studies, renal toxicity was evident in both rats and mice following inhalation or gavage exposures. Maltoni and Cotti (1986) identified pathological changes in the renal tubule of rats following 1-2 years of either oral or inhalation exposure. Similar changes were also observed in a chronic gavage study in female mice conducted by NCI, (NCI, 1976), however that study scored Unacceptable in EPA data quality evaluation due to confounding mortality. The toxicity included damage to the renal tubules (e.g., both cytomegaly and karyomegaly). In a chronic gavage study, kidney toxicity was observed in almost 100 percent of rodents at high doses (NTP, 1988). Under inhalation exposure scenarios, male rats were more susceptible than female rats or mice to kidney toxicity. As noted earlier, this toxicity is likely caused by DCVC formation, with possible roles for TCOH and TCA (U.S. EPA, 2011e). Increased relative kidney weight compared to body weight was also observed in both mice and rats following inhalation exposure over several weeks to months (Boverhof et al., 2013; Woolhiser et al., 2006; Kjellstrand et al., 1983).

3.2.3.1.3 Neurotoxicity

Neurotoxicity has been demonstrated in animal and human studies under both acute and chronic exposure conditions (U.S. EPA, 2011e). Due to the effects on the nervous system, TCE was initially synthesized for use as an anesthetic in humans in the early part of the 20th century.These anesthetic-like effects occurred at high concentrations. CNS depression has been consistently observed following acute exposure of humans to TCE (see Section 3.2.3.1.7).

Among newer studies not previously discussed in (U.S. EPA, 2011e), a single repeat-dose experimental study in rats (Liu et al., 2010) along with a few epidemiological studies that identified specific neurological outcomes were identified in EPA's literature search. These studies only add to and do not contradict the hazard conclusions from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). Therefore, EPA primarily relied on the previous hazard conclusions.

Human Data

Evaluation of the human studies has reported the following TCE-induced neurotoxic effects: alterations in trigeminal nerve and vestibular function, auditory effects, changes in vision, alterations in cognitive function, changes in psychomotor effects, and neurodevelopmental outcomes (U.S. EPA, 2011e).

Multiple epidemiological studies in different populations have reported TCE-induced abnormalities in trigeminal nerve function in humans, with a few studies not reporting any association (U.S. EPA, 2011e). The strongest evidence of human neurological hazard is for observed changes in trigeminal nerve function or morphology and impairment of vestibular function in a High quality study on workers exposed to TCE for a mean of 16 years (Ruijten et al., 1991). Fewer and more limited epidemiological studies are suggestive of TCE exposure being associated with delayed motor function, and changes in auditory, visual, and cognitive function or performance, and neurodevelopmental abnormalities (U.S. EPA, 2011e).

Human studies have consistently reported vestibular system-related symptoms such as headaches, dizziness, and nausea following TCE exposure. Although these symptoms are subjective and self-reported, these effects have been reported extensively in human chamber, occupational, and geographic-based/drinking water studies (U.S. EPA, 2011e). Additionally, several newer epidemiological studies have found an association between TCE exposure and neurodegenerative disorders such as Amyotrophic Lateral Sclerosis (Bove et al., 2014a) and Parkinson's disease (Bove et al., 2014b; Goldman et al., 2012).

Animal Data

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) reviewed many animal studies reporting a variety of neurotoxic effects under different exposure conditions. Animal studies have reported the following TCE-induced neurotoxic effects: morphological changes in the trigeminal nerve, disruption of the auditory system, visual changes, structural or functional changes in the hippocampus, sleep disturbances and changes in psychomotor effects (U.S. EPA, 2011e). Key and supporting studies considered in this risk evaluation identified significant decreases in wakefulness following 6 weeks of TCE inhalation exposure (Arito et al., 1994) and demyelination of the hippocampus following 8 weeks of drinking water exposure (Isaacson et al., 1990) in rats. Neuronal degeneration (Gash et al., 2008) and diminished sciatic nerve regeneration (Kjellstrand et al., 1987) were also observed following TCE exposure in rodents, however those studies scored Low and Unacceptable, respectively in data quality evaluation. More recent studies have observed both sedative (Wilmer et al., 2014) and stimulatory effects (Shelton and Nicholson, 2014) of TCE via inhalation at doses at or above 5000 ppm. Rats administered TCE via gavage for 6 weeks demonstrated loss of dopaminergic neurons at 500 and 1000 mg/kg-day, with changes in behavior and reduced mitochondrial activity with increased oxidative stress observed at 1000 mg/kg-day (Liu et al., 2010).

3.2.3.1.4 Immunotoxicity (including sensitization)

Immune-related effects following TCE exposures have been observed in both animal and human studies. In general, these effects were associated with inducing enhanced immune responses as opposed to immunosuppressive effects. Of concern are the immune-related and inflammatory effects reported in TCE-exposed animals and humans. These effects may influence a variety of other conditions of considerable public health importance, such as cancer and atherosclerosis (U.S. EPA, 2011e).

EPA's literature search identified a single acute inhalation study in rats that identified a novel endpoint for impaired response to infection (Selgrade and Gilmour, 2010). This study was discussed in the TCE IRIS assessment (U.S. EPA, 2011e) but was not included in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). All other studies supported the hazard conclusions of the 2014 TCE Risk Assessment (U.S. EPA, 2014b). Therefore, EPA primarily relied on the previous hazard conclusions for all other endpoints.

Human Studies

Studies have reported a relationship between systemic autoimmune diseases, such as scleroderma, and occupational exposure to TCE. The TCE IRIS assessment (U.S. EPA, 2011e) performed a meta-analysis of a number of human studies evaluating a possible connection between scleroderma and TCE exposure. Results indicated a significant odds ratio (OR) in men, whereas women showed a lower but not significant OR. These results may not reflect a true gender difference because the incidence of this disease is very low in men (approximately one per 100,000 per yr) and somewhat higher in women (approximately one per 10,000 per yr). In addition, these results may be affected by gender-related differences in exposure prevalence, the reliability of the exposure assessment, gender-related differences in susceptibility to TCE toxicity or chance (U.S. EPA, 2011e).

Increased levels of human inflammatory cytokines have been observed in both workers exposed occupationally to TCE and infants exposed to TCE via indoor air. (U.S. EPA, 2011e). These findings were supported by studies in mice (described below) in which short exposures to TCE resulted in increased levels of inflammatory cytokines.

The epidemiological database also provides evidence of immunosuppression based on reduced IgG antibody levels in TCE-exposed workers (Zhang et al., 2013).

Animal Data

Numerous studies have shown increased autoimmune responses in autoimmune-prone mice, including changes in cytokine levels similar to those reported in human studies, with more severe effects, including autoimmune hepatitis, inflammatory skin lesions, and alopecia, manifesting at longer exposure periods (U.S. EPA, 2011e). Key studies identified evidence of autoimmunity from chronic TCE exposure in both non-autoimmune prone (Keil et al., 2009) and autoimmune prone (Kaneko et al., 2000) mice. Evidence of localized immunosuppression has also been reported in mice and rats (Boverhof et al., 2013; Woolhiser et al., 2006; Sanders et al., 1982). Support for immunotoxicity hazard is further supported by decreased thymus weight and cellularity in the non-autoimmune prone mice following up to 30 weeks of drinking water exposure (Keil et al., 2009).

Inhalation exposure to TCE has been shown to suppress pulmonary host defenses and enhance susceptibility to respiratory infection in mice co-exposed to aerosolized pathogenic bacteria. Increased mortality was observed post-infection following exposure to TCE concentrations of 50ppm or greater, with corresponding dose-dependent effects on bacterial clearance, percentage of infected mice, and alveolar phagocytosis (Selgrade and Gilmour, 2010).

Sensitization / Hypersensitivity

Limited epidemiological data do not support an association between TCE exposure and allergic respiratory sensitization or asthma. However, there have been a large number of case reports in TCE-exposed workers developing a severe hypersensitivity skin disorder, distinct from contact dermatitis, and often accompanied by systemic effects (e.g., hepatitis, lymph node changes, and other organ

effects). These effects appeared after inhalation exposures ranging from less than 9 to greater than 700 ppm TCE. Similar sensitization/hypersensitivity effects have been observed in guinea pigs and mice following TCE exposure via drinking water (U.S. EPA, 2011e).

3.2.3.1.5 Reproductive toxicity

Both the epidemiological and animal studies provide suggestive, but limited, evidence of adverse outcomes to female reproductive outcomes. However, much more extensive evidence exists in support of an association between TCE exposures and male reproductive toxicity (U.S. EPA, 2011e).

The reasonably available human data that associate TCE with adverse effects on male reproductive function are limited in sample size and provide little quantitative dose data. However, the animal data provide strong and compelling evidence for TCE-related male reproductive toxicity. Strengths of the animal database include the presence of both functional and structural outcomes, similarities in adverse treatment-related effects observed in multiple species, and evidence that metabolism of TCE in male reproductive tract tissues is associated with adverse effects on sperm measures in both humans and animals. Additionally, some aspects of a putative mode of action (e.g., perturbations in testosterone biosynthesis) appear to have some commonalities between humans and animals (U.S. EPA, 2011e).

EPA did not identify any new repeat-dose experimental studies in animals or human epidemiological studies that would contribute significant additional hazard information for this endpoint. Therefore, EPA relied primarily on conclusions from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).

Human Data

Most human studies support an association between TCE exposure and alterations in sperm density and quality, as well as changes in sexual drive or function and serum endocrine levels. Chia et al. (1996) observed decreased normal sperm morphology along with hyperzoospermia in male workers averaging over five years occupational exposure. Fewer epidemiological studies exist linking decreased incidence of fecundability (time-to-pregnancy) and menstrual cycle disturbances in women with TCE exposures (U.S. EPA, 2011e).

Animal Data

Laboratory animal studies provide evidence for similar effects, particularly for male reproductive toxicity. These animal studies have reported effects on sperm, libido/copulatory behavior, and serum hormone levels, although some studies that assessed sperm measures did not report treatment-related alterations (U.S. EPA, 2011e). Identified key and supporting studies have observed TCE-related histopathological lesions in the testes or epididymides, altered *in vitro* sperm-oocyte binding, and increased incidence of irregular sperm in rodents (Kan et al., 2007; Xu et al., 2004; Kumar et al., 2001; Kumar et al., 2000). Forkert et al. (2002) also observed effects on the epididymis, however that study was Unacceptable in data quality evaluation. Similarly, decreased *in vitro* fertilization resulted from exposure of male rats to TCE in drinking water in one study (Duteaux et al., 2004), however that study scored a Low in data quality evaluation.

Fewer animal studies are reasonably available for the female reproductive toxicity endpoint. While *in vitro* oocyte fertilizability has been reported to be reduced as a result of TCE exposure in rats, a number of other laboratory animal studies did not report adverse effects on female reproductive function effects (U.S. EPA, 2011e). The key study Narotsky et al. (1995) observed delayed parturition in female rats. Exposure of either males or females to TCE in feed resulted in reduced successful copulation and an associated decrease in the number of live pups and litters (George et al., 1986).

3.2.3.1.6 Developmental Toxicity

An evaluation of the human and animal developmental toxicity data suggests an association between pre- and/or postnatal TCE or TCE metabolite exposures and potential developmental adverse outcomes. Heart malformations observed after developmental TCE exposure in animal studies were identified in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) as the most sensitive developmental toxicity endpoint for dose-response analysis. The developmental toxicity information is briefly described below, including information from the 2014 assessment and more recent studies.

For developmental toxicity other than congenital heart defects EPA did not identify any repeat-dose experimental studies in animals or human epidemiological studies that would contribute significant additional information for this hazard. Therefore, EPA relied primarily on conclusions from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) for these other endpoints. For congenital heart defects, EPA evaluated more recent epidemiological studies, mechanistic studies, and a single experimental animal study that provide conflicting evidence for this endpoint.

Human Data

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) evaluated numerous human studies that examined the possible association of TCE with various developmental outcomes, including prenatal (e.g., spontaneous abortion and perinatal death, decreased birth weight, and congenital malformations) and postnatal (e.g., growth, survival, developmental neurotoxicity, developmental immunotoxicity, and childhood cancers) health outcomes. Most of these were occupational epidemiology studies. In addition, geographically-based epidemiological studies have been conducted in various parts of the United States, including Arizona (Tucson Valley), Colorado (Rocky Mountain Arsenal), Massachusetts, New York (Endicott), Camp Lejeune, North Carolina and Milwaukee, Wisconsin (U.S. EPA, 2011e).

The Endicott, New York, and the Camp Lejeune studies focused on reproductive and developmental outcomes. Some of these studies have reported associations between parental exposure to TCE and spontaneous abortion or perinatal death, and decreased birth weight. However, other occupational and geographically-based studies have failed to detect a positive association between TCE exposure and developmental toxicity in humans (U.S. EPA, 2011e).

There have been some epidemiological studies that have consistently reported an increased incidence of birth defects in TCE-exposed populations. For instance, ATSDR has conducted studies at Camp Lejeune, North Carolina, where individuals were exposed to VOC-contaminated drinking water (Ruckart et al., 2014, 2013). TCE was one of the main contaminants found in the drinking water. Ruckart et al. found an association between neural tube defects and TCE exposure above 5 ppb during the first trimester of pregnancy, however either negative or null associations were identified between TCE exposure and other developmental effects (e.g., reduced birth weight, oral cleft defects). Yauck et al. (2004) observed a strong relative risk estimate for cardiac malformations in infants from Milwaukee, Wisconsin born to TCE-exposed mothers aged 38 years or older. In addition to older age, increased risk was also independently associated with other confounders including alcohol use, hypertension, and diabetes. Forand et al., (2012) (an update for the Endicott, NY community) reported significant relative risk estimates for low birth weight, small for gestational age, and cardiac defects. See the below section for further discussion of congenital heart defects.

Other studies have also identified an association between exposure to TCE exposure and

developmental effects. One study reported increased risk of spina bifida to offspring of TCE-exposed mothers (Swartz et al., 2015), and both statistically significant and non-significant associations have been observed between exposure to the TCE metabolites trichloracetic acid and trichloroethanol with various outcomes including oral clefts, urinary tract malformations, and limb defects (Cordier et al., 2012). In contrast, (Brender et al., 2014) found no statistically significant association with neural tube defects, spina bifida, anenocephaly, any oral cleft, cleft palate, cleft lip with or without cleft palate, any limb deficiency, or longitudinal or transverse limb deficiencies. The study did identify an increased risk of septal heart defects (see below section) in older mothers, however. As for human developmental neurotoxicity, the available studies collectively suggest that the developing brain is susceptible to TCE toxicity. These studies have reported an association with TCE exposure and CNS birth defects and postnatal effects such as delayed newborn reflexes, impaired learning or memory, aggressive behavior, hearing impairment, speech impairment, encephalopathy, impaired executive and motor function and attention deficit (U.S. EPA, 2011e).

Animal Data

Many of the TCE-related developmental effects reported in humans have been observed in key and supporting animal studies: increased fetal resorptions (Narotsky et al., 1995), developmental neurotoxicity (Fredriksson et al., 1993; Taylor et al., 1985), developmental immunotoxicity (Peden-Adams et al., 2006), and congenital heart defects anomalies (Johnson et al., 2003; Dawson et al., 1993). Healy et al. (1982) observed increased resorptions, skeletal abnormalities, and decreased fetal weight, but the study scored Unacceptable in data quality evaluation. Some of the observed effects appear to be strain-specific (U.S. EPA, 2011e). Among newer studies identified in the EPA literature search, increased locomotor and exploratory activities were observed following drinking water exposures to mice during nervous system development (Blossom et al., 2013), however these effects were not consistently dose-responsive.

Congenital Heart Defects

In vivo animal studies in rats and chicks have identified an association between TCE exposures and cardiac defects[17] in the developing embryo and/or fetus (U.S. EPA, 2011e). The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) identified congenital heart defects following TCE exposure via drinking water as the most sensitive human health endpoint for dose-response analysis and risk evaluation based on data from (Johnson et al., 2003) and (Dawson et al., 1993), despite public criticisms of insufficient data reporting and other issues in these studies. Mechanistic studies have also examined various aspects of the induction of cardiac malformations. Human studies have also identified statistically significant increased risk of developmental cardiac defects following TCE exposure (Brender et al., 2014; Forand et al., 2012; Goldberg et al., 1990). The critical window for cardiac development is 1-2 weeks for rodents, 1-2 weeks for chickens, and from the 3rd to the 8th week for the human fetus.

The scientific literature also has examples of relatively well-conducted studies in rats and mice that did not observe an increase in TCE-induced cardiac malformations. Most prominent among these include an inhalation study in rats (Carney et al., 2006) and an oral gavage study in rats (Fisher et al., 2001). Of note however, while (Fisher et al., 2001) did not report statistically-significant increases in combined

[17] "Cardiac" (or "heart") "defects," "malformations," and "abnormalities" are used throughout this risk evaluation to refer to adverse findings in the developing heart. These terms, in addition to "congenital heart defects" (CHD), are used in experimental animal, epidemiological, and/or clinical studies to characterize or categorize various morphological cardiovascular outcomes in the fetus or neonate. For the purpose of this risk evaluation, they are used interchangeably.

cardiac and cardiovascular effects, there was a very high background incidence of cardiovascular defects in soybean oil-control rats and the authors did observe a 19% increase in cardiac-specific defects (per-litter, significance not calculated) following TCE treatment compared to controls. During the development of this risk evaluation, a study was completed that also did not identify a statistically significant increase in cardiac defects following TCE exposure via drinking water (Charles River Laboratories, 2019). Several epidemiological studies also report either negative (Lagakos et al., 1986) or equivocal (Yauck et al., 2004; Bove et al., 1995) statistical associations between TCE exposure and heart defects. Gilboa et al. (2012) identified a statistically significant association of perimembranous ventricular septal defects with exposure to chlorinated solvents as a class, but not to TCE alone.

In previous assessments EPA concluded that the weight of evidence supports TCE exposure posing a potential hazard for congenital malformations, including cardiac defects in offspring (Makris et al., 2016; U.S. EPA, 2014b, 2011e). Given both the conflicting results and the publication of newer animal, epidemiological, and in vitro studies since the completion of the 2014 TCE Risk Evaluation, EPA re-evaluated the weight of evidence for congenital heart defects (see Section 3.2.4.1.6 and Appendix G).

3.2.3.1.7 Overt Toxicity Following Acute/Short Term Exposure

Acute studies in animals consist of single exposures at high doses specifically designed for assessing the dose at which lethality occurs or for examining overt toxicity. The interim acute exposure guideline levels (AEGLs) document for TCE was consulted and used in this assessment to briefly summarize the acute toxicity data (NAC/AEGL, 2009).

In humans, TCE odors can be detected at concentrations of ≥50 ppm. It was once commonly used as an anesthetic agent with concentrations ranging from 5,000 to 15,000 ppm for light anesthetic use and from 3,500 to 5,000 ppm for use as an analgesic. Information on the toxicity of TCE in humans comes from either case reports in the medical/occupational literature or experimental human inhalation studies. Lethality data in humans have been reported following accidental exposure to TCE. However, there is insufficient information about the exposure characterization of these incidents (NAC/AEGL, 2009).

Human inhalation studies have shown that acute exposure to TCE results in irritation and central nervous system (CNS) effects in humans. Mild subjective symptoms and nose and throat irritation were reported by human volunteers exposed to 200 ppm TCE for 7 hrs/day on the first day of exposure during a 5-day exposure regimen. The study also reported minimal CNS depression following TCE exposure (NAC/AEGL, 2009). Laboratory studies have additionally demonstrated acute effects of TCE on the respiratory tract in the form of both localized irritation and broad fibrosis, likely dependent on oxidative metabolism. (U.S. EPA, 2011e).

CNS depression and effects on neurobehavioral functions were seen in human volunteers exposed to 1,000 ppm TCE for a 2-hr period. In the same studies, volunteers were also exposed to 100 or 300 ppm TCE for 2 hrs. Some subjects had similar CNS effects at the middle concentration (300 ppm), with no such effects observed at the 100 ppm. A different study reported slight to marginal neurobehavioral effects after exposure to 300 ppm TCE for 2.5 hrs. Cardiac arrhythmias have also been reported in humans exposed to high concentration of TCE. Several animal studies have reported neurobehavioral effects and the potential for inducing cardiac sensitization following acute inhalation exposure to TCE (NAC/AEGL, 2009).

The NIOSH Skin Notation Profile for TCE (Hudson and Dotson, 2017) summarizes data providing evidence for skin irritation and/or corrosion from dermal TCE exposure, with effects including rashes,

blistering, and burning sensations. Eye effects and CNS effects also resulted following simultaneous vapor inhalation along with percutaneous penetration. Skin irritation potential varied greatly among individuals in volunteer studies, with some exhibiting extreme pain and others hardly reporting any effects. Studies on both humans and animals demonstrate that TCE is a moderate skin sensitizer, with hypersensitivity reactions observed following exposure to both TCE and various metabolites.

3.2.3.2 Genotoxicity and Cancer Hazards

3.2.3.2.1 Kidney cancer

The TCE IRIS assessment concluded that TCE is "carcinogenic to humans" based on convincing evidence of a causal relationship between TCE exposure in humans and kidney cancer. A review of TCE by the International Agency for Research on Cancer (IARC) also supported this conclusion (IARC, 2014). The carcinogenic classification was based on a review of more than 30 human studies, including studies in TCE degreasing operations, and meta-analyses of the cohort and case- control studies. Relative risk estimates for increased kidney cancer were consistent across a large number of epidemiological studies of different designs and populations from different countries and industries (Appendix C,(U.S. EPA, 2011b). This strong consistency of the epidemiologic data on TCE and kidney cancer argues against chance, bias, and confounding as explanations for the elevated kidney cancer risks (U.S. EPA, 2011e).

Cancer bioassays with TCE in animals (i.e., both gavage and inhalation exposure routes) did not show increased kidney tumors in mice, hamsters, or female rats, but did show a slight increase in male rats. Kidney tumors in rats are relatively rare (U.S. EPA, 2011e).

The toxicokinetic data and the genotoxicity of DCVC further suggest that a mutagenic mode of action is involved in TCE-induced kidney tumors, although cytotoxicity followed by compensatory cellular proliferation cannot be ruled out. As for the mutagenic mode of action, both genetic polymorphisms (GST pathway) and mutations to tumor suppressor genes have been hypothesized as possible mechanistic key events in the formation of kidney cancers in humans (U.S. EPA, 2011e).

3.2.3.2.2 Liver cancer

U.S. EPA concluded that TCE exposure causes liver tumors in mice but not rats and the meta-analysis of human data on liver and gallbladder/biliary passages indicated "*…a small, statistically significant increase in risk*". Multiple TCE metabolites (i.e., and thus pathways) likely contribute to TCE-induced liver tumors (U.S. EPA, 2011e).

Previous meta-analyses of the cohort, case-control, and community (geographic) studies reporting liver and biliary tract cancer, primary liver cancer, and gallbladder and extra-hepatic bile duct cancer (see Appendix C in (U.S. EPA, 2011b)) reported a small, statistically significant summary relative risk (RRm, overall RR from meta-analysis) for liver and gallbladder/biliary cancer with overall TCE exposure. However, the meta-analyses reported a lower, nonstatistically significant RRm for primary liver cancer when using the highest exposure groups (U.S. EPA, 2011b).

With respect to liver carcinogenicity, TCE and its oxidative metabolites TCA, DCA, and CH are clearly carcinogenic in mice, with strain and sex differences in potency. Data in other laboratory animal species are limited; thus, except for DCA which is carcinogenic in rats, inadequate evidence exists to evaluate the hepatocarcinogenicity of TCE and its metabolites in rats or hamsters (U.S. EPA, 2011e).

3.2.3.2.3 Cancer of the immune system

Human studies have reported cancers of the immune system resulting from TCE exposure. Lymphoid tissue neoplasms arise in the immune system and result from events that occur within immature lymphoid cells in the bone marrow or peripheral blood (leukemias), or more mature cells in the peripheral organs (non-Hodgkin's lymphoma). The broad category of lymphomas can be divided into specific types of cancers, including non-Hodgkin's lymphoma, Hodgkin lymphoma, multiple myeloma, and various types of leukemia (e.g., acute and chronic forms of lymphoblastic and myeloid leukemia). Leukemia during childhood has been observed in a number of studies in children exposed to TCE, however this association has not been confirmed (U.S. EPA, 2011e).

One of the three cancers for which the TCE IRIS assessment based its cancer findings was non-Hodgkin's lymphoma (NHL) (the other two being kidney and liver cancer) (U.S. EPA, 2011e). The human epidemiological database identifies a statistically significant association between TCE exposure and NHL (Appendix C, (U.S. EPA, 2011b). Further support comes from animal studies reporting rates of lymphomas and/or leukemias following TCE exposure (U.S. EPA, 2011e).

3.2.3.2.4 Other cancers

Reproductive System

The effects of TCE on cancers of the reproductive system have been examined for males and females in both epidemiological and experimental animal studies. The epidemiological literature includes data on prostate in males and cancers of the breast and cervix in females. The experimental animal literature includes data on prostate and testes in male rodents; and uterus, ovary, mammary gland, vulva, and genital tract in female rodents. The evidence for these cancers is generally not robust (U.S. EPA, 2011e).

Other cancers

There is limited evidence of increased risk for esophageal cancer following TCE exposure in males only. The reasonably available evidence is not statistically sensitive enough for informing quantitative evaluations of esophageal cancer risk from TCE. There is some evidence of association for bladder or urothelial cancer and high cumulative TCE exposure, however the reasonably available studies examine multiple sites and do not completely account for potential confounding factors. In several studies examining the relationship between TCE exposure and cancer of the brain or central nervous system (CNS), the data does not provide strong evidence in either direction, although there is some association of TCE exposure with CNS cancers in children (U.S. EPA, 2011e).

3.2.4 Weight of Scientific Evidence

3.2.4.1 Non-Cancer Hazards

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of scientific evidence (WOE) conclusions for all non-cancer endpoints other than congenital heart defects. For the previous WOE evaluations of all other endpoints, see the 2011 EPA IRIS Assessment (U.S. EPA, 2011e) and the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b).

3.2.4.1.1 Liver toxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) for this hazard.

Animal data demonstrating increased liver weight, cytotoxicity, hypertrophy, and peroxisome proliferation is supported by human data demonstrating changes in plasma or bile acid liver enzyme levels and hypersensitivity-induced liver damage. Overall, liver toxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis.

3.2.4.1.2 Kidney toxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) for this hazard.

The kidney is one of the more sensitive targets of TCE, with toxicity resulting from conjugative metabolites such as DCVC. Both animal and human studies consistently observe induction of kidney toxicity (e.g., damage to renal tubules and nephropathy) and progression of existing kidney disease. Overall, kidney toxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis.

3.2.4.1.3 Neurotoxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) for this hazard.

In addition to anesthetic effects at high concentrations, human evidence concludes that TCE exposure induces abnormalities in trigeminal nerve function, and TCE exposure has also been associated with neurodegenerative disorders. These effects have been confirmed in animal studies which additionally demonstrate a variety of neurological effects from TCE exposure. Overall, neurotoxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis.

3.2.4.1.4 Immunotoxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) for this hazard.

Both animal and human studies demonstrate that TCE exposure can result in either autoimmune responses or immunosuppression. There is also evidence of both systemic and localized hypersensitivity resulting in skin sensitization and autoimmune hepatitis. Selgrade et al (2010) demonstrated reduced response to respiratory infection. There are no other reasonably available studies that examined respiratory immunotoxicity, however this endpoint is consistent with other data on immunosuppression. Overall, immunotoxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis, including both systemic and respiratory endpoints.

There is only qualitative information available for sensitization and hypersensitivity, so this hazard was not carried forward for dose-response analysis.

3.2.4.1.5 Reproductive toxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) for this hazard.

Both human and animal data provide strong evidence for male reproductive effects from TCE. Effects observed include effects on sperm, male reproductive organs, hormone levels, and sexual behavior. There is insufficient evidence for determining whether TCE contributes to female reproductive toxicity.

Overall, male reproductive toxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis.

3.2.4.1.6 Developmental Toxicity

The EPA literature search (U.S. EPA, 2017i) did not identify any new evidence that significantly contributes to or challenges the previously established weight of evidence (WOE) conclusions for this hazard other than for congenital heart defects.

There is substantial evidence from both animal and human studies that TCE exposure is associated with various developmental outcomes, ranging from decreased birth weight to pre- and postnatal mortality. Other hazards also present following developmental exposure, including developmental immunotoxicity and developmental neurotoxicity. While the epidemiological literature does not consistently observe developmental effects, effects that have been observed in multiple human studies have been corroborated by animal data.

Overall, based on suggestive epidemiologic data and fairly consistent laboratory animal data, developmental toxicity following TCE exposure is supported by the weight of evidence. Therefore, this hazard was carried forward for dose-response analysis.

Developmental toxicity endpoints will be considered for both acute and chronic scenarios. Although developmental studies typically involve multiple exposures, they are considered relevant for evaluating single exposures because evidence indicates that certain developmental effects may result from a single exposure during a critical window of development (Davis et al., 2009; Van Raaij et al., 2003). This is consistent with EPA's *Guidelines for Reproductive Toxicity Risk Assessment* (U.S. EPA, 1996) and *Guidelines for Developmental Toxicity Risk Assessment* (U.S. EPA, 1991), which state that repeated exposure is not a necessary prerequisite for the manifestation of developmental toxicity. This is a health protective assumption.

Congenital Heart Defects

The congenital heart defects endpoint for TCE has been widely discussed since the release of the 2011 IRIS Assessment (U.S. EPA, 2011e). The primary basis for this endpoint was a developmental drinking water study in rats, (Johnson et al., 2003), that has been the source of extensive controversy. The study administered 0 ppb, 2.5 ppb, 250 ppb, 1.5 ppm, and 1100 ppm to pregnant Sprague-Dawley rats via drinking water for the entire duration of pregnancy. On the last day of pregnancy, dams were euthanized, and the heart and great vessels of fetuses were examined for abnormalities. The study reported statistically significant increases in variety of cardiac defects at multiple dose levels in the incidence of a broad array of cardiac defects. EPA considered the constellation of observed effects in totality, as opposed to any particular individual defects.

The authors reported (Johnson et al., 2005) that the study data were derived from a 6-year academic research program and consolidated data from several cohorts. Control data were combined from 6 independent cohort experiments; the data from the highest two TCE doses had been previously published by the laboratory (Dawson et al., 1993). Although study methods were generally consistent throughout the research program, there are potential concerns of genetic drift due to the TCE dose groups being administered up to 6 years apart, and the control vehicle used in the Dawson et al., 1993 study was filtered tap water while distilled water was used in all subsequent study cohorts. Both (Dawson et al., 1993) and (Johnson et al., 2003) were deficient in adequate reporting of methods and

raw scoring data; however, many of those concerns have been alleviated by subsequent communications to EPA (Johnson, 2014, 2008). The positive findings reported in (Dawson et al., 1993) and (Johnson et al., 2003) have not been confirmed by another laboratory, so controversy over the results remains. When considering the totality of information provided (not only what was in the initial publications), both (Dawson et al., 1993) and (Johnson et al., 2003) received a Medium in data quality evaluation.

EPA previously published weight of evidence (WOE) analyses both as part of the 2014 TCE Risk Assessment and as a peer-reviewed journal article (Makris et al., 2016), which concluded that the totality of data does support congenital heart defects as a human health hazard for TCE. These WOE analyses utilized modified Bradford-Hill criteria (Hill, 1965) to evaluate the overall evidence for causality following study quality review. Recently, (Wikoff et al., 2018) published a WOE analysis focusing only on animal and epidemiological data that came to the opposite conclusion using a Risk of Bias assessment for internal study validity. During the development of this risk evaluation, EPA received a study sponsored by the Halogenated Solvents Industry Alliance (HSIA) (Charles River Laboratories, 2019) that attempted to replicate the (Johnson et al., 2003) study, examining the incidence of developmental cardiac defects following administration of TCE to rats via drinking water. This study was subsequently peer reviewed and published in the scientific literature.

Charles River Study

Charles River Laboratories (2019) performed a developmental toxicity study according to principles of Good Laboratory Practice. The study authors administered TCE to pregnant Sprague-Dawley rats via drinking water at concentrations of 0 ppm, 0.25 ppm, 1.5 ppm, 500 ppm, and 1000 ppm in reverse osmosis-filtered water from gestation day 1 through 21. Retinoic acid (RA) served as the positive control and was administered via gavage (3mg/ml, 5mg/kg-bw) on gestation days 6-15. The study authors did not observe a statistically significant increase of interventricular septal defects in TCE-treated fetuses (2.4% in negative control, 3.7% at highest dose) or any other types of cardiac defects identified in the study.

While the results of the Charles River study (2019) results appear to contradict the results observed by (Johnson et al., 2003) and (Dawson et al., 1993), EPA concludes that the Charles River study methodology was likely of reduced sensitivity and therefore does not entirely replicate the study conditions of those earlier studies. In short, the methodology and positive control data indicate that the Charles River study (2019) was primarily focused on ventricular septal defects (VSDs) and therefore did not sufficiently examine the complete range of potential cardiac defects. The Johnson study (2003) specifically described assessment of valves and observed both valve and atrial septal defects using their laboratory dissection and examination methodology. In contrast, while the Stuckhardt and Poppe dissection method (1984) used by the Charles River study should allow visualization of valves, the Charles River study did not report valve defects in any TCE group or the RA positive control group even though many other published reports have identified valve defects following administration of TCE or RA. Additionally, the Stuckhardt and Poppe method (1984) does not include examination of the heart for atrial septal defects, and the Charles River study did not report any atrial septal defects in either the RA positive control group or the TCE groups. In fact, the Charles River study (2019) observed a similar percentage of VSDs as (Johnson et al., 2003). Considering total VSDs, 3.5% of fetuses showed a VSD in Charles River vs 3.8% in Johnson at the highest dose, with 1.5% in Charles River vs 2.2% in Johnson at 1.5ppm. When considering only membranous VSDs (the only type observed in the Charles River study), observed incidences were actually higher in Charles River at the highest dose (3.5% vs 2.86%). Meanwhile, a substantial percentage of the total cardiac defects observed in (Johnson et al., 2003) were valvular or atrial.

As further indication of the potentially limited sensitivity of (Charles River Laboratories, 2019), the defects observed from exposure to the retinoic acid (RA) positive control were also somewhat limited compared to the broader RA literature (which did identify atrial septal defects). Additionally, the other oral TCE study (Fisher et al., 2001), which did not identify a statistically significant increase in cardiac defects following TCE administration at a high dose via gavage, identified a significant number of additional defects that match those identified in (Johnson et al., 2003) and (Dawson et al., 1993) (including atrial septal and valve defects). Therefore, (Charles River Laboratories, 2019) insufficiently replicates the methodology of (Johnson et al., 2003), and the results do not entirely contradict the conclusions of that study. Based on these considerations along with some data reporting errors, (Charles River Laboratories, 2019) received a Medium in data quality evaluation, the same as (Dawson et al., 1993) and (Johnson et al., 2003). For a more detailed analysis of the (Charles River Laboratories, 2019) study, see Appendix G.1.

While (Charles River Laboratories, 2019) was not considered a close enough replication to (Johnson et al., 2003) to sway the weight of evidence for the endpoint on it's own, EPA did consider (Charles River Laboratories, 2019) to be an overall well-conducted study, and it was incorporated into the WOE analysis for the cardiac defects endpoint along with all other relevant studies identified in the literature.

WOE Analysis

In order to address the conflicting results of the previous WOE assessments (U.S. EPA, 2014b; Makris et al., 2016; Wikoff et al., 2018), in support of this risk evaluation EPA performed another WOE analysis. This analysis included all relevant primary literature cited in (Makris et al., 2016), the 2014 TCE Risk Assessment (U.S. EPA, 2014b), and any additional on-topic studies identified in the systematic review literature search (U.S. EPA, 2017i). Additionally, EPA also incorporated any newer studies published after the end date of the literature search, including an *in vitro* mechanistic study (Harris et al., 2018) and the recently completed *in vivo* drinking water study (Charles River Laboratories, 2019), comprising 45 studies in total (42 scoring Acceptable). After reviewing a sampling of recent literature on systematic approaches to performing weight-of-evidence evaluation, EPA adopted the methodology described in [*Weight of Evidence in Ecological Assessment. Risk Assessment Forum. EPA/100/R16/00.* (U.S. EPA, 2016i)], which advocates presenting evidence on a semiqualitative scale on the basis of three evidence areas: reliability, outcome/strength, and relevance (see Appendix G.2.1 for more details on selection of approach and methodological details).

In short, the overall grade for each study was defined by the lowest-amplitude score of each evidence area, and those overall study grades were integrated to select a representative overall summary score for each line of evidence (epidemiological, *in vivo*, or mechanistic). Independently, the area scores of each study were averaged to obtain integrated areas scores for each line of evidence, however these were not used to determine the overall summary score. Functionally, this scoring methodology is similar to that used by (Wikoff et al., 2018), although that analysis focused on data quality reliability through a risk of bias assessment. Importantly, (Wikoff et al., 2018) did not evaluate any mechanistic data, which may explain the different overall conclusions between that study and this analysis. Importantly, this WOE assessment also incorporated data on TCE metabolites, which are believed to be the toxicologically active agent for many of the observed cardiac effects as well as other developmental outcomes.

The overall weight-of-evidence for TCE-induced congenital cardiac defects is presented in Table 3-6. Epidemiological, toxicological and mechanistic studies were available. The epidemiology studies as a group provide suggestive evidence for an effect of TCE on cardiac defects in humans (summary score of +). Oral *in vivo* studies provided ambiguous to weakly positive (0/+) results for TCE itself, but positive results for its TCA and DCA metabolites (+), while inhalation studies contributed negative evidence (-).

Overall, the *in vivo* animal toxicity studies provided mixed, ambiguous evidence for an effect of TCE (summary score of 0). Mechanistic studies provided strong and consistent supporting information for effects of TCE and metabolites on cardiac development and precursor effects (summary score of ++).

The database overall was determined to be both reliable and relevant. Integration of the three evidence areas resulted in an overall summary score of (+), demonstrating positive overall evidence that TCE may produce cardiac defects in humans (based on positive evidence from epidemiology studies, mixed evidence from animal toxicity studies, and stronger positive evidence from mechanistic studies).

See Appendix G.2 for the complete WOE narrative and methodology. The complete scoring table and detailed evaluation of all studies is presented in *[Data Table for Congenital Heart Defects Weight of Evidence Analysis. Docket: EPA-HQ-OPPT-2019-0500]*.

Table 3-6. Overall Summary Scores by Line of Evidence for Cardiac Defects from TCE

Evidence Area	Summary Score
Epidemiology studies	+
In vivo animal toxicity studies	0
Mechanistic studies	++
Overall	+

The differences in observed responses across studies may be partially attributed to experimental design differences. These differential responses may also represent varying susceptibility among mammalian species, strains, and populations. It is possible that animals showing a greater incidence of defects following TCE exposure represent an especially susceptible population, and genetic drift may preclude a true replication of previous study conditions (Makris et al., 2016).

Mode of Action

A number of studies have been conducted to elucidate the mode of action for TCE-related cardiac teratogenicity. During early cardiac morphogenesis, outflow tract and atrioventricular endothelial cells differentiate into mesenchymal cells. These mesenchymal cells have characteristics of smooth muscle-like myofibroblasts and form endocardial cushion tissue, which is the primordia of septa and valves in the adult heart. Many of the cardiac defects observed in humans and laboratory species involved septal and valvular structures. Thus, a major research area has focused on the disruptions in cardiac valve formation in avian *in ovo* and *in vitro* studies following TCE treatment. These mechanistic studies have revealed TCE's ability to alter the endothelial cushion development, which could be a possible mode of action underlying the cardiac defects involving septal and valvular morphogenesis in rodents and chickens. Other modes of actions may also be involved in the induction of cardiac malformation following TCE exposure. For example, studies have reported TCE-related alterations in cellular Ca^{2+} fluxes during cardiac development (Caldwell et al., 2008; Selmin et al., 2008; Collier et al., 2003). Of note, early stages of cardiac development are quite similar across various species (Makris et al., 2016). Therefore, these mechanistic data provide support to the plausibility of TCE-related cardiac effects in humans (U.S. EPA, 2011e). EPA also notes that teratogens may function through a multitude of pathways, often resulting in a constellation of effects. Therefore, evidence of a single dominant MOA is not required in order for the data to support a plausible mechanism of TCE-induced congenital heart defects.

Several *in vitro* studies have observed non-monotonic dose responses in gene activation and other molecular changes following TCE exposure at varying concentrations (Palbykin et al., 2011; Makwana et al., 2010). Specifically, TCE exposure induced expression of oxidative stress genes (Makwana et al., 2010) and increased DNA hypermethylation of a calcium-ATP pump promoter in developing cardiac tissue (Palbykin et al., 2011) only at lower and not higher doses, resulting in multimodal calcium responses (Caldwell et al., 2008). TCE also increased significantly increased gene expression of the oxidative metabolism enzyme CYP2H1 specifically in cardiac tissue only at the lower dose ((Makwana et al., 2013)). In (Harris et al., 2018), expression of genes involved in cardiac development and metabolism were either reduced (low dose) or increased (high dose), depending on the administered concentration. These results may explain the non-monotonic polynomial dose-response observed in (Johnson et al., 2003), whereby toxicological outcomes present at different doses equating to either inhibition or activation of particular gene expression (Harris et al., 2018). This differential gene expression would in turn lead to dose-specific downstream metabolic and phenotypic effects.

Overall, an association between increased congenital cardiac defects and TCE exposure is supported by the weight of evidence, in agreement with previous EPA analyses (U.S. EPA, 2014b; Makris et al., 2016). Therefore, this endpoint was carried forward for dose-response analysis.

3.2.4.1.7 Overt Toxicity Following Acute/Short Term Exposure

There is strong evidence for overt toxicity in humans following acute exposure to high concentrations of TCE. AEGL guidelines indicate the concentrations at which increasing levels of toxicity are established following acute inhalation exposure to TCE. High concentrations of TCE have been shown to result in respiratory and dermal irritation, CNS depression, cardiac arrhythmia, and even death.

While overt toxicity following acute or short term exposure to TCE is supported by the weight of evidence, studies examining the acute outcomes described above were not selected for assessing acute risks due to a lack of sufficient dose-response information. EPA considered more sensitive endpoints for estimation of risks following acute TCE exposure, namely all developmental toxicity endpoints and reduced response to respiratory infection (Selgrade and Gilmour, 2010). Other acute studies described above were not selected for assessing acute risks due to a lack of sufficient dose-response information.

3.2.4.2 Cancer Hazards

Meta-analyses were performed in the 2011 EPA TCE IRIS Assessment (Appendix C, (U.S. EPA, 2011b)) in order to statistically evaluate the epidemiological data for NHL, kidney cancer, and liver cancer. The IRIS Assessment also investigated the association of TCE with lung cancer, primarily as a means to examine smoking as a potential confounder for the kidney cancer studies (Appendix C, (U.S. EPA, 2011b)). In that assessment EPA identified a statistically significant association between TCE exposure and NHL, kidney cancer, and liver cancer. An association was not identified for lung cancer, suggesting that there was no confounding from smoking. That assessment concluded that TCE is carcinogenic to humans by all routes of exposures, most strongly supported by the data on kidney cancer. The consistency of increased kidney cancer relative risk (RR) estimates across a large number of independent studies of different designs and populations from different countries and industries provided compelling evidence given the difficulty, a priori, in detecting effects in epidemiologic studies when the RRs were modest and the cancers were relatively rare, indicating that individual studies had limited statistical power. This strong consistency of the epidemiologic data on TCE and kidney cancer argued against chance, bias, and confounding as explanations for the elevated kidney cancer risks.

The IRIS Toxicological Review of TCE (U.S. EPA, 2011e) also cited other lines of supporting evidence for TCE carcinogenicity in humans by all routes of exposure:

"First, multiple chronic bioassays in rats and mice have reported increased incidences of tumors with TCE treatment via inhalation and gavage, including tumors in the kidney, liver, and lymphoid tissues − target tissues of TCE carcinogenicity also seen in epidemiological studies."

"A second line of supporting evidence for TCE carcinogenicity in humans consists of toxicokinetic data indicating that TCE is well absorbed by all routes of exposure, and that TCE absorption, distribution, metabolism, and excretion are qualitatively similar in humans and rodents."

"Finally, available mechanistic data do not suggest a lack of human carcinogenic hazard from TCE exposure."

A statistically significant association was not identified for lung cancer and it was not considered as contributing to the overall oral slope factor or inhalation unit risk. However, the results of the lung cancer meta-analysis were interpreted to minimize any concern for confounding effects of smoking on the other cancers.

For this risk evaluation, EPA performed new meta-analyses incorporating both the initial group of studies assessed in the 2011 EPA TCE IRIS Assessment and any newer, on-topic studies of Acceptable data quality identified in the literature search performed according to the *Application of Systematic Review in TSCA Risk Evaluations* (U.S. EPA, 2018b). EPA utilized similar methodology as was employed in the 2011 EPA TCE IRIS Assessment (U.S. EPA, 2011e) while also incorporating consideration of data quality evaluation as described in (U.S. EPA, 2018b). Additionally, EPA included sensitivity analyses as needed to partition the results based on both heterogeneity and data quality score. When more than one report was available for a single study population, only the most recent publication or the publication reporting the most informative data for TCE was selected for inclusion in the meta-analysis. While the updated meta-analysis builds off of (U.S. EPA, 2011b), the results presented below represent a standalone, new analysis. See Appendix H for full details and results.

3.2.4.2.1 Meta-Analysis Results

The initial results of meta-analyses for NHL, kidney cancer and liver cancer showed moderate heterogeneity among studies, due largely to the influence of the study by Vlaanderen et al. (2013). Random-effects models are consequently preferred to fixed-effects models due to the degree of heterogeneity. These reduced the influence of the (Vlaanderen et al., 2013) study and demonstrated stronger positive associations (greater meta-RR value) of all cancers with exposure to TCE, although the liver cancer meta-RR was not significant. The evidence for an association between TCE exposure and NHL was further strengthened by a subsequent meta-analysis on studies reporting cohorts categorized as experiencing "high" exposure to TCE, which demonstrated a greater meta-RR compared to "any" exposure.

The study of Vlaanderen et al. (2013) carries very large statistical weight due to its large sample size, but its sensitivity to detect any true effect of TCE is likely to be low. The study is based on a large general population cohort with exposures estimated by linking job titles recorded in national census data to a job-exposure matrix. The prevalence and average intensity of TCE exposure are low in the study population and the indirect method of estimating exposures has significant potential to misclassify exposure. Further, the study was not scored High for data quality in EPA's review (it scored Medium). There was therefore reason to believe that omitting the Vlaanderen et al.(2013) study would improve the sensitivity of meta-analytic results for all three cancers. In sensitivity analyses omitting the study of (Vlaanderen et al., 2013), between-study heterogeneity was significantly reduced or eliminated.

Resulting meta-RRs for exposure to TCE were strengthened and were statistically significant for all three cancers.

Analyses stratified by a data quality score also indicated stronger associations of all cancers with TCE exposure in studies that scored High for data quality compared to studies that scored Medium or Low; notably, the latter group included the influential study of (Vlaanderen et al., 2013). Studies that scored high showed no heterogeneity of effects for NHL and kidney cancer, but moderate heterogeneity remained for liver cancer.

In summary, meta-analyses accounting for between-study heterogeneity, influential observations, and data quality consistently indicate positive associations of NHL, kidney cancer and liver cancer with exposure to TCE. This conclusion generally agrees with that of other governmental and international organizations. The International Agency for Research on Cancer (IARC) (IARC, 2014) found sufficient evidence for the carcinogenicity of TCE in humans. IARC definitively stated that TCE causes kidney cancer and determined that a positive associated has been identified for NHL and liver cancer. Based on the weight of evidence when accounting for both these authoritiative assessments and the results of EPA's meta-analyses, cancer was carried forward for dose-response analysis, incorporating extra cancer risk from all three cancer types.

3.2.4.2.2 Mode of Action

Kidney Cancer

Genotoxicity

The predominant mode of action (MOA) for kidney carcinogenicity involves a genotoxic mechanism through formation of reactive GSH metabolites (e.g., DCVC, DCVG). This MOA is well-supported, as toxicokinetic data indicates that these metabolites are present in both human blood and urine, and these metabolites have been shown to be genotoxic both *in vitro* and in animal studies demonstrating kidney-specific genotoxicity (U.S. EPA, 2011e).

Cytotoxicity and other mechanisms

Observed nephrotoxicity in both human and animal studies, especially at elevated concentrations, provides some evidence of a cytotoxic MOA. Data comparing relative dose-response analysis of nephrotoxicity and kidney cancer incidence suggests that cytotoxicity can occur at doses below those causing carcinogenicity in animal bioassays, however this data also indicates that nephrotoxicity is not sufficient or rate-limiting for renal carcinogenesis. Therefore, a causal or predictive link between cytotoxicity and carcinogenicity cannot be established. There is inadequate experimental support for other potential MOAs such as peroxisome proliferator activated receptor alpha (PPARα) induction, α2μ-globulin nephropathy, and formic acid-related nephrotoxicity (U.S. EPA, 2011e).

Conclusion

There is clear evidence of a genotoxic MOA for kidney cancer, either on its own or in combination with other mechanisms. While the kidney is highly sensitive to TCE-induced cytotoxicity, the contribution of cytotoxicity toward kidney carcinogenesis cannot be determined. Renal cytotoxicity may instead serve as a promoter step in tumorigenesis following genotoxic initiation, or it may merely represent an independent pathway of toxicity (U.S. EPA, 2011e).

Liver Cancer

Genotoxicity

The strongest data supporting mutagenic potential of TCE or potential liver metabolites comes from data on the intermediate metabolite chloral hydrate (CH), which induces a variety of genotoxic effects both *in*

vitro and *in vivo*. The peak *in vivo* concentrations of CH in tissue are substantially less than is required for induction of genotoxicity in many *in vitro* assays, however there is some evidence of *in vivo* genotoxicity at doses comparable to those inducing cancer in chronic bioassays. Overall, the data are insufficient to conclude that a mutagenic MOA is operating, however it cannot be ruled out. (U.S. EPA, 2011e).

PPARα receptor activation

While strong evidence exists for TCA-mediated PPARα receptor activation (resulting in downstream perturbation of cell apoptosis and proliferation signaling) based on observed peroxisome proliferation and increased marker activity in rodents treated with TCE, TCA, or DCA, this appears to occur at a higher dose than what induces liver tumors in mice. TCE, TCA, and DCA have been found to be weak peroxisome proliferators, and some data suggests that PPARα activation may not be sufficient for carcinogenesis. The reasonably available data clearly supports a role of PPARα activation in liver tumorigenesis, however any key causal effects are likely mediated by multiple mechanisms and neither causality, sufficiency, or necessity of PPARα signaling in liver carcinogenicity can be established (U.S. EPA, 2011e).

Other mechanisms

There is limited evidence for a tumorigenic role of increased liver weight, growth selection, cytotoxicity, oxidative stress, and/or glycogen accumulation. Heritable epigenetic changes such as altered DNA methylation patterns, which disrupt the balance of gene expression and may lead to over- or under-expression of various tumor suppressors and promoters, have been associated with liver cancer and other tumors in general. Additionally, TCE has been shown to promote hypomethylation (resulting in increased gene expression) *in vivo* and *ex vivo* in liver tissue. DNA hypomethylation can be sufficient for liver carcinogenesis based on choline/methionine deficiency studies, however the applicability of this mechanism to TCE-induced carcinogenesis is unknown as these changes could either be causally or consequentially related to carcinogenicity (U.S. EPA, 2011e).

Conclusions

The reasonably available data is inadequate to support any singular MOA. TCE-induced liver carcinogenesis appears to be very complex and likely involves multiple contributing mechanisms. The strongest evidence exists for involvement of both genotoxicity and PPARα activation, however a causal relationship cannot be established because the dose levels required to elicit outcomes through both MOAs are higher than those demonstrating tumorigenic activity (U.S. EPA, 2011e).

Non-Hodgkin Lymphoma

There is insufficient data reasonably available for suggesting any particular MOA for NHL.

Overall Conclusions

TCE is carcinogenic by a genotoxic mode of action at least for kidney cancer, while a predominant mode of action cannot be determined for the other tumor types. Per EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), overall, the totality of the reasonably available data/information and the WOE analysis for the cancer endpoint was sufficient to support a linear non-threshold model. The application of a linear non-threshold model is justified based on the genotoxic MOA for kidney cancer, the combined relative contributions of multiple tumor types, and the positive associations observed via meta-analysis for all three cancers in epidemiological studies based on low-level, environmental exposure levels (as opposed to relying on extrapolation from high doses in a rodent bioassay).

3.2.5 Dose-Response Assessment

3.2.5.1 Selection of Studies for Dose-Response Assessment

The EPA evaluated data from studies described above (Section 3.2.3.1) to characterize the dose-response relationships of TCE and selected studies and endpoints to quantify risks for specific exposure scenarios. One of the additional considerations was that the selected key studies had adequate information to perform dose-response analysis for the selected PODs. The EPA defines a POD as the dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound in the dose for an estimated incidence, or a change in response level from a dose-response model (i.e., BMD), a NOAEL or a LOAEL for an observed incidence or change in the level of response.

Based on the weight of the evidence evaluation, six health effect domains were selected for non-cancer dose-response analysis: (1) liver; (2) kidney; (3) neurological; (4) immunological; (5) reproductive; and (6) developmental. Additionally, dose-response analysis was performed for cancer based on observed incidences of kidney cancer, liver cancer, and non-Hodgkin lymphoma. These hazards have been carried forward for dose-response analysis. While there is also evidence to support overt toxicity following acute exposure, endpoints for these effects were not carried forward for dose-response analysis. For a complete discussion, see Section 3.2.4.1.

Studies that evaluated each of the health effect domains were identified in Section 3.2.3, and are considered in this section for dose-response analysis. In order to identify studies for dose-response analysis, several attributes of the studies were reviewed. Preference was given to studies using designs reasonably expected to detect a dose-related response. Chronic or subchronic studies are generally preferred over studies of less-than-subchronic duration for deriving chronic and subchronic reference values. Studies with a broad exposure range and multiple exposure levels are preferred to the extent that they can provide information about the shape of the exposure-response relationship. Additionally, with respect to measurement of the endpoint, studies that can reliably measure the magnitude and/or degree of severity of the effect are preferred.

Experimental animal studies considered for each hazard and effect were evaluated using systematic review quality considerations discussed in the Systematic Review Methods section. Only studies that scored an acceptable rating in data evaluation were considered for use in dose-response assessment. In addition to the data quality score, considerations for choosing from among these studies included study duration, relevance of study design, and the strength of the toxicological response. Details on these considerations for each endpoint are provided below.

Given the different TCE exposures scenarios considered (both acute and chronic), different endpoints were used based on the expected exposure durations. For non-cancer effects and based on a weight-of-evidence analysis of toxicity studies from rats, risks for developmental effects that may result from a single exposure were considered for both acute (short-term) and chronic (long-term, continuous) exposures, whereas risks for other adverse effects (e.g., liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity, and reproductive toxicity) were only considered for repeated (chronic) exposures to TCE. Although developmental studies typically involve multiple exposures, they are considered relevant for evaluating single exposures because evidence indicates that certain developmental effects may result from a single exposure during a critical window of development (Davis et al., 2009; Van Raaij et al., 2003; U.S. EPA, 1991). This is consistent with EPA's Guidelines for Reproductive Toxicity Risk Assessment (U.S. EPA, 1996) which state that repeated exposure is not a necessary prerequisite for the manifestation of developmental toxicity. Consequently, in this risk evaluation EPA accepted the Agency's default assumption and concluded that developmental endpoints are applicable when assessing

acute exposures, where it is assumed that the risk of their occurrence depends on the timing and magnitude of exposure. This is a health protective approach and assumes that a single acute exposure could lead to the same effects if that exposure occurs during a critical window within the pregnancy term. A single acute study examining pulmonary immunotoxicity following 3h TCE inhalation exposure (Selgrade and Gilmour, 2010) was also considered for acute exposure scenarios. Overt toxicity studies (Section 3.2.3.1.7) were not used for the acute POD because they were often only single-dose studies and the doses at which acute toxic effects or lethality were observed were significantly higher than those that caused toxic effects in developmental studies.

3.2.5.1.1 Liver toxicity

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) determined that the studies of (Woolhiser et al., 2006; Buben and O'Flaherty, 1985; Kjellstrand et al., 1983) were suitable for the dose-response assessment of the liver health effects domain. These three studies reported dose-responsive increases in liver/body weight ratios. (Buben and O'Flaherty, 1985) and (Kjellstrand et al., 1983) also reported cytotoxicity and histopathology in mice. All three of these studies scored Medium or High in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and were therefore utilized for dose-response analysis.

3.2.5.1.2 Kidney toxicity

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) considered five animal studies reporting kidney toxicity for further non-cancer dose-response analysis. (Maltoni et al., 1986), (NCI, 1976) and (NTP, 1988) reported histological changes in the kidney, whereas (Kjellstrand et al., 1983) and (Woolhiser et al., 2006) reported increased kidney/body weight ratios (U.S. EPA, 2011e). NCI (1976) scored Unacceptable in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and therefore was excluded from dose-response analysis. All of the other studies scored Medium in data quality and were therefore utilized for dose-response analysis.

3.2.5.1.3 Neurotoxicity

Among the human studies, (Ruijten et al., 1991) was the only epidemiological study that the IRIS program deemed suitable for further evaluation in the TCE's dose-response assessment for neurotoxicity. Only the following four animal studies were considered suitable for dose-response analysis for the neurotoxicity endpoint in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b): (Arito et al., 1994), (Isaacson et al., 1990), (Gash et al., 2008), and (Kjellstrand et al., 1987). Kjellstrand (1987) scored Unacceptable in in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and therefore was excluded from dose-response analysis. Gash et al. (2008) scored a Low in data evaluation and was also not carried forward to dose-response analysis given the other, higher quality studies available. Ruijten et al. (1991), Arito et al. (1994), and Isaacson et al. (1990) all scored Medium or High for data quality and were therefore utilized for dose-response analysis.

3.2.5.1.4 Immunotoxicity

Only the following four animal studies were suitable for the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) non-cancer dose-response analysis for the immunotoxicity endpoint: (Keil et al., 2009), (Kaneko et al., 2000), (Sanders et al., 1982), and (Woolhiser et al., 2006). For this Risk Evaluation, EPA also assessed the endpoint of acute immunosuppression observed in (Selgrade and Gilmour, 2010). In Selgrade et al (2010), mice were infected via respiration with aerosolized *S. zooepidemicus* bacteria following 3h TCE exposure. Mortality, bacterial, clearance from the lung,

percent of mice infected, and phagocytic index were assessed following co-exposure. Mortality was selected as the most statistically sensitive endpoint due to a larger numbers of mice per exposure group and more dose groups, however "percent of mice infected" was also considered for dose-response analysis (Appendix F.2). All of these studies scored Medium or High in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and were therefore utilized for dose-response analysis.

3.2.5.1.5 Reproductive toxicity

Among the human studies, (Chia et al., 1996) was the only epidemiological study that the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) deemed suitable for further evaluation in the TCE's dose-response assessment for reproductive toxicity. Only the following eight reproductive animal toxicity studies were considered suitable for non-cancer dose-response analysis in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b): (Kumar et al., 2000), (Kumar et al., 2001), (Kan et al., 2007), (Xu et al., 2004), (Narotsky et al., 1995), (George et al., 1986), (Duteaux et al., 2004), and (Forkert et al., 2002). Forkert et al. (2002) scored Unacceptable in EPA's data quality evaluation and therefore was excluded from dose-response analysis, however it had the same POD as (Kan et al., 2007), which scored Medium. Duteaux et al. (2004) scored a Low for data quality and was not carried forward to dose-response analysis given the other, higher quality studies available. The remaining studies all scored Medium or High for data quality [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and were therefore utilized for dose-response analysis.

3.2.5.1.6 Developmental toxicity

The 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) found 5 animal studies that were suitable for non-cancer dose- response analysis for the following developmental outcomes: pre- and postnatal mortality; pre- and postnatal growth; developmental neurotoxicity; and congenital heart malformations (Appendix L of that document).

Although the focus of the discussion below is on these 5 studies and corresponding endpoints, it is important to mention that developmental immunotoxicity has also been demonstrated in TCE-treated animals. The most sensitive immune system response was reported by (Peden-Adams et al., 2006). In this study, B6C3F1 mice were exposed to TCE via drinking water. Treatment occurred during mating and through gestation to TCE levels of 0, 1.4, or 14 ppm. After delivery, pups were further exposed for either 3 or 8 more weeks at the same concentration levels that the dams received in drinking water. Suppressed PFC response was seen in male pups after 3 and 8 weeks of exposure, whereas female pups showed the suppression of PFC response and delayed hypersensitivity at 1.4 ppm following 8 weeks. At the higher concentration (14 ppm), both of these effects were observed again in both males and females following 3 or 8 weeks of postnatal exposure. A LOAEL of 0.37 mg/kg-bw/day served as a POD for the decreased PFC and increased delayed hypersensitivity responses (U.S. EPA, 2011e). While this endpoint exhibits one of the lower PODs among developmental toxicity studies, the study scored a "Low" in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* due to concerns over statistical reliability and dose precision (difficult to calculate precise dosage). Additionally, it could not be accurately PBPK modeled because exposure occurred *in* utero, through nursing, and after weaning. Therefore, this study was not considered further for dose-response assessment, although developmental immunotoxicity will still be considered qualitatively.

Pre- and Postnatal Mortality and Growth

The following two studies were considered suitable for non-cancer dose-response analysis for pre- and

postnatal mortality and growth effects in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b): (Healy et al., 1982) and (Narotsky et al., 1995). Healy et al. (1982) scored Unacceptable in in EPA's data quality evaluation [*Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and therefore was excluded from dose-response analysis. (Narotsky et al., 1995) scored a High and was therefore utilized for dose-response analysis.

Developmental Neurotoxicity

There is evidence of alterations in animal brain development and in behavioral parameters (e.g., spontaneous motor activity and social behaviors) following TCE exposure during the development of the nervous system. Among all of the reasonably available studies, there were two oral studies that reported behavioral changes which were used in the dose-response evaluation for developmental toxicity: (Fredriksson et al., 1993) and (Taylor et al., 1985). (Taylor et al., 1985) scored a Low in EPA's data quality evaluation due to the same issues as (Peden-Adams et al., 2006) and was not considered further for dose-response assessment. (Fredriksson et al., 1993) scored a Medium despite some uncertainty concerning the statistical validity of its sampling methodology [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* and was therefore utilized for dose-response analysis.

Congenital Heart Defects

The fetal cardiac defects reported in (Dawson et al., 1993) and (Johnson et al., 2003) were identified as the most sensitive endpoint within the developmental toxicity domain and across all of the health effects domains evaluated in the TCE IRIS assessment. Johnson et al. (Johnson et al., 2003) reported data from different experiments over a several-year period in which pregnant Sprague-Dawley rats (9-13/group; 55 in control group) were exposed to TCE via drinking water. Treatment of pregnant rats occurred during the entire gestational period (i.e., GD 0 to GD22). The study was a follow-up to Dawson et al. (1993), which demonstrated increasing incidence of congenital heart defects at the highest two dose groups that were later pooled and re-analyzed in (Johnson et al., 2003).

Much of the controversy surrounding the reliability of the (Johnson et al., 2003) study relates to the pooling of control animals and data across several years, including the use of different vehicles (tap water vs distilled water). EPA therefore compared the data from (Johnson et al., 2003) and from (Dawson et al., 1993), the earlier study comprising the highest two doses of the (Johnson et al., 2003) study in which data was not pooled and only a single vehicle was used. Unfortunately, EPA was unable to use a nested benchmark dose (BMD) model because individual pup data could not be easily tracked to a particular dam, so this data is less statistically reliable. Both studies scored a "Medium" in in EPA's data quality evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]*, which incorporated all available information on the two studies, including subsequent errata and communications to EPA (Johnson et al., 2014; Johnson, 2014, 2008; Johnson et al., 2005). While the original publications had extensive data and methodology reporting issues, many of the data quality concerns from the original study were mitigated by the information provided in these updates. These updates provided the following information which was lacking in the initial publications:

1) Individual fetal cardiac malformation data for each litter
2) Individual maternal terminal body weight data
3) Detailed description of fetal evaluation procedures including:
    - methods used to blind fetal examiners to treatment group
    - protocol for unanimous confirmation of any observed cardiac defects by the three principle investigators

3) Additional information on animal husbandry and randomized group assignment of dams to study group
4) Transparency regarding experimental variables across the dates of the experiments

Because both studies passed data evaluation with the same score and statistics could only be performed using a pup as the statistical unit for (Dawson et al., 1993), EPA decided to utilize the (Johnson et al., 2003) data for dose-response analysis, which has increased statistical sensitivity from the additional two dose levels and allowed a nested design for BMD modeling analysis in order to account for litter effects. Additionally, some defects originally identified in (Dawson et al., 1993) were later reclassified or recharacterized in (Johnson et al., 2003), so (Johnson et al., 2003) contains the more updated analysis.

3.2.5.1.7 Cancer

The 2019 meta-analysis of all relevant studies examining kidney cancer, liver cancer, or NHL (Appendix H) came to the same conclusion as the previous EPA meta-analysis in the 2011 IRIS Assessment (U.S. EPA, 2011e). Therefore, EPA utilized the same inhalation unit risk and oral slope factor estimates as were derived in (U.S. EPA, 2011e) and cited in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). A linear non-threshold assumption was applied to the TCE cancer dose-response analysis because there is sufficient evidence that TCE-induced kidney cancer operates primarily through a mutagenic mode of action while it cannot be ruled out for the other two cancer types.

The 2011 IRIS Assessment (U.S. EPA, 2011e) selected the epidemiological kidney cancer data Charbotel et al (2006) as the best representative dose-response data for derivation of an oral slope factor and inhalation unit risk value as a case-control study with quantitative cumulative exposure estimates based on a task-exposure matrix based on decades of measurement. Charbotel et al (2006) received a High score for data quality both overall and for the exposure domain in EPA's data evaluation [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]*. Therefore, EPA relied on its previous dose-response analysis from this study.

3.2.5.2 Potentially Exposed and Susceptible Subpopulations (PESS)

TSCA requires that a risk evaluation "determine whether at chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of cost or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use." 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 susceptibility*. EPA addresses the subpopulations identified as relevant based on *greater exposure* in Section 2.3.3.

There is some evidence that certain populations may be more susceptible to exposure to TCE. Factors affecting susceptibility examined in the available studies on TCE include lifestage, gender, genetic polymorphisms, race/ethnicity, preexisting health status, lifestyle factors, and nutrition status. Factors

that affect early lifestage susceptibility include exposures during gestation, such as transplacental transfer, and during infancy, such as breast milk ingestion, early lifestage-specific toxicokinetics, and early lifestage-specific health outcomes including developmental cardiac defects. Gender-specific differences also exist in toxicokinetics (e.g., cardiac outputs, percent body fat, expression of metabolizing enzymes) and susceptibility to toxic endpoints (e.g., gender-specific effects on the reproductive system, gender differences in baseline risks to endpoints such as scleroderma or liver cancer). Genetic variation likely has an effect on the toxicokinetics of TCE. Pre-existing diminished health status may alter the response to TCE exposure. Individuals with increased body mass may have an altered toxicokinetic response due to the increased uptake of TCE into fat. Other conditions that may alter the response to TCE exposure include diabetes and hypertension, and lifestyle and nutrition factors such as alcohol consumption, tobacco smoking, nutritional status, physical activity, and socioeconomic status (U.S. EPA, 2011e). Among life stages, the most susceptible is likely to be pregnant women and their developing fetus based on the hazard findings from reviewing the reasonably available literature for this assessment, which conclude that developmental toxicity is among the most sensitive acute health effects associated with TCE exposure. Among pregnant women, older women may be especially susceptible to TCE-induced cardiac defects in their offspring. Maternal age is known to have a large influence on the incidence of congenital heart defects, and multiple studies cited in this Risk Evaluation identified a significantly stronger association of TCE with developmental cardiac defects (Brender et al., 2014; Yauck et al., 2004). Additional maternal risk factors for susceptibility to congenital cardiovascular defects include diabetes, infection status, drug exposure, and stress, among others (Jenkins et al., 2007).

Significant variability in human susceptibility to TCE toxicity may result from differences in metabolic potential, given the existence of CYP isoforms and the variability in CYP-mediated TCE oxidation (U.S. EPA, 2011e). Increased enzymatic activity of cytochrome P450 2E1 (CYP2E1) and glutathione-S-transferase (GST) polymorphisms may influence TCE susceptibility due to effects on the production of toxic metabolites (U.S. EPA, 2011e). More specifically, there appears to be greater susceptibility to TCE-induced kidney cancer in those individuals that carry an active polymorphism in a gene associated with the GST metabolic pathway. Particularly, the gene is associated with the β-lyase gene region which is responsible for converting DCVC to the unstable intermediate DCVT. Also, there are some human studies suggesting a role for mutations to the tumor suppressor gene, von Hippel Lindau (VHL gene). This tumor suppressor gene appears to be inactivated in certain TCE-induced kidney cancers (U.S. EPA, 2011e). In the 2014 TCE risk evaluation (U.S. EPA, 2014b), EPA performed a population analysis to systematically estimate uncertainty and variability across several metabolic factors, including human variability related to oxidative metabolism and glutathione conjugation as a result of GST activity. Integration of these factors into a probabilistic model resulted in a distribution of human equivalent concentrations/doses (HECs/HEDs) for each endpoint. HEC_{99}/HED_{99} values representing the most metabolically sensitive 1% of the population, a susceptible subpopulation, were used for risk evaluation, and EPA utilized the same analysis for this assessment.

3.2.5.3 Derivation of Points of Departure (PODs)

Point of departures (PODs) were identified for those studies that had suitable data for dose-response analysis, described above. PODs can be a NOAEL or LOAEL for an observed incidence, or change in level of response, or the lower confidence limit on the dose at the benchmark dose (BMDL). PBPK modeling was used to estimate internal dose PODs (idPOD) and subsequently the human equivalent concentrations/doses (HECs/HEDs) based on the oral and inhalation PODs identified in earlier steps. The PBPK modeling integrated internal dose-metrics based on TCE's mode of action and the role of different TCE metabolites in toxicity (U.S. EPA, 2011e). Note that the effects within the same health effect domain were generally assumed to have the same relevant internal dose-metrics, with some

exceptions. Compared to the 2014 TSCA Work Plan Chemical Risk Assessment, an additional POD from Selgrade (2010) has also been added for acute exposure scenarios.

For this assessment, when an endpoint can be BMD and PBPK modeled, default cumulative acute UF = 10 (UF_A and UF_H both = 3 based only on toxicodynamic uncertainty (UF_{TD}); UF_S and UF_L = 1) and default cumulative chronic UF = 100 (UF_S = 10 if the study covers less than 10% of lifetime). See Appendix O for details on the criteria for selection of appropriate BMD models and UFs for each endpoint.

POD Selection Metrics

The below sections present all studies considered for dose-response analysis. From this list, the studies were selected from each health domain /organ system that best represent each available endpoint. For some health domains with multiple endpoints this resulted in multiple studies being selected for consideration in risk estimation. In selecting the most representative studies and PODs, EPA considered the following factors:

- Data quality evaluation score
- Species (i.e. animal or human)
- Exposure duration
- Dose range
- Cumulative uncertainty factor
- Relevance to the endpoint of interest and human exposure scenarios

Dose metric selection is based on a determination of which toxicokinetic measure is most predictive of localized effects from TCE exposure. These factors were evaluated for each independent endpoint, and EPA considered use of the most health-protective POD only after first considering each of the above factors. See the 2011 EPA TCE IRIS Assessment (U.S. EPA, 2011e) for more details on dose-metric and benchmark response (BMR) determinations for all endpoints except that from Selgrade and Gilmour (2010). BMD modeling results for (Selgrade and Gilmour, 2010) are presented in Appendix F.

3.2.5.3.1 Non-Cancer PODs for Acute Exposure

Acute exposure in humans is defined for occupational settings as exposure over the course of a single work shift (8 hours) and for consumers as a single 24-hour day. Although developmental studies typically involve multiple exposures, they are considered relevant for evaluating single exposures because evidence indicates that certain developmental effects may result from a single exposure during a critical window of development (Davis et al., 2009; Van Raaij et al., 2003; U.S. EPA, 1991). This is consistent with EPA's *Guidelines for Reproductive Toxicity Risk Assessment* (U.S. EPA, 1996), which state that repeated exposure is not a necessary prerequisite for the manifestation of developmental toxicity. Therefore, developmental endpoints were considered relevant for calculating risks associated with acute occupational or consumer exposure. Single-exposure studies identifying a dose-responsive specific health outcome were also considered for deriving PODs representative of risks following acute exposures.

HECs for developmental toxicity were adjusted to reflect a 24-hr value, consistent with both occupational and consumer exposure values. The POD from Selgrade (2010), a 3hr acute inhalation study, was adjusted to a 24hr HEC value for occupational risk estimates due to limited reasonably available occupational exposure information below 8hr time periods. The 3hr POD was used without adjustment for estimation of consumer risks due to available exposure estimates for 3hr time periods.

Developmental Toxicity Endpoints

-- *Prenatal Mortality*

(Narotsky et al., 1995) was also discussed above in the reproductive toxicity section, but also identified mortality to the developing fetus following *in utero* TCE exposure. F344 timed-pregnant rats (8-12 dams/group) were treated with TCE by gavage during GD 6 to 15. The $BMDL_{01}$ for increased resorptions was 32.2 mg/kg-bw/day (U.S. EPA, 2011e).

-- *Developmental Neurotoxicity*

(Fredriksson et al., 1993) treated male NMRI mouse pups (12/group, selected from 3−4 litters) with TCE via gavage (0, 50, or 290 mg/kg-bw/day) during postnatal days (PND) 10 to 16. Locomotor behavior was evaluated at PND 17 and 60. TCE-treated mice showed decreased rearing activity at both dose levels on PND 60, but not PND 17, resulting in a LOAEL of 50 mg/kg-bw/day as a POD (U.S. EPA, 2011e).

-- *Congenital Heart Malformations*

(Johnson et al., 2003) reported a statistically and biologically significant increase in the formation of heart defects at the 0.048 mg/kg-bw/day and higher dose levels (concentrations of 0, 0.00045, 0.048, 0.218 or 129 mg/kg-bw/day) measured on both an individual fetus basis and a litter basis. A $BMDL_{01}$ HEC_{99} of 0.0037 ppm and HED_{99} of 0.0052 mg/kg-bw/day were identified as the inhalation and oral PODs, respectively, for heart malformations in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). EPA quantified the totality of cardiac defects instead of any particular defect, as cardiac teratogens can result in a diverse constellation of effects (e.g., retinoic acid, see Appendix G.1.2.2).

The BMR selection from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b) for (Johnson et al., 2003) was also reassessed based on the non-monotonic dose-response, decreased incidence from control at the 2.5ppb dose level, and reduced statistical power due to a less than recommended number of litters assessed for each dose group. These concerns were discussed as part of a re-analysis of the 2011 dose-response assessment in (Makris et al., 2016), which acknowledged the uncertainty inherent in a selection of a 1% BMR:

"*BMD inference at the 1% extra-risk level is highly uncertain, because BMD and BMDL values vary by several orders of magnitude depending on the modeling assumptions. This is attributed in part to the lack of monotonicity at the lowest dose and the apparent supralinearity of the overall exposure-response relationship. Additional doses would be required to better specify the curve shape in the low-dose region. More reliable inference can be made for higher BMRs…*

There is substantial model and parameter uncertainty at the 1% level of extra risk, although 1% is the appropriate BMR based on severity of the effect (i.e., cardiac malformations). These uncertainties can be attributed primarily to having too few data points in the low-dose range, where more data would be required to adequately characterize the dose-response shape. Uncertainty decreases for higher BMR levels (5% and 10% extra risk), although 10% exceeds the range of the data for some models".

In reevaluating the BMR, EPA considered both biological and statistical factors:

1. The biological severity of the effect
2. The range of observable data relative to the BMR and resulting BMDL
3. The influence of study design and sample size on statistical sensitivity
4. Confidence in the model fit and variance

After considering all these factors, EPA determined that the biological severity of the effect, potentially lethal heart defects, strongly supported a BMR of 1%. For statistical considerations, EPA referred to the nested BMD modeling results from Appendix F.4.2.1 in (U.S. EPA, 2011e). In these results, the BMDL for both a 1% and 5% BMR easily fall within the experimental dose range, increasing confidence in the target BMRs. The observed incidence for the lowest dose in (Johnson et al., 2003) was reduced from controls, adding uncertainty to the modeling estimate, however the difference was not statistically significant. A larger sample size for the treated groups may have increased the statistical sensitivity at lower doses. The BMD model actually displays better visual fit at the lower end of the dose range, near the control, suggesting that a lower BMR may actually represent a more accurate model estimate.

In evaluating model fit, EPA determined that the BMD:BMDL ratio was adequate (3.1), indicating reasonably small variance. The original reported p-value for the model fit was poor (p = 0.0129). However, there were limitations in the way BMDS calculated p-values at that time (i.e., subgrouping individual litter results) and limitations in the fitting of inter-litter correlations in the 2011 version of BMDS. Accordingly, EPA conducted further modeling with this data in the original 2011 assessment and with the latest version of BMDS:

- 2011 Re-analysis: An R program was applied which demonstrated an adequate model fit (Appendix F in (U.S. EPA, 2011e)). This approach still relied on the subgrouping of individual litter results but regrouped the litter data 100 times and reported the percentage of times the estimated p-value indicated appropriate model fit.
- New BMDS Analysis (2019): BMD modeling was re-run on the (Johnson et al., 2003) dataset using the latest version of the BMDS nested models (v3.1.1), which no longer requires subgrouping litter data to calculate p-values. The resulting BMDLs and AICs agreed with results in the 2011 IRIS Assessment (U.S. EPA, 2011e). However, the p-value of = 0.661 from the updated BMDS nested model run (Appendix N) is significantly improved, demonstrating strong model fit and confirming the 2011 conclusion that the modeling results for cardiac malformation data are appropriate for reference value derivation.

Based on the above considerations and the improved model fit from the updated BMD modeling run, EPA determined that use of a 1% BMR is most appropriate for risk estimation. The difference between the 1% and 5% BMR POD values is 5.2-fold. Results for both 1% and 5% extra risk BMR options (along with 10%) are presented in Appendix N.

Immunotoxicity

-- Immunosuppression (diminished response to infection)

In addition to the previously described developmental toxicity studies, (Selgrade and Gilmour, 2010) was deemed suitable for dose-response analysis of immunotoxicity based on observed decreased response to infection. In Selgrade et al (2010), female CD-1 mice were infected via respiration with aerosolized *S. zooepidemicus* bacteria following 3h exposure to 0, 5, 10, 25, 50, 100, or 200 ppm of TCE. Mortality was assessed for all dose groups, with statistically significant and dose-responsive increases observed at 50 ppm and above. Bacterial clearance from the lung, percent of mice infected, and phagocytic index were also assessed for 0, 50, 100, and 200ppm dose groups. This study examined pulmonary immunological responses to respiratory infection following inhalation of TCE and is therefore only applicable to inhalation exposure. The inclusion of the Selgrade and Gilmour (2010) study is an addition

to this risk evaluation and was not previously evaluated for dose-response analysis in the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). This study was discussed in the 2011 IRIS Assessment (U.S. EPA, 2011e) but was excluded from the 2014 Risk Assessment in an oversight.

For (Selgrade and Gilmour, 2010), BMD modeling was performed on the endpoints of mortality and percentage of mice infected (see [*Personal Communication to OPPT. Raw Data Values from Selgrade and Gilmour, 2010. Docket: EPA-HQ-OPPT-2019-0500*]). A reliable BMDL could not be obtained from the percentage infected data because BMDs and BMDLs from all models were well below the lowest data point and cannot be considered reliable. For mortality, a BMR of 1% increase was selected due to the severity of the effect. Based on evidence of systemic chronic immunosuppression (Sanders et al., 1982; Woolhiser et al., 2006), this acute endpoint was applied to systemic exposure. Based on assumed ppm equivalence across species (U.S. EPA, 2011e), the $BMDL_1$ also serves as the HEC for 3hr exposure, while 1.74 ppm is the HEC for 24hr exposure. Route-to-route extrapolation and allometric scaling based on values from (U.S. EPA, 1988) and subsequent allometric scaling results in a dermal HED of 2.74 mg/kg.

Table 3-7: Dose-response analysis of selected studies considered for acute exposure scenarios

Target Organ/ System	Species	Duration	POD Type [1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Develop-mental Effects	Rat (female)	Gestational days 6 to 15	$BMDL_{01}$= 32.2 mg/kg-bw/day	Increased resorptions	TotMetab BW34	57	23	29	28	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; Total UF=10	(Narotsky et al., 1995)	High (1.3)
	Rat (female)	22 days throughout gestation (gestational days 0 to 22)	$BMDL_{01}$ = 0.0207 mg/kg-bw/day	Congenital heart defects	TotOx Metab BW34	0.012	0.0037	0.0058	0.0052	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; Total UF=10	(Johnson et al., 2003)	Medium (1.9)
	Rat (male pups)	Postnatal days 10 to 16	LOAEL = 50 mg/kg-bw/day	Decreased rearing activity	TotMetab BW34	8	3	4.2	4.1	UF_S=1; UF_A= 3; UF_H=3; UF_L=10; Total UF=100	(Fredriksson et al., 1993)	Medium (1.7)
Immune System	Rat (female)	3hr/day, single dose; followed by respiratory infection	$BMDL_{01}$ = 13.9 ppm	Immuno-suppression	N/A[4]	N/A[4]	1.74[4]	N/A[4]	2.74[4,5]	UF_S=1; UF_A= 3; UF_H=10; UF_L=1; *Total UF=30*	(Selgrade and Gilmour, 2010)	High (1.6)

[1] POD type can be NOAEL, LOAEL, or BMDL. EPA adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric.
[4] Data from Selgrade and Gilmour, 2010) was not subject to PBPK modeling due to uncertainty concerning the most appropriate dose metric. The BMDL value adjusted for a 24hr exposure will be used as the POD for occupational risk estimates, while the 3hr value will be used for consumer risk estimates. This value is presented in the HEC_{99} column but does not represent any particular percentile since it was not PBPK-modeled.
[5] A dermal HED was obtained through route-to-route extrapolation using breathing rate and body weight data on male CD-1 mice (insufficient female data was reasonably available) from (U.S. EPA, 1988) and allometric scaling based on (U.S. EPA, 2011d) using a dosimetric adjustment factor of 0.14 for mice.

Table 3-7 presents the derived PODs from all studies considered for dose-response analysis of acute exposure scenarios. EPA selected studies representative of the distinct endpoints of prenatal mortality, congenital defects, developmental neurotoxicity, and response to infection. Most of the developmental toxicity studies utilized the PBPK dose metric of TotMetabBW34, or the total amount TCE metabolized per unit adjusted body weight. This dose metric was selected because for these endpoints there is insufficient information for site-specific or mechanism-specific determinations of an appropriate dose-metric, however in general TCE toxicity is associated with metabolites rather than the parent compound. TotOxMetab34, or the total amount TCE oxidized per unit adjusted body weight, was used for deriving HEC/HED values for congenital heart defects because evidence demonstrating effects from TCA and

DCA (see Section 3.2.4.1.6) suggests that oxidative metabolism is important for TCE-induced heart malformations.

The LogProbit model was selected for BMD modeling results of (Selgrade and Gilmour, 2010) data because it was the model with the lowest AIC, using a BMR of 1% based on the endpoint of mortality. Data from (Narotsky et al., 1995) and (Johnson et al., 2003) were also BMD modeled. A BMR of 1% ER was selected for (Johnson et al., 2003) based on the severity of the effect and absence of a strong statistical justification for raising the value (see discussion above). A BMR of 1% was also selected for (Narotsky et al., 1995) because of the severity of the effect (full-litter resorptions) and low background response. A LOAEL was used as a POD for (Fredriksson et al., 1993), which was not BMD modeled. For acute exposures, subchronic-to-chronic UF does not apply, so $UF_S = 1$ for all studies. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling, dose metric selection, and BMR selection.

Differences from standard UF values are explained below:
A UF_A value of 3 was applied to (Selgrade and Gilmour, 2010) because cross-species scaling based on blood:air partition coefficient or allometric scaling for body weight was used to adjust the HEC/HED as necessary. A UF_H of 10 was applied to that study because the data was not subject to PBPK modeling and therefore a HEC99/HED99 value was not applied which would have accounted for human toxicokinetic variability.

The selected studies are bold in the table above. The endpoints were each represented by a single study. While there are some methodological and statistical concerns about (Johnson et al., 2003) and (Fredriksson et al., 1993), based on the WOE for the endpoints and data quality scores of at least Medium, all four of the studies will be utilized for quantitative risk estimation following acute exposures. There is also some inherent uncertainty extrapolating from the response to pulmonary infection observed in (Selgrade and Gilmour, 2010) to a systemic response across multiple exposure routes, but an acute systemic response to infection is likely based on the systemic immunosuppression observed in a chronic study (Keil et al., 2009).

3.2.5.3.2 Non-Cancer PODs for Chronic Exposures

Chronic exposure was defined for occupational settings as exposure reflecting a 40-hour work week. Chronic exposure was not considered relevant to to consumers based on expected use patterns (Section 2.3.2.7.1). Non-cancer endpoints selected as most relevant for calculating risks associated with chronic (repeated) occupational exposures to TCE included effects on the to the liver, kidney, nervous system, immune system, reproductive system, and development, with all HECs adjusted to reflect a 24-hr value, consistent with calculated occupational exposure values.

Liver toxicity

-- *Increased liver weight and cytotoxicity/hypertrophy*

(Kjellstrand et al., 1983) exposed NMRI male mice (10-20/group) with up to nine different TCE concentrations. These concentrations ranged from 37 to 3,600 ppm and included an air control group. Exposures were conducted for various durations (1, 2, 4, 8, 16, or 24 hrs/day) and for different time frames (from 30 to 120 days). EPA calculated a benchmark concentration lower-bound confidence limit of 21.6 ppm based on the 10% benchmark response ($BMDL_{10}$) for increased liver/body weight ratios, with cytotoxicity and histopathology also observed.

(Buben and O'Flaherty, 1985) exposed Swiss-Cox male mice (12-15 group) to TCE by gavage. Mice were exposed to a range of TCE doses (100 to 3,200 mg/kg-bw/day plus control) for 5 days/week for 6

weeks. A $BMDL_{10}$ of 82 mg/kg-bw/day was identified as the POD for increased liver/body weight ratios, with cytotoxicity and histopathology also observed.

In (Woolhiser et al., 2006), Sprague-Dawley female rats (16/group) were exposed to TCE via inhalation at concentrations of 0, 100, 300, or 1,000 ppm for 6 hrs/day, 5 days/week for 4 weeks. A $BMDL_{10}$ of 25 ppm was estimated for increased liver/body weight ratio.

Table 3-8: Dose-response analysis of selected studies considered for evaluation of liver toxicity

Target Organ System	Species	Duration	POD Type [1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Liver	**Mouse (male)**	**Continuous and intermittent exposures, variable time periods for 30-120 days**	**$BMDL_{10}$= 21.6 ppm**	**Increased liver/body weight ratio and cytotoxicity/ hypertrophy**	**AMetLiv1 BW34**	**25**	**9.1**	**9.0**	**7.9**	**UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10***	**(Kjellstrand et al., 1983)**	**Medium (1.8)**
	Mouse (male)	6 weeks	$BMDL_{10}$= 82 mg/kg-bw/day		AmetLiv1 BW34	32	11	12	10	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Buben and O'Flaherty, 1985)	High (1.3)
	Rat (female)	6 hr/day, 5 days/week for 4 weeks	$BMDL_{10}$= 25 ppm	Increased liver/body weight ratio	AmetLiv1 BW34	53	19	19	16	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Woolhiser et al., 2006)	Medium (2)*

[1] POD type can be NOAEL, LOAEL, or BMDL. EPA adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric. * Woolhiser 2006 was downgraded from a High, with calculated score = 1.3.

Table 3-8 presents the derived PODs from all studies considered for dose-response analysis. Increased liver/body weight ratio was the only endpoint modeled from all studies based on the dose metric AMetLiv1BW34, or the amount of TCE oxidized in liver per unit adjusted body weight. This dose metric was selected because evidence suggests that hepatic oxidative metabolism is involved in TCE liver toxicity and dose-response relationships using this metric showed greater consistency than other considered metrics. All studies were BMDL modeled. A BMR of 10% RD was used to represent a minimal, biologically significant amount of change in relative liver weight. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling, dose metric selection, and BMR selection.

Differences from standard UF values are explained below:
All three studies were assigned UF_S = 1 despite shorter exposure duration because although the studies were subchronic, hepatomegaly (enlarged liver) occurs rapidly with TCE exposure, and no differences were observed in severity of relative kidney weight increases between 30 and 120 days in (Kjellstrand et al., 1983).

The data from (Kjellstrand et al., 1983) was selected to represent the liver toxicity hazard. (Woolhiser et al., 2006) was excluded from further consideration because additional signs of toxicity were not observed, indicating that the increased liver weight was likely merely adaptive. (Kjellstrand et al., 1983) was selected over (Buben and O'Flaherty, 1985) because it covered up to 120 days exposure as opposed to only 42 days. Additionally, (Kjellstrand et al., 1983) utilized the widest dose range of any study, imparting more precision in the POD estimate.

Kidney toxicity

-- *Kidney Pathology*

(Maltoni et al., 1986) exposed Sprague-Dawley male rats (116-124/group) to TCE via inhalation (0, 100, 300, or 600 ppm) for 7 hrs/day, 5 days/week for 104 weeks (and allowed all rats to continue unexposed until they died). The investigators also conducted an oral (gavage) study that dosed rats with a range of TCE doses (50 to 250 mg/kg-bw/day) for 4-5 days/week for 52 weeks. $BMDL_{10}$ values of 40.2 ppm and 34 mg/kg-bw/day were calculated for the inhalation and gavage studies, respectively, based on renal tubular pathological changes (meganucleocytosis) observed in male rats (U.S. EPA, 2011e). These changes included dose-dependent enlargement of tubuli cells (cytomegaly) and their nuclei (karyomegaly) leading to dysplasia, which may serve as a precursor to cancer and/or morphological indicators of damaged kidney function (Maltoni et al., 1986).

In another oral (gavage) study (NTP, 1988), the National Toxicology Program exposed Marshall female rats (44-50/group) to TCE (i.e., 0, 500, or 1,000 mg/kg-bw/day) for 5 days/week for 104 weeks. Rats developed toxic nephropathy following TCE exposure. A $BMDL_{05}$ of 9.45 mg/kg-bw/day was calculated for the observed kidney effects (U.S. EPA, 2011e).

-- *Increased Relative Kidney Weight*

(Woolhiser et al., 2006) conducted an inhalation study that exposed Sprague-Dawley female rats (16/group) to 0, 100, 300 or 1,000 ppm TCE for 6 hrs/day for 5 days/weeks for 4 weeks. At the end of the study, rats exhibited increased kidney/body weight ratios and a $BMDL_{10}$ of 15.7 ppm was estimated for these effects (U.S. EPA, 2011e).

Increased kidney/body weight ratios were also seen in (Kjellstrand et al., 1983). NMRI male mice (10-20/group) were exposed to a range of TCE concentrations (37 to 3,600 ppm) for 30 to 120 days on continuous and intermittent exposure regimens. A $BMDL_{10}$ of 34.7 ppm was identified as the POD for increased kidney/body weight ratios (U.S. EPA, 2011e).

Table 3-9: Dose-response analysis of selected studies considered for evaluation of kidney toxicity

Target Organ System	Species	Duration	POD Type [1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Kidney	Rat (female)	5 days/week for 104 weeks	$BMDL_{05}$ = 9.45 mg/kg-bw/day	Toxic nephropathy	ABioact DCVC BW34	0.042	0.0056	0.033	0.0034	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(NTP, 1988)	Medium (2)*
	Rat (male) - Oral	**4-5 days/week for 52 weeks**	**$BMDL_{10}$ = 34 mg/kg-bw/day**	**Pathology changes in renal tubule**	**ABioact DCVC BW34**	**0.19**	**0.025**	**0.15**	**0.015**	**UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10***	**(Maltoni et al., 1986)**	Medium (2)*
	Rat (male) - Inhal.	7 hrs/day, 5 days/week for 2 years	$BMDL_{10}$= 40.2 ppm	Pathology changes in renal tubule	ABioact DCVC BW34	0.28	0.038	0.22	0.023	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Maltoni et al., 1986)	Medium (2)*
	Rat (female)	6 hr/day, 5 days/week for 4 weeks	$BMDL_{10}$= 15.7 ppm	Increased kidney weight/body weight ratio	ABioact DCVC BW34	0.099	0.013	0.078	0.0079	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Woolhiser et al., 2006)	Medium (2)*
	Mouse (male)	Continuous and intermittent exposures for 30-120 days	$BMDL_{10}$ = 34.7 ppm	Increased kidney weight/body weight ratio	AMet GSH BW34	0.88	0.12	0.69	0.07	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Kjellstrand et al., 1983)	Medium (1.8)

[1] POD type can be NOAEL, LOAEL, or BMDL. EPA adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric. *NTP 1998 was downgraded from a High, with calculated score = 1.2; Maltoni 1986 was downgraded from a High, with calculated scores = 1.4 (oral) and 1.3 (inhalation); Woolhiser 2006 was downgraded from a High, with calculated score = 1.3.

Table 3-9 presents the derived PODs from all studies considered for dose-response analysis. The studies considered for dose-response analysis identified either indications of kidney pathology or increase kidney/body weight ratio. All rat studies utilized ABioactDCVCBW34, or the amount of DCVC bioactivated in the kidney per unit adjusted body weight, because GSH-conjugative bioactivation of TCE into metabolites such as DCVC in the kidney is expected to be responsible for kidney toxicity. AMetGSHBW34, or the amount of TCE conjugated with GSH per unit adjusted body weight, was utilized for mice studies because PBPK information on DCVC activation in mice is not reasonably available. All studies were BMDL modeled. A BMR of 5% ER was used for (NTP, 1988) because toxic nephropathy is a severe toxic effect. (Maltoni et al., 1986) used a BMR of 10% ER because meganuclocytosis is considered minimally adverse, while both studies examining increased relative kidney weight used a standard BMR of 10% RD. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling, dose metric selection, and BMR selection.

Differences from standard UF values are explained below:
(Woolhiser et al., 2006) and (Kjellstrand et al., 1983) were assigned UF_S = 1 despite shorter exposure duration because no differences were observed in severity of relative kidney weight increases between 30 and 120 days in (Kjellstrand et al., 1983).

EPA determined that kidney pathology was a better indicator of adverse kidney effects than increased relative organ weight and therefore only that endpoint was selected to represent kidney toxicity. While there are concerns about the procedure of continuing observation until spontaneous death in (Maltoni et al., 1986) due to the potential for confounding effects from autophagy or infection, there are unlikely to be significant artifacts from this methodology affecting the interpretation of kidney lesions. There was random allocation to study groups and kidney lesions were not observed in the control or lowest dose group. Therefore, background false positives were not an issue and the observed dose-response is expected to be independent of this confounder. Additionally, a 2011 review of pathology results from other cancer studies performed in this laboratory (Ramazzini Institute) by the NTP Pathology Working Group (Malarkey and Bucher, 2011) found good agreement on the interpretation of most solid tumors and only identified significant differences among inflammatory cancers of the blood and respiratory tract.

Both (Maltoni et al., 1986) and (NTP, 1988) scored a Medium in data quality, however (Maltoni et al., 1986) tested exposure over a sufficiently similar duration with a more appropriate dose range. The elevated doses in (NTP, 1988) resulted in massive nephrotoxicity and introduce large uncertainty in BMD modeling the effects at low doses well below the tested doses with a BMR well below the observed effect incidence in the study. Therefore, the BMDL and resulting HEC/HED from (Maltoni et al., 1986) was considered more reliable. Among the inhalation and oral results from (Maltoni et al., 1986), with few other differences among the data the lower resulting oral POD was selected to represent the endpoint in order to be health-protective. Of note, this represents a change from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b), which selected the POD from (NTP, 1988) to represent kidney toxicity.

Neurotoxicity

-- *CNS Depression*

(Arito et al., 1994) exposed Wistar male rats (5/group) to TCE via inhalation to concentrations of 0, 50, 100, or 300 ppm for 8 hrs/day, 5 days/week for 6 weeks. Exposure to all of the TCE concentrations significantly decreased the amount of time spent in wakefulness during the exposure period. Some carry over was observed in the 22 hr-post exposure period, with significant decreases in wakefulness seen at 100 ppm TCE. Significant changes in wakefulness- sleep elicited by the long-term exposure

appeared at lower exposure levels. The LOAEL for sleep changes was 12 ppm (i.e., LOAEL, adjusted for continuous exposure) (U.S. EPA, 2011e).

-- *Trigeminal nerve effects*

(Ruijten et al., 1991) evaluated the TCE exposures and possible health effects of 31 male printing workers (mean age: 44 yrs) and 28 unexposed control subjects (mean age: 45 yrs). The exposure duration was expressed as "cumulative exposure" (concentration × time). Using historical monitoring data, mean exposures were calculated as 704 ppm × number of years worked, where the mean number of years was 16 (range: 160-2,150 ppm x yr) (U.S. EPA, 2011e). The study measured the trigeminal nerve function by using the blink reflex, but no abnormal findings were observed. However, the study found a statistically significant average increase in the latency response time in TCE-exposed workers on the masseter reflex test, another test commonly used to measure the integrity of the trigeminal nerve. The POD derived from the dataset was a LOAEL of 14 ppm (U.S. EPA, 2011e).

-- *Neuronal demyelination*

(Isaacson et al., 1990) dosed weanling Sprague-Dawley male rats (12/dose group) via the oral route (drinking water) in an experimental protocol for an 8-week period. The control group had unexposed rats for 8 weeks. The experimental group#1 exposed rats to 47 mg/kg-bw/day TCE for 4 weeks and then no TCE exposure for 4 weeks. The experimental group#2 exposed rats to 47 mg/kg-bw/day TCE for 4 weeks, no TCE exposure for the following 2 weeks, and then 24 mg/kg-bw/day TCE for the final 2 weeks. Rats in group#2 reported a decreased latency to find the platform in the Morris water maze test. While these results actually suggest increased cognitive performance, all of the TCE-treated groups exhibited hippocampal demyelination, with effects more severe in the twice-exposed group. The LOAEL for neurodegenerative effects (i.e., demyelination in the hippocampus) was 47 mg/kg-bw/day (U.S. EPA, 2011e).

Table 3-10: Dose-response analysis of selected studies considered for evaluation of neurological effects

Target Organ System	Species	Duration	POD Type [1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Nervous system	**Rat (male)**	**8 hrs/day, 5 days/weeks for 6 weeks**	**LOAEL = 12 ppm**	**Significant decreases in wakefulness**	**TotMetab BW34**	**13**	**4.8**	**6.6**	**6.5**	**UF_S=3; UF_A= 3; UF_H=3; UF_L=10; *Total UF=300***	**(Arito et al., 1994)**	**Medium (2)***
	Human (both sexes)	**Mean of 16 years**	**LOAEL = 14 ppm**	**Trigeminal nerve effects (increased latency in masseter reflex)**	**TotMetab BW34**	**14**	**5.3**	**7.4**	**7.3**	**UF_S=1; UF_A= 1; UF_H=3; UF_L=3; *Total UF=10***	**(Ruijten et al., 1991)**	**Medium (1.7)**
	Rat (male)	8 weeks (intermittent)	LOAEL = 47 mg/kg-bw/day	Demyelination of hippocampus	TotMetab BW34	18	7.1	9.4	9.2	UF_S=10; UF_A= 3; UF_H=3; UF_L=10; *Total UF=1000*	(Isaacson et al., 1990)	Medium (2)*

[1] POD type can be NOAEL, LOAEL, or BMDL. EPA adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric. *Arito 1994 was downgraded from a High, with calculated score = 1.6; Isaacson 1990 was downgraded from a High, with calculated score = 1.6

Table 3-10 presents the derived PODs from all studies considered for dose-response analysis. The reasonably available datasets for considering neurotoxicity included single studies for each of the three endpoints of central nervous system (CNS) depression, trigeminal nerve effects, and neuronal demyelination. The TotMetabBW34 dose metric, or the total amount TCE metabolized per unit adjusted body weight, was used for all three studies. Thise dose metric was selected because for these endpoints there is insufficient information for site-specific or mechanism-specific determinations of an appropriate

dose-metric, however in general TCE toxicity is associated with metabolites rather than the parent compound. LOAELs were used as PODs for all studies, and none were BMD modeled. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling and dose metric selection.

Differences from standard UF values are explained below:
(Arito et al., 1994) was assigned $UF_S = 3$ (instead of 10) despite being only a 6 week study because effects observed at 6 weeks exposure were only minimally different than effects at 2 weeks (differences observed post-exposure).
(Ruijten et al., 1991) was assigned $UF_S = 1$ because the data was based on a mean of 16 years of human exposure. $UF_L = 3$ (instead of 10) due to the observed effect being an early marker and representing a minimal degree of change.

EPA did not select (Isaacson et al., 1990), demonstrating demyelination of the hippocampus, to represent the neurotoxicity hazard because dosing during the study was not continuous and the resulting POD was subject to a large cumulative uncertainty factor (1000). (Arito et al., 1994) and (Ruijten et al., 1991) were both considered for use in quantitative risk estimation as they were relatively well-conducted studies examining independent endpoints within the hazard of neurological effects.

Immunotoxicity

-- *Thymus Effects / Autoimmunity*

(Keil et al., 2009) exposed B6C3F1 mice (10/group), a standard test strain not genetically prone to develop autoimmune disease, to TCE via drinking water for 27 or 30 weeks at concentrations in water of 0, 1.4, or 14 ppm (0.35 or 3.5 mg/kg-bw/day). The study reported a significant decrease in thymus weight concentrations and thymic cellularity as well as an increase in autoantibodies to ssDNA and dsDNA. A LOAEL of 0.35 mg/kg-bw/day was identified as the POD for the thymic and autoimmune effects (U.S. EPA, 2011e).

-- *Autoimmunity*

(Kaneko et al., 2000) exposed auto-immune prone mice (5/group) to TCE via inhalation at concentrations of 0, 500, 1,000, or 2,000 ppm for 4 hrs/day, 6 days/week, for 8 weeks. At concentrations ≥ 500 ppm, mice exhibited dose-related liver inflammation, splenomegaly and hyperplasia of lymphatic follicles. Immunoblastic cell formation in lymphatic follicles was observed in mice treated with 1,000 ppm TCE. The LOAEL of 70 ppm (adjusted for continuous 24hr exposure) was identified for these effects (U.S. EPA, 2011e).

-- *Immunosuppression*

In (Sanders et al., 1982), male and female CD-1 mice (7-25/group) were given TCE in drinking water concentrations of 0, 0.1, 1.0, 2.5, or 5.0 mg/mL (0, 18, 217, 393 or 660 mg/kg-bw/day) for 4 or 6 months. Female mice showed decreased humoral immunity at 2.5 and 5 mg/mL (393 or 660 mg/kg-bw/day), whereas cell-mediated immunity and bone marrow stem cell colonization decreased at all four concentrations. Male mice were relatively unaffected after both 4 and 6 months of exposure. A LOAEL of 18 mg/kg-bw/day was identified as the POD for immunosuppressive effects (U.S. EPA, 2011e).

Another study that was previously discussed for liver and kidney effects (Woolhiser et al., 2006) also reported immunosuppressive effects. Sprague-Dawley female rats (16/group) were treated with 0, 100, 300 or 1,000 ppm TCE for 6 hrs/day, 5 days/week for 4 weeks. Four days prior to study termination, the rats were immunized with sheep red blood cells (SRBC), and within 24 hrs following the last exposure to TCE, a plaque-forming cell (PFC) assay was conducted to determine effects on splenic anti-SRBC IgM response. At 1,000 ppm, rats demonstrated a 64% decrease in the PFC assay response.

A $BMDL_{1SD}$ of 24.9 ppm was identified for this immunosuppressive effect (U.S. EPA, 2011e).

Table 3-11: Dose-response analysis of selected studies considered for evaluation of immune effects

Target Organ System	Species	Duration	POD Type [1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Immune system	Mouse (female)	27-30 weeks	LOAEL = 0.35 mg/kg-bw/day	Decrease in thymus weight and thymus cellularity	TotMetab BW34	0.092	0.033	0.049	0.048	UF_S=1; UF_A= 3; UF_H=3; UF_L=10; *Total UF=100* [4]	(Keil et al., 2009)	High (1.6)
	Mouse (female)	**27-30 weeks**	**LOAEL = 0.35 mg/kg-bw/day**	**Autoimmunity (increased anti- dsDNA and ssDNA antibodies)**	**TotMetab BW34**	**0.092**	**0.033**	**0.049**	**0.048**	**UF_S=1; UF_A= 3; UF_H=3; UF_L=3; *Total UF=30*** [4]	**(Keil et al., 2009)**	**High (1.6)**
	Mouse (males; auto-immune prone strain)	4 hrs/day, 6 days/week for 8 weeks	LOAEL = 70 ppm	Autoimmunity (changes in immunoreactive organs)	TotMetab BW34	97	37	44	42	UF_S=10; UF_A= 3; UF_H=1; UF_L=10; *Total UF=300*	(Kaneko et al., 2000)	High (1.5)
	Mouse (female)	**16 or 24 weeks (4 or 6 months)**	**LOAEL = 18 mg/kg-bw/day**	**Immuno-suppression**	**TotMetab BW34**	**4.8**	**1.7**	**2.5**	**2.5**	**UF_S=1; UF_A= 3; UF_H=3; UF_L=10; *Total UF=100***	**(Sanders et al., 1982)**	**High (1.4)**
	Rat (female)	6 hrs/day, 5 days/week for 4 weeks	$BMDL_{1SD}$= 24.9 ppm	Immuno-suppression	TotMetab BW34	29	11	14	14	UF_S=10; UF_A= 3; UF_H=3; UF_L=1; *Total UF=100*	(Woolhiser et al., 2006)	High (1.1)

[1] POD type can be NOAEL, LOAEL, or BMDL. The IRIS program adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric.
[4] Two different effects were reported by Keil et al (2009): decreased thymic weight and cellularity and autoimmunity. A total UF of 100 was used for the thymus toxicity, whereas a total UF of 30 was used for the autoimmune effects. The TCE IRIS assessment allocated different LOAEL-to-NOAEL uncertainty factors (UF_L) based on the severity of the effects, which resulted in different total UF (U.S. EPA, 2011e).

Table 3-11 presents the derived PODs from all studies considered for dose-response analysis. These studies covered the endpoints of thyroid effects, autoimmunity, and immunosuppression. The TotMetabBW34 dose metric, or the total amount TCE metabolized per unit adjusted body weight, was used for all three studies. This dose metric was selected because for these endpoints there is insufficient information for site-specific or mechanism-specific determinations of an appropriate dose-metric, however in general TCE toxicity is associated with metabolites rather than the parent compound. LOAELs were used as PODs for all studies except (Woolhiser et al., 2006), which was BMD modeled with a BMR of 1 SD because it was unclear what should constitute the cutoff point for a minimal, biologically significant change. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling, dose metric selection, and BMR selection.

Differences from standard UF values are explained below:
(Keil et al., 2009) was assigned UF_L = 3 (instead of 10) due to the observed effect being considered an early, subclinical or pre-clinical early marker of disease.

Decreased thymus weight and cellularity as observed in (Keil et al., 2009) was not considered for use in risk estimation because EPA determined that this effect is insufficiently adverse compared to the other endpoints. Of note, elimination of this endpoint and corresponding change in total UF represents a change from the 2014 TSCA Work Plan Chemical Risk Assessment (U.S. EPA, 2014b). The data from (Keil et al., 2009) was selected to represent autoimmunity however, because the study was of longer duration than

(Kaneko et al., 2000) with a smaller cumulative uncertainty factor. (Sanders et al., 1982) was selected to represent immunosuppression because the study was of a much longer duration than (Woolhiser et al., 2006).

Reproductive toxicity

-- *Male Reproductive Effects*

(Chia et al., 1996) examined a cohort of 85 workers in an electronics factory. The workers provided urine, blood, and sperm samples. The mean urine TCA level was 22.4 mg/g creatinine (range: 0.8–136.4 mg/g creatinine). In addition, 12 workers provided personal 8-hr air samples, which resulted in a mean TCE exposure of 29.6 ppm (range: 9–131 ppm). There were no controls in the study. Males experienced decreased percentage of normal sperm morphology and hyperzoospermia. A $BMDL_{10}$ of 1.4 ppm was identified as the POD for these effects (U.S. EPA, 2011e).

(Xu et al., 2004) exposed male CD-1 mice (27/group) to TCE at concentration of 0 or 1,000 ppm for 6 hrs/day, 5 days/week for 6 weeks. Inhalation exposure to TCE did not result in altered body weight, testis and epididymis weights, sperm count, or sperm morphology or motility. Percentages of acrosome-intact sperm populations were similar between treated and control animals. However, decreased *in vitro* sperm-oocyte binding and reduced *in vivo* fertilization were observed in TCE-treated male mice. A LOAEL of 180 ppm (adjusted for continuous 24hr exposure) was identified as the POD for these effects (U.S. EPA, 2011e).

(Kumar et al., 2000) and (Kumar et al., 2001) exposed male Wistar rats by inhalation at concentrations of 0 or 376 ppm TCE. Both study protocols exposed rats for 4 hrs/day, 5 days/week, but had variable duration scenarios. For instance, (Kumar et al., 2000) treated rats for the following exposure durations: 2 weeks (to observe the effect on the epididymal sperm maturation phase), 10 weeks (to observe the effect on the entire spermatogenic cycle), 5 weeks with 2 weeks of rest (to observe the effect on primary spermatocytes differentiation to sperm), 8 weeks with 5 weeks of rest (to observe effects on an intermediate stage of spermatogenesis), or 10 weeks with 8 weeks of rest (to observe the effect on spermatogonial differentiation to sperm). (Kumar et al., 2001) exposed rats for either 12 or 24 weeks.

(Kumar et al., 2000) reported altered testicular histopathology, increased sperm abnormalities, and significantly increased pre- and/or postimplantation loss in litters in the groups with 2 or 10 weeks of exposure, or 5 weeks of exposure with 2 of weeks rest. Multiple sperm effects were observed in another study by Kumar (2001). After 12 weeks of TCE exposure, rats exhibited decreased number of spermatogenic cells in the seminiferous tubules, fewer spermatids as compared to controls, and the presence of necrotic spermatogenic cells. Following 24 weeks of exposure, male rates showed reduced testes weights and epididymal sperm count and motility, testicular atrophy, smaller tubules, hyperplastic Leydig cells, and a lack of spermatocytes and spermatids in the tubules. Testicular marker enzymes were altered at both 12 and 24 weeks of exposure. A LOAEL of 45 ppm was identified as the POD for the sperm and male reproductive effects reported in both studies (U.S. EPA, 2011e).

(Kan et al., 2007) also provided evidence for the damage to the epididymis epithelium and sperm. CD-1 male mice (4/group) were exposure by inhalation to 0 or 1,000-ppm TCE for 6 hrs/day, 5 days/week for 1 to 4 weeks. As early as 1 week after TCE exposure, exposed mice showed degeneration and sloughing of epithelial cells. These effects increased in severity at 4 weeks of exposure. A LOAEL of 180 ppm (adjusted for continuous 24hr exposure) was identified as a POD for the effects in the epididymis epithelium.

-- *Female Reproductive Effects*

(Narotsky et al., 1995) administered TCE to F344 timed-pregnant rats (8-12 dams/group) by gavage. Dams were exposed to TCE doses of 0, 10.1, 32, 101, 320, 475, 633, 844 or 1125 mg/kg-bw/day during gestational days (GD) 6 to 15. The study was a prequel to a complicated protocol with other chemicals in a mixture study. Delayed parturition was observed at ≥475 mg/kg- bw/day. The LOAEL for female reproductive effects was 475 mg/kg-bw/day (U.S. EPA, 2011e).

-- *Diminished Reproductive Behavior*

George et al. (1986) administered TCE to both male and female F344 rats (20 each treated, 40 each controls) in feed with estimated doses of 0, 72, 186, or 389 mg/kg-bw/day. Breeders were exposed for one week premating and then for 13 weeks while cohabitating. Pregnant females were subsequently exposed throughout gestation (an additional 4 weeks). Copulation was reduced equally following either exposed males or exposed females cohabitating with control mates (highest dose only examined). This corresponded with a dose-responsive decrease in the number of litters produced per breeding pair and the number of live pups per litter.

Table 3-12: Dose-response analysis of selected studies considered for evaluation of reproductive effects

Target Organ System	Species	Duration	POD Type[1] (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs) [2]	Reference	Data Quality [3]
Reproductive system	**Human (male)**	**Measured values after an 8-hr work shift; mean 5.1 years on the job**	**$BMDL_{10}$ = 1.4 ppm**	**Hyper-zoospermia**	**TotMetab BW34**	**1.4**	**0.5**	**0.74**	**0.73**	**UF_S=10; UF_A= 1; UF_H=3; UF_L=1; *Total UF=30***	**(Chia et al., 1996)**	**Medium (1.8)**
	Rat (male)	4 hrs/day, 5 days/week, 2-10 weeks exposed, 2-8 weeks unexposed	LOAEL = 45 ppm	Sperm effects and male reproductive tract effects	TotMetab BW34	32	13	16	16	UF_S=10; UF_A= 3; UF_H=3; UF_L=10; *Total UF=1000*	(Kumar et al., 2000)	Medium (1.7)
		4 hrs/day, 5 days/week for 12 or 24 weeks									(Kumar et al., 2001)	High (1.4)
	Mouse (male)	6 hrs/day, 5 days/week for 1-4 weeks	LOAEL = 180 ppm	Effects on epididymis epithelium	TotMetab BW34	190	67	80	73	UF_S=10; UF_A= 3; UF_H=3; UF_L=10; *Total UF=1000*	(Kan et al., 2007)	Medium (2)*
	Mouse (male)	6 hrs/day, 5 days/week for 6 weeks	LOAEL = 180 ppm	Sperm effects (decreased in vitro sperm-oocyte binding and *in vivo* fertilization)	TotMetab BW34	190	67	80	73	UF_S=10; UF_A= 3; UF_H=3; UF_L=10; *Total UF=1000*	(Xu et al., 2004)	High (1.4)
	Rat (female dams)	**9 days (during gestational days 6 to 15)**	**LOAEL = 475 mg/kg-bw/day**	**Delayed parturition**	**TotMetab BW34**	**98**	**37**	**47**	**44**	**UF_S=1; UF_A= 3; UF_H=3; UF_L=10; *Total UF=100***	**(Narotsky et al., 1995)**	**High (1.3)**
	Rat (male/ female)	Breeders exposed 1 week premating and then for 13 weeks cohabitating	LOAEL = 389 mg/kg-bw/day	Decreased copulation; reduced numbers of live litters/pair and pups/litter	TotMetab BW34	204	71	85	77	UF_S=1; UF_A= 3; UF_H=3; UF_L=10; UF_D=1 *Total UF=100*	(George et al., 1986)	High (1.1)

[1] POD type can be NOAEL, LOAEL, or BMDL. The IRIS program adjusted all values to continuous exposure.
[2] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF.
[3] See [*Data Quality Evaluation of Human Health Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500]* for full evaluation by metric. *Kan 2007 was downgraded from a High, with calculated score = 1.6.

Table 3-12 presents the derived PODs from all studies considered for dose-response analysis. The majority of studies identified effects indicative of male reproductive toxicity, with one study demonstrating female reproductive toxicity. The TotMetabBW34 dose metric, or the total amount TCE metabolized per unit adjusted body weight, was used for all three studies. This dose metric was selected because for these endpoints there is insufficient information for site-specific or mechanism-specific determinations of an appropriate dose-metric, however in general TCE toxicity is associated with metabolites rather than the parent compound. For (Chia et al., 1996), the 2011 IRIS Assessment (U.S. EPA, 2011e) notes some additional uncertainty in the dose estimate because exposure groups were defined by ranges and exposure was estimated by conversion of urinary TCA. LOAELs were used as PODs for all studies except (Chia et al., 1996), which was BMD modeled with a standard BMR of 10% extra risk. The 2011 IRIS Assessment (U.S. EPA, 2011e) indicates some uncertainty in the biological signficance of this BMR because the study used a lower cutoff to define hyperzoospermia than other studies. See Section 3.2.2.1 and (U.S. EPA, 2011e) for more details on TCE PBPK modeling, dose metric selection, and BMR selection.

For male reproductive toxicity, (Chia et al., 1996) was selected over the other studies because it was a human study over a mean 5.1 year period compared to the other studies which were in mice and all for only a few weeks except for (Kumar et al., 2001). Additionally, (Chia et al., 1996) only has a cumulative uncertainty factor of 30, compared to 1000 for the other three studies. (Narotsky et al., 1995) received a High in data quality evaluation and was deemed suitable for quantitative assessment of female reproductive toxicity based on delayed parturition (giving birth). While (George et al., 1986) received a High in data quality evaluation, it is unclear whether the observed effects are a result of true reproductive toxicity or merely behavioral changes (i.e. unsuccessful copulation vs. reduced libido). Effects on copulation are also likely downstream of any specific male or female reproductive endpoints, which have more sensitive PODs than (George et al., 1986). Therefore, the POD for reduced copulation was not selected to represent the reproductive toxicity hazard.

Developmental toxicity

As described above in Section 3.2.5.3.1, developmental effects may result from single as well as repeated exposures at a developmentally critical period; therefore the same endpoints are relevant for both acute and chronic exposure scenarios. The only difference between acute and chronic exposure scenarios in evaluating developmental toxicity is the benchmark MOE for (Fredriksson et al., 1993). The subchronic-to-chronic UF_S = 3 for chronic exposure, because the study only exposed pups during postnatal days 10-16, suggesting that exposure during a longer period of development may have exacerbated the observed effects (UF_S would not = 10 because neurological development only occurs over a portion of a lifetime). This results in a cumulative UF and benchmark MOE of 300. See Section 3.2.5.3.1 for a detailed description of the developmental toxicity endpoints.

3.2.5.3.3 Cancer POD for Lifetime Exposures

EPA utilized linear low-dose extrapolation for derivation of PODs accounting for all three cancer types. Regarding low-dose extrapolation, a key consideration in determining what extrapolation approach to use is the mode(s) of action. However, mode-of-action data are lacking or limited for each of the cancer responses associated with TCE exposure, with the exception of the kidney tumors (see Section 3.2.4.2.2). For the other TCE-induced cancers, the mode(s) of action is unknown. When the mode(s) of action is identified as genotoxic or cannot be clearly defined, EPA generally uses a linear approach to estimate low-dose risk (U.S. EPA, 2005), based on the following general principles:

1) A chemical's carcinogenic effects may act additively to ongoing biological processes, given that diverse human populations are already exposed to other agents and have substantial background incidences of various cancers.

2) A broadening of the dose-response curve (i.e., less rapid fall-off of response with decreasing dose) in diverse human populations and, accordingly, a greater potential for risks from low-dose exposures (Lutz et al., 2005; Zeise et al., 1987) is expected for two reasons: First, even if there is a threshold concentration for effects at the cellular level, that threshold is expected to differ across individuals. Second, greater variability in response to exposures would be anticipated in heterogeneous populations than in inbred laboratory species under controlled conditions (due to, e.g., genetic variability, disease status, age, nutrition, and smoking status).

3) The general use of linear extrapolation provides reasonable upper-bound estimates that are believed to be health-protective (U.S. EPA, 2005) and also provides consistency across assessments.

Dose-response analysis of kidney cancer utilized ABioactDCVCBW34, or the amount of DCVC bioactivated in the kidney per unit adjusted body weight, for the same rationale as described above for

kidney non-cancer effects. Dose-response modeling for kidney cancer from Charbotel et al. (2006) was performed by linear regression weighted by the inverse of variances for RR estimates. Consistent with EPA's Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), the same data and methodology were also used to estimate the exposure level (ECx: ―effective concentration corresponding to an extra risk of x%) and the associated 95% lower confidence limit of the effective concentration corresponding to an extra risk of 1% (LECx [lowest effective concentration], x = 0.01). A 1% extra risk level is commonly used for the determination of the POD for epidemiological data. Use of a 1% extra risk level for these data is supported by the fact that, based on the actuarial program, the risk ratio (i.e., Rx/Ro) for an extra risk of 1% for kidney cancer incidence is 1.9, which is in the range of the ORs reported by Charbotel et al (ORs range from 1.16 - 2.16 across exposure tertiles). Thus, 1% extra risk was selected for determination of the POD, and, consistent with *EPA's* Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), the LEC value corresponding to that risk level was used as the actual POD. For more details, see Section 5.2.2 in the 2011 IRIS Assessment (U.S. EPA, 2011e). Based on the results of the meta-analysis (Section 3.2.4.2.1 and Appendix H) confirming a positive association between TCE exposure and all three cancer sites, the derived PODs will remain the same as for (U.S. EPA, 2011e) and (U.S. EPA, 2014b).

The inhalation unit risk (IUR) for TCE is defined as a plausible upper bound lifetime extra risk of cancer from chronic inhalation of TCE per unit of air concentration. The estimate of the inhalation unit risk for TCE is 2.20×10^{-2} per ppm (2×10^{-2} per ppm [4×10^{-6} per $\mu g/m^3$]) rounded to one significant figure), based on human kidney cancer risks reported by Charbotel et al. (2006) and adjusted 4-fold upward for potential additional risk for NHL and liver cancer. This estimate is based on High-quality human data, thus avoiding the uncertainties inherent in interspecies extrapolation. This value is supported by inhalation unit risk estimates demonstrating multisite carcinogenicity in several rodent bioassays, the most sensitive of which range from 1×10^{-2} to 2×10^{-1} per ppm [2×10^{-6} to 3×10^{-5} per $\mu g/m^3$].

The IUR from Charbotel et al. (2006) (calculated as 5.49×10^{-3} per ppm) was adjusted by a factor of four to account for estimating risk to all three cancer types combined (i.e., lifetime extra risk for developing any of the three types of cancer) versus the extra risk for kidney cancer alone. Although only the Charbotel et al. (2006) study was found adequate for direct estimation of inhalation unit risks, the available epidemiologic data provide sufficient information for estimating the *relative* potency of TCE across tumor sites. Section 5.2.2 of the 2011 IRIS Assessment (U.S. EPA, 2011e) describes the process for this adjustment. In short, extra lifetime cancer risks were summed across the three cancer types and the ratio of the sum of the extra risks to the extra risk for kidney alone was derived. EPA calculated this ratio using two sets of data: the summary RR estimates from the 2011 meta-analyses for NHL, kidney cancer, and liver cancer, and the SIR estimates for all three cancer types from the Raaschou-Nielsen et al. (2003) study. The value for the ratio of the sum of the extra risks to the extra risk for RCC alone was 3.28 from the first calculation (using meta-analysis results) and 4.36 from the second calculation (using Raaschou-Nielsen et al. data). The geometric and arithmetic mean of these two values is 3.8, and EPA decided to round up to 4 based on the imprecision of the adjustment factor.

The oral slope factor (OSF) for TCE is defined as a plausible upper bound lifetime extra risk of cancer from chronic ingestion of TCE per mg/kg/day oral dose. The estimate of the oral slope factor is 4.64×10^{-2} per mg/kg/day (5×10^{-2} per mg/kg/day rounded to one significant figure), resulting from PBPK model-based route-to-route extrapolation of the inhalation unit risk estimate based on the human kidney cancer risks reported in Charbotel et al. (2006) and adjusted 5-fold upward for potential risk for NHL and liver cancer. For this adjustment, individual IUR estimates were first obtained for each site based on the ratios of extra risk relative to kidney. Those site-specific IUR estimates were then

extrapolated to the equivalent OSFs using site-specific dose metrics,[18] and those individual OSFs were summed to obtain a ratio of 5.0 relative to kidney cancer alone. Uncertainty in the PBPK model-based route-to-route extrapolation is relatively low, however variability stemming from the requirement of using distinct dose-metrics for the different target tissues resulted in a larger 5-fold adjustment, as opposed to the 4-fold adjustment calculated for the IUR. Extrapolation using different dose-metrics yielded expected population mean risks within about a two-fold range, and, for any particular dose-metric, the 95% CI for the extrapolated population mean risks for each site spanned a range of no more than about threefold. The resulting combined OSF value is supported by oral slope factor estimates from multiple rodent bioassays, the most sensitive of which range from 3×10^{-2} to 3×10^{-1} per mg/kg/day.

EPA decided not to use the IUR or OSF to calculate the theoretical cancer risk associated with a single (acute) exposure to TCE. NRC (2001) published methodology for extrapolating cancer risks from chronic to short-term exposures to mutagenic carcinogens, however these methods were published with the caveat that extrapolation of lifetime theoretical excess cancer risks to single exposures has great uncertainties. Thus, this risk evaluation plan risk assessment for TCE does not estimate excess cancer risks for acute exposures because the relationship between a single short-term exposure to TCE and the induction of cancer in humans has not been established in the current scientific literature. Risk estimates for cancer will be based on lifetime exposure durations, represented as Lifetime Average Daily Concentration/Dose (LADC/LADD).

3.2.5.4 Selected PODs for Human Health Hazard Domains

Table 3-13 and Table 3-14 list the studies and corresponding HECs, HEDs, and UFs that EPA is using in the TCE Risk Evaluation following acute and chronic exposure. Table 3-15 provides the cancer PODs for evaluating lifetime exposure. Key studies in Table 3-13 and Table 3-14 are briefly described in Section 3.2.5.1. Presenting PODs for the HEC/HED_{50} and HEC/HED_{99} values is intended to provide a sense of the difference between the median and 99% confidence bound for the combined uncertainty and variability. Calculations of $HEC_{50/99}$ and $HED_{50/99}$ ratios generally showed a 2-3 fold difference for the various studies described in Section 3.2.5.3. The exception was for studies reporting kidney effects, which showed high $HEC_{50/99}$ and $HED_{50/99}$ ratios (7 to 10-fold) due to larger uncertainty in the rodent internal dose estimates for the GSH metabolism dose metrics (e.g., ABioActDCVCBW34) (U.S. EPA, 2011e) and greater influence of human variability. Confidence in these metrics was lower for mouse data due to an absence of GSD-specific in vivo data, however uncertainty was similar as to other metrics for rat and human data (U.S. EPA, 2011e). The HEC/HED_{99} values represent the PODs that are expected to be protective of sensitive subpopulations, accounting for the majority of identified toxicokinetic human variability.

[18] Kidney: ABioactDCVCBW34; NHL: TotMetabBW34; Liver: AMetLiv1BW34

2520 **Table 3-13: Dose-response analysis of selected studies considered for acute exposure scenarios**

Target Organ/ System	Species	Duration	POD Type (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs)	Reference	Data Quality
Develop-mental Effects	Rat (female)	Gestational days 6 to 15	$BMDL_{01}$= 32.2 mg/kg-bw/day	Increased resorptions	TotMetab BW34	57	23	29	28	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; Total UF=10	(Narotsky et al., 1995)	High
	Rat (female)	22 days throughout gestation (gestational days 0 to 22)	$BMDL_{01}$ = 0.0207 mg/kg-bw/day	Congenital heart defects	TotOx Metab BW34	0.012	0.0037	0.0058	0.0052	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; Total UF=10	(Johnson et al., 2003)	Medium
	Rat (male pups)	Postnatal days 10 to 16	LOAEL = 50 mg/kg-bw/day	Decreased rearing activity	TotMetab BW34	8	3	4.2	4.1	UF_S=1; UF_A= 3; UF_H=3; UF_L=10; Total UF=100	(Fredriksson et al., 1993)	Medium
Immune System	Rat (female)	3hr/day, single dose; followed by respiratory infection	$BMDL_{01}$ = 13.9 ppm	Immuno-suppression	N/A[1]	N/A[1]	1.74 [1]	N/A[1]	2.74 [1,2]	UF_S=1; UF_A= 3; UF_H=10; UF_L=1; *Total UF=30*	(Selgrade and Gilmour, 2010)	High

[1] Data from (Selgrade and Gilmour, 2010) was not subject to PBPK modeling due to uncertainty concerning the most appropriate dose metric. The BMDL value adjusted for a 24hr exposure will be used as the POD for occupational risk estimates, while the 3hr value will be used for consumer risk estimates. This value is presented in the HEC_{99} column but does not represent any particular percentile since it was not PBPK-modeled.
[2] A dermal HED was obtained through route-to-route extrapolation using breathing rate and body weight data on male CD-1 mice (insufficient female data was reasonably available) from (U.S. EPA, 1988) and allometric scaling based on (U.S. EPA, 2011d) using a dosimetric adjustment factor of 0.14 for mice.

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Table 3-14: Dose-response analysis of selected studies considered for chronic exposure scenarios

Target Organ System	Species	Duration	POD Type (applied dose)	Effect	Dose Metric	HEC_{50} (ppm)	HEC_{99} (ppm)	HED_{50} (mg/kg)	HED_{99} (mg/kg)	Uncertainty Factors (UFs)	Reference	Data Quality
Liver	Mouse (male)	Continuous and intermittent exposures, variable time periods for 30-120 days	$BMDL_{10}$= 21.6 ppm	Increased liver/body weight ratio and cytotoxicity/ hypertrophy	AMetLiv1 BW34	25	9.1	9.0	7.9	UFS=1; UFA= 3; UFH=3; UFL=1; *Total UF=10*	(Kjellstrand et al., 1983)	Medium
Kidney	Rat (male) - Oral	4-5 days/week for 52 weeks	$BMDL_{10}$ = 34 mg/kg-bw/day	Pathology changes in renal tubule	ABioact DCVCBW34	0.19	0.025	0.15	0.015	UFS=1; UFA= 3; UFH=3; UFL=1; *Total UF=10*	(Maltoni et al., 1986)	Medium
Nervous System	Rat (male)	8 hrs/day, 5 days/weeks for 6 weeks	LOAEL = 12 ppm	Significant decreases in wakefulness	TotMetab BW34	13	4.8	6.6	6.5	UFS=3; UFA= 3; UFH=3; UFL=10; *Total UF=300*	(Arito et al., 1994)	Medium
	Human (both sexes)	Mean of 16 years	LOAEL = 14 ppm	Trigeminal nerve effects (increased latency in masseter reflex)	TotMetab BW34	14	5.3	7.4	7.3	UFS=1; UFA= 1; UFH=3; UFL=3; *Total UF=10*	(Ruijten et al., 1991)	Medium
Immune System	Mouse (female)	27-30 weeks	LOAEL = 0.35 mg/kg-bw/day	Autoimmunity (increased anti-dsDNA and ssDNA antibodies)	TotMetab BW34	0.092	0.033	0.049	0.048	UFS=1; UFA= 3; UFH=3; UFL=3; *Total UF=30*	(Keil et al., 2009)	High
	Mouse (female)	16 or 24 weeks (4 or 6 months)	LOAEL = 18 mg/kg-bw/day	Immunosuppression	TotMetab BW34	4.8	1.7	2.5	2.5	UFS=1; UFA= 3; UFH=3; UFL=10; *Total UF=100*	(Sanders et al., 1982)	High
Repro-ductive System	Human (male)	Measured values after an 8-hr work shift; mean 5.1 years on the job	BMDL10 = 1.4 ppm	Decreased normal sperm morphology and hyperzoospermia	TotMetab BW34	1.4	0.5	0.74	0.73	UFS=10; UFA= 1; UFH=3; UFL=1; Total UF=30	(Chia et al., 1996)	Medium
	Rat (female dams)	9 days (during gestational days 6-15)	LOAEL = 475 mg/kg-bw/day	Delayed parturition	TotMetab BW34	98	37	47	44	UFS=1; UFA= 3; UFH=3; UFL=10; Total UF=100	(Narotsky et al., 1995)	High
Develop-mental Effects	Rat (female)	Gestational days 6 to 15	$BMDL_{01}$= 32.2 mg/kg-bw/day	Increased resorptions	TotMetab BW34	57	23	29	28	UFS=1; UFA= 3; UFH=3; UFL=1; Total UF=10	(Narotsky et al., 1995)	High
	Rat (female)	22 days (gestational days 0-22)	$BMDL_{01}$ = 0.0207 mg/kg-bw/day	Congenital heart defects	TotOx Metab BW34	0.012	0.0037	0.0058	0.0052	UFS=1; UFA= 3; UFH=3; UFL=1; Total UF=10	(Johnson et al., 2003)	Medium
	Rat (male pups)	Postnatal days 10-16	LOAEL = 50 mg/kg-bw/day	Decreased rearing activity	TotMetab BW34	8	3	4.2	4.1	UFS=3; UFA= 3; UFH=3; UFL=10; Total UF=300	(Fredriksson et al., 1993)	Medium

Table 3-15: Cancer Points of Departure for Lifetime Exposure Scenarios

POD Type	Oral Slope Factor	Inhalation Unit Risk	Extra Risk Benchmark
POD (extra risk per dose/concentration)	0.0464 per mg/kg	0.022 per ppm	1×10^{-4}

As stated in Section 3.2.5.3.3, these PODs represent the plausible upper bound lifetime extra risk of cancer per unit dose or air concentration. The linear non-threshold assumption underlying the derivation of these values is appropriate based on the mutagenic mode of action for kidney cancer (with an unclear mode of action for the other two cancer types). The PODs are derived from a single High quality kidney cancer study (Charbotel et al., 2006) and the combined estimates account for the additional relative contribution from the other two cancers.

For TCE, EPA, consistent with OSHA (878 F.2d 389 (D.C. Cir. 1989) and 2016 NIOSH guidance (Whittaker et al., 2016), 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 1×10^{-4} is not a bright line and EPA has discretion to find unreasonable risks based on other benchmarks as appropriate based on analysis. It is important to note that exposure related considerations (duration, magnitude, population exposed) can affect EPA's estimates of the excess lifetime cancer risk (ELCR). Cancer assessment is only applicable to evaluation of occupational exposure scenarios, because consumer exposures were only evaluated as acute scenarios (Section 2.3.2.2).

3.2.6 Assumptions and Key Sources of Uncertainty for Human Health Hazard

3.2.6.1 Confidence in Hazard Identification and Weight of Evidence

There is high confidence in the database for human health hazard. All 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 study for each identified endpoint from among a broad selection of studies, taking into account factors such as data quality evaluation score, species, exposure duration, dose range, cumulative uncertainty factor, and relevance. The only identified study that examined developmental immunotoxicity (Peden-Adams et al., 2006) scored a Low in data evaluation and a POD could not be sufficiently derived.

EPA has high confidence in the overall weight of scientific evidence. EPA did not identify any information that would question the previous WOE regarding the evaluation of liver, kidney, neurological, immunological, reproductive toxicity, and developmental toxicity (other than cardiac malformations). For cancer, EPA performed an updated meta-analysis that found positive statistical associations between human TCE exposure and cancer of kidney, liver, and NHL types, in agreement with the previous meta-analyses performed in 2011 (Appendix C, (U.S. EPA, 2011b). For congenital heart defects, EPA performed a thorough WOE assessment (Appendix G.2), examining all pertinent studies in the reasonably available literature. While some uncertainty remains in the dose-response analysis of the (Johnson et al., 2003) study and the resulting POD, there is medium confidence in the qualitative relevance of the endpoint to human toxicity based on the results of the WOE.

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.

If a BMDL is used as the POD, there are uncertainties regarding the appropriate dose-response model to apply to the data, but these should be minimal if the modeling is in the observable range of the data. There are also uncertainties about what BMR to use to best approximate the desired exposure level (i.e. threshold, see above). For continuous endpoints, in particular, it is often difficult to identify the level of change that constitutes the threshold for an adverse effect. While a 1% BMR is justified for many of the PODs derived in this assessment based on the severity of the endpoint, it can potentially amplify BMD model and parameter uncertainty. This is especially of concern for endpoints with greater uncertainties in the dose-response assessment such as the congenital heart defects endpoint from (Johnson et al., 2003), however a reanalysis of the BMR selection for this endpoint concluded that the 1% BMR was in fact most appropriate (Section 3.2.5.3.1).

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, 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.

The PBPK-based POD estimates include uncertainties about the appropriate dose-metric for each effect, although there was better information about relevant dose-metrics for some effects than for others (see Section 3.2.5.3). The 2011 TCE IRIS Assessment determined that the PBPK model was most reliable for dose metrics of oxidative metabolism flux .There remains substantial uncertainty in the extrapolation of GSH conjugation from mice to humans due to limitations in the reasonably available data. This dose metric is specifically applicable to kidney endpoints, which are believed to result from renal bioactivation through GSH conjugation. In this manner, the HEC/HED_{99} values (which account for both modeling uncertainty and interspecies/intraspecies toxicokinetic variability) may potentially overestimate kidney toxicity for a proportion of the population, however use of these values are expected to sufficiently account for the majority of human toxicokinetic variability, including increased biological susceptibility (see Section 3.2.5.2). Of note, there was significantly less uncertainty for extrapolation of rat GSH conjugation data, which was used for the selected kidney PODs, compared to data from mice. Despite any limitations of the model, overall uncertainty for the selected PODs is reduced by the use of a PBPK model. Use of the PBPK model resulted in data-derived HEC/HED_{99}

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 for both interspecies and intraspecies toxicokinetic variability. Data-derived values are always preferred to default uncertainty adjustments and improve confidence in the adjusted PODs.

3.2.6.3 Cancer Dose Response

Potential sources of uncertainty associated with Charbotel et al. (2006) include the modest sample size of the study and localized population (86 kidney cancer cases, 37 associated with TCE exposure from a specific region in France), the retrospective estimation of TCE in study subjects, and potential confounding effects from exposure to other degreasing agents. These uncertainties do not significantly affect confidence in the study results because Charbotel et al. (2006) was a well conducted, High quality study that used a comprehensive exposure assessment with a detailed occupational questionnaire and sensitivity and regression analyses found no statistical effect on the cancer POD from a sensitivity analysis adjusting for exposure to other chemicals (U.S. EPA, 2011e).

The two major sources of uncertainty in quantitative cancer risk estimates are generally interspecies extrapolation and high-dose to low-dose extrapolation. The unit risk estimate for kidney cancer incidence derived from the Charbotel et al. (2006) results is not subject to interspecies uncertainty because it is based on human data. A major uncertainty remains in the extrapolation from occupational exposures to lower environmental exposures. There was some evidence of a contribution to increased kidney cancer risk from peak exposures; however, there remained an apparent dose-response relationship for RCC risk with increasing cumulative exposure without peaks, and the odds ratio (OR) for exposure with peaks compared to exposure without peaks was not significantly elevated (Charbotel et al., 2006) Although the actual exposure-response relationship at low exposure levels is unknown, the conclusion that a mutagenic mode of action is operative for TCE-induced kidney tumors supports the linear low-dose extrapolation that was used (U.S. EPA, 2005). The weight of evidence also supports involvement of processes of cytotoxicity and regenerative proliferation in the carcinogenicity of TCE, although not with the extent of support as for a mutagenic mode of action. In particular, data linking TCE-induced proliferation to increased mutation or clonal expansion are lacking, as are data informing the quantitative contribution of cytotoxicity. Because any possible involvement of a cytotoxicity mode of action would be additional to mutagenicity, the dose-response relationship would nonetheless be expected to be linear at low doses. Therefore, the additional involvement of a cytotoxicity mode of action does not provide evidence against the use of linear extrapolation from the POD.

The upward adjustment of the cancer PODs based on additional contributions from liver and NHL cancer was based on peer-reviewed methodology as explained in the 2011 IRIS Assessment (U.S. EPA, 2011e). This approach is reasonable, however it is unknown whether these statistical methods resemble the true combined extra risk from these three cancers. Additionally, the IUR adjustment was rounded up to 4-fold from a mean of 3.8 and route-to-route extrapolation results in a 5-fold adjustment for the OSF. When combined with the above factors and the fact that the cancer PODs represent upper-bound values, these uncertainties may potentially lead to overestimation of risk, but any differences from the true IUR/OSF values are unlikely to vary by more than ~2-fold.

3.2.6.4 Confidence in Human Health Hazard Data Integration and Representative Endpoints

Acute Non-Cancer

There is medium overall confidence in the database, weight of evidence, and dose-response for acute non-cancer endpoints. There are four endpoints relevent to acute exposure scenarios, covering three distinct endpoints from developmental toxicity studies and an immunological endpoint from an acute co-

infection study. Two of the four studies scored Medium in data quality, while one developmental endpoint and the acute immunotoxicity study scored High. The PODs cover several orders of magnitude, with benchmark MOEs of either 10 or 100. Confidence is reduced from a high due to the data quality scores, the wide range of PODs, and controversy over the most sensitive POD, from (Johnson et al., 2003). For developmental endpoints, there is some uncertainty extrapolating from chronic developmental toxicity studies to acute exposure, especially in assuming a consistent dose-response. This is a health protective assumption consistent with EPA Guidance (U.S. EPA, 1996; U.S. EPA, 1991), however this may possibly result in an overestimation of risk for some scenarios. For the acute immunotoxicity study (Selgrade and Gilmour, 2010) there is some inherent uncertainty extrapolating from the observed responses to pulmonary infection to a systemic response across multiple exposure routes, however an acute systemic response to infection is likely based on the systemic immunosuppression observed in multiple chronic studies (Sanders et al., 1982; Woolhiser et al., 2006). Confidence is raised from the robust WOE analysis performed on the congenital heart defects endpoint (see Appendix G), the presence of a variety of endpoints including a study using acute TCE administration, and reduced uncertainty factors due to the use of a PBPK model or allometric scaling.

Representative Acute Non-Cancer Endpoint

Based on the following considerations, the POD for mortality due to immunosuppression from (Selgrade and Gilmour, 2010) is considered to be the most robust and best representative POD for acute non-cancer scenarios. Confidence in the use of this study for evaluating acute exposure scenarios is High. Considerations for selection of this study and the High confidence rating include the following:

1) The study scored a High in data quality evaluation
2) The study used a broad dose range, with several concentrations above and below the LOAEL
3) The response data followed a consistent dose-response curve
4) The data is based on an acute exposure study so there is no uncertainty resulting from extrapolating from a repeated-dose study
5) The study demonstrated multiple assays supporting the apical outcome
6) The endpoint is severe

Chronic Non-Cancer

There is high overall confidence in the database, weight of evidence, and dose-response for chronic non-cancer endpoints. There are eleven endpoints relevant to chronic exposure scenarios across six health domains. Seven of the studies scored Medium in data quality, while the other four scored High. The PODs cover several orders of magnitude with benchmark MOEs ranging from 10 to 300. Confidence is high because there is strong WOE in support of all health effects, the PODs for three most sensitive endpoints differ by within an order of magnitude from each other, and the majority of PODs and have reduced uncertainty factors due to the use of a PBPK model.

Representative Chronic Non-Cancer Endpoint

Based on the following considerations, the POD for autoimmunity from (Keil et al., 2009) is considered to be the most robust and best representative POD for chronic non-cancer scenarios. Confidence in the use of this study for evaluating acute exposure scenarios is High. Considerations for selection of this study and the High confidence rating include the following:

1) The study scored a High in data quality evaluation
2) The study was of chronic duration (27-30 weeks) so uncertainty is reduced by not requiring a subchronic-to-chronic UF
3) The endpoint is associated with both functional immunological markers (increased anti-self antibodies) and immunological organ changes (thymus weight and cellularity)

4) The use of an early clinical marker as an endpoint and dose range are are expected to account for susceptibilities of subpopulations in disease progression

Cancer

There is medium to high overall confidence in the database, weight of evidence, and dose-response for cancer. Meta-analyses on the full database of relevant epidemiological studies confirm a statistically significant association between human exposure to TCE and the incidence of kidney cancer, liver cancer, or NHL. The IUR/OSF is derived from a High quality study (Charbotel et al., 2006) on kidney cancer, with the PODs adjusted upward to account for the additional two cancer sites. Confidence is slightly reduced due to some uncertainty over the precision of the dose-response estimate in accounting for all three cancer sites and in the GSH metabolism dose metrics but remains medium-high due to strong evidence for a mutagenic mode of action.

4 RISK CHARACTERIZATION

4.1 Environmental Risk

EPA took fate, exposure, and environmental hazard into consideration to characterize environmental risk of TCE. EPA determined that no further analysis beyond what was presented in the problem formulation document would be done for environmental exposure pathways for sediment for aquatic and terrestrial organisms, or land application of biosolids, water, or soil pathways for terrestrial organisms, in this risk evaluation. As stated in Section 2.1 Fate and Transport, TCE is not expected to accumulate in wastewater biosolids, soil, sediment, or biota. TCE is expected to volatilize from the water surface or from moist soil as indicated by its physical chemical properties (e.g., Henry's law constant) and by microbial biodegradation under some conditions. The EPI Suite™ volatilization module estimates that the half-life of TCE in a model river will be 1.2 hours and the half-life in a model lake will be 110 hours. Biodegradation of TCE in the environment is dependent on a variety of factors and thus, a wide range of degradation rates have been reported (ranging from days to years). TCE is not expected to accumulate in aquatic organisms due to low measured BCFs and estimated BAF.

Environmental exposure pathways for surface water for aquatic organisms are assessed and presented in this draft risk evaluation. As stated in Section 2.2 Environmental Exposures, modeled surface water concentrations of TCE ranged from 1.27E-5 ppb to 9,937.5 ppb from facilities releasing the chemical to surface water. Measured surface water concentrations near facilities range from 0.4 ppb to 447 ppb from published literature (1976-1977). Measured surface water concentrations in ambient water range from below the detection limit to 2.0 ppb in the Water Quality Portal (2013-2017) and from below the detection limit to 17 ppb in the published literature (1996-2001).

As stated in Section 3.1 Environmental Hazards, the reasonably available environmental hazard data indicate that TCE presents hazard to aquatic organisms. For acute exposures to invertebrates, toxicity values ranged from 7.8 to 33.85 mg/L (integrated into a geometric mean of 16 mg/L). For chronic exposures, toxicity values for fish and aquatic invertebrates were as low as 7.88 mg/L and 9.2 mg/L, respectively. These data also indicated that TCE presents hazard for aquatic plants, with toxicity values in algae as low as 0.03 mg/L (geometric mean between a NOEC and a LOEC), and a wide range in toxicity between algae species (EC_{50}s ranging from 26.24 – 820 mg/L).

A total of 25 aquatic environmental hazard studies were identified for TCE as acceptable. They were given mostly high and medium quality ratings during data evaluation (See [*Data Quality Evaluation of Environmental Hazard Studies* and *Environmental Hazard Data Extraction Table. Docket: EPA-HQ-OPPT-2019-0500*]). The [*Data Quality Evaluation of Environmental Hazard Studies. Docket: EPA-HQ-OPPT-2019-0500*] document presents details of the data evaluations for each study, including scores for each metric and the overall study score.

Given TCE's conditions of use under TSCA outlined in the 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 Risk Estimation for Aquatic.

4.1.1 Risk Estimation Approach

EPA used modeled exposure data from E-FAST, as well as monitored data from the Water Quality Portal (www.waterqualitydata.us) and reasonably available literature, to characterize the risk of TCE to

aquatic species. Risk quotients (RQs) were calculated using modeled surface water concentrations from E-FAST, monitored data, reasonably available literature, and the COCs calculated in the hazard section of this document (Section 3.1.5). An RQ is defined as:

RQ = Predicted Environmental Concentration / Effect Level or COC

An RQ equal to 1 indicates that environmental exposures are the same as the COC. If the RQ is above 1, the exposure is greater than the COC. If the RQ is below 1, the exposure is less than the COC. The COCs for aquatic organisms shown in Table 3-2 and the environmental concentrations shown in Section 2.2.6.2 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 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 potential for adverse effects in aquatic organisms, especially for chronic exposures. Therefore, the number of days that a COC was exceeded was also calculated using E-FAST. The days of exceedance modeled in E-FAST are not necessarily consecutive and could occur sporadically throughout the year. For TCE, EPA assumed continuous aquatic exposure for the longer exposure scenarios (i.e. 117-365 days per year of exceedance of a COC), and more of an interval or pulse exposure for shorter exposure scenarios (i.e. 1-40 days per year of exceedances of a COC). Due to the volatile properties of TCE, it is more likely that a chronic exposure duration will occur when there are long-term consecutive days of release versus an interval or pulse exposure which would more likely result in an acute exposure duration.

4.1.2 Risk Estimation for Aquatic

To characterize potential risk due to TCE exposure, RQs were calculated based on modeled data from E-FAST for sites that had surface water discharges of TCE according to TRI and DMR data (see Table 4-1). Surface water concentrations of TCE were modeled for 214 releases. Direct releases from facilities (releases 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 2.2.3, 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.

These facilities were modeled in E-FAST and all RQs are listed in Appendix E.2. 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 $\geq$ 1, or a chronic RQ $\geq$ 1 and 20 days or more of exceedance for the chronic COC) are presented in Table 4-1. All facilities were below these thresholds for manufacturing, spot cleaning and carpet cleaning, and commercial printing and copying, indicating no risks to aquatic organisms for these conditions of use.

Processing as a Reactant:

Of the 443 facilities processing TCE as a reactant (including 440 unknown sites modeled in E-FAST), one facility had acute RQs $\geq$ 1, or chronic or algae RQs $\geq$ 1 with 20 days or more of exceedances. Assuming 20 days of releases, Praxair Technology Center in Tonawanda, NY had a chronic RQs of 3.81

with 20 days of exceedance, and an algae COCs representing the most sensitive species of algae of 1,000 with 20 days of exceedance. In other words, the surface water concentration modeled for this facility was 3.81 times higher than the COC for chronic exposures, and 1,000 times higher than the COC for the most sensitive species of algae. Assuming 260 days of releases from the facility, the algae RQ representing the most sensitive species was 56.33 with 350 days of exceedance. However, for algae species as a whole, RQs for this site were 0.06 assuming 20 days of release and 0.00 assuming 350 days of release, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole. Risks were identified at this site for other aquatic organisms for chronic exposures, with a surface water concentration 3.81 times higher than the chronic COC and 20 days of exceedance.*

Repackaging:

Of the six facilities repackaging TCE, one had algae RQs ≥ 1 with 20 days or more of exceedances. Assuming 20 days of release per year, Hubbard-Hall Inc in Waterbury, CT had an RQ for the most sensitive species of alge as high as 113.04 with 20 days of exceedance. Assuming this facility released TCE for 250 days per year, the RQ is 9.06 with 194 days of exceedance. However, for algae species as a whole, RQs for this site were 0.01 for 20 days of releases, and 0.00 for 250 days, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at these sites, but not for algae species as a whole. No risks were identified for other aquatic organisms in this condition of use.*

Open-top Vapor Degreasing:

Of the 64 open-top vapor degreasing facilities, three sites had acute RQs ≥ 1, or chronic or algae RQs ≥ 1 with 20 days or more of exceedances. Assuming 20 days of releases, US Nasa Michoud Assembly Facility in New Orleans, LA had acute RQs of 3.11, a chronic RQs of 12.61 with 20 days of exceedance, and an algae COCs representing the most sensitive species of algae of 3,312.50 with 20 days of exceedance. Assuming 260 days of relese from the facility, the algae RQ representing the most sensitive species was 255.21 with 260 days of exceedance. However, for algae species as a whole, RQs for this site were 0.01 assuming 260 days of release, and 0.19 assuming 20 days of release, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole. Risks were identified at this site for other aquatic organisms for acute and chronic exposures, with a surface water concentration 3.11 times higher than the acute COC and 12.61 times higher than the chronic COC and 20 days of exceedance.*

GM Components Holdings LLC in Lockport, NY had an RQ for the most sensitive species of algae of 3.66 with 117 days of exceedance, assuming 260 days of release per year. Assuming 20 days of release, this site has an RQ for the most sensitive species of algae of 48.16 with 20 days of exceedance. However, for algae species as a whole, RQs for this facility were 0.00 for this site, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole.*

Akebono Elizabethtown Plant in Elizabethtown, KY had an RQ for the most sensitive species of algae of 1.62 with 27 days of exceedance, assuming 260 days of release per year. However, for algae species as a whole, RQs for this facility were 0.00 for this site, meaning the concentration did not exceed the

COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole.*

Adhesives, Sealants, Paints, and Coatings:

Of the 54 facilities using TCE as adhesives, sealants, paints, and coatings, one site had algae RQs ≥ 1 with 20 days or more of exceedances. Raytheon Company in Portsmouth, RI had an RQ for the most sensitive species of alge as high as 44.44, assuming 20 days of release per year. In other words, the surface water concentration modeled for this facility was 44.44 times higher than the COC for the most sensitive species of algae (3 ppb). Additionally, this COC was exceeded for 20 days. Assuming this facility released TCE for 250 days per year, the RQ is 3.61 with 250 days of exceedance. However, for algae species as a whole, RQs for this facility were 0.00, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole. No risks were identified for other aquatic organisms for this condition of use.*

Other Industrial Uses:

Of the 21 facilities with other industrial uses of TCE, three sites had algae RQs ≥ 1 with 20 days or more of exceedances. Eli Lilly And Company-Lilly Tech Ctr in Indianapolis, IN had an RQ for the most sensitive species of alge of 3.01, assuming 250 days of release per year. In other words, the surface water concentration modeled for this facility was 3.01 times higher than the COC for the most sensitive species of algae (3 ppb). Additionally, this COC was exceeded for 35 days. Washington Penn Plastics in Frankfort, KY had an RQ for the most sensitive species of alge of 2.51, assuming 250 days of release per year. Additionally, this COC was exceeded for 22 days. Keeshan and Bost Chemical Co., Inc. in Manvel, TX had an RQ for the most sensitive species of algae of 66.67 with 20 days of exceedance, assuming 20 days of release per year. Assuming 350 days of release, this site has an RQ for the most sensitive species of algae of 3.17 with 350 days of exceedance. However, for algae species as a whole, RQs for these facilities were 0.00, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at these sites, but not for algae species as a whole. No risks were identified for other aquatic organisms for this condition of use.*

Industrial Processing Aid:

Of the six industrial processing aid facilities, one site had algae RQs ≥ 1 with 20 days or more of exceedances. Entek International LLC in Lebanon, OR had an RQ for the most sensitive species of algae as high as 46.11, assuming 20 days of release per year. In other words, the surface water concentration modeled for this facility was 46.11 times higher than the COC for the most sensitive species of algae (3 ppb). Additionally, this COC was exceeded for 20 days. Assuming this facility released TCE for 300 days per year, the RQ is 3.10 with 140 days of exceedance. However, for algae species as a whole, RQs for this facility were 0.00, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole. No risks were identified for other aquatic organisms for this condition of use.*

Other Commercial Uses:

Of the nine facilities with other commercial uses of TCE, one site had algae RQs ≥ 1 with 20 days or more of exceedances. Park Place Mixed Use Development in Annapolis, MD had an RQ for the most sensitive species of algae as high as 36.67, assuming 20 days of release per year. In other words, the surface water concentration modeled for this facility was 36.67 times higher than the COC for the most

sensitive species of algae (3 ppb). Additionally, this COC was exceeded for 20 days. Assuming this facility released TCE for 250 days per year, the RQ is 3.00 with 250 days of exceedance. However, for algae species as a whole, RQs for this facility were 0.00, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at this site, but not for algae species as a whole. No risks were identified for other aquatic organisms in this condition of use.*

Process Solvent Recycling and Worker Handling of Wastes:
Of the five facilities with other commercial uses of TCE, three sites had algae RQs ≥ 1 with 20 days or more of exceedances. Assuming 20 days of release per year, Clean Water Of New York Inc in Staten Island, NY had an RQ for the most sensitive species of alge as high as 46.08 with 20 days of exceedance. Assuming this facility released TCE for 250 days per year, the RQ is 3.92 with 250 days of exceedance. Assuming 20 days of release, Veolia Es Technical Solutions LLC in Middlesex, NJ had an RQ for the most sensitive species of alge of 11.91 with 20 days of exceedance. And assuming 250 days of releases, Clean Harbors Deer Park LLC in La Porte, TX had an RQ for the most sensitive species of alge of 2.86 with 110 days of exceedance. However, for algae species as a whole, RQs for at all three facilities were 0.00, meaning the concentration did not exceed the COC of 52,000 ppb which represents nine different species of algae. *Therefore, there may be risk for some of the most sensitive species of algae at these sites, but not for algae species as a whole. No risks were identified for other aquatic organisms in this condition of use.*

Wastewater Treatment Plants (WWTPs):
Of the nine WWTPs, one site had algae RQs ≥ 1 with 20 days or more of exceedances. New Rochelle STP in New Rochelle, NY had an RQ for the most sensitive species of alge of 4.26, assuming 20 days of release per year. This means that the surface water concentration modeled for this facility was 4.26 times higher than the COC for the most sensitive species of algae (3 ppb). Additionally, this COC was exceeded for 20 days. Assuming this facility released TCE for 365 days per year, the RQ is only 0.23 with 0 days of exceedance. A WWTP is likely to be operating at greater than 20 days of release, therefore the RQ associated with the high-end days of release scenario (365 days) is likely more representative of actual conditions. *Therefore, no risks to aquatic species were for this facility or condition of use.*

220 **Table 4-1. Environmental Risk Quotients for Facilities Releasing TCE to Surface Water as Modeled in E-FAST** (RQs ≥ 1 in **bold**)

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	COC Type	COC (ppb)	Days of Exceedance (days/year) [h]	Risk Quotient
OES: Processing as a Reactant										
Praxair Technology Center, Tonawanda, NY NPDES: NY0000281	Surface Water	NPDES NY0000281	Still body	350	0.00169	169	Acute	3,200	NA	0.05
							Chronic	788	0	0.21
							Algae	3	350	**56.33**
							Algae (HC_{05})	52,000	0	0.00
				20	0.03	3000	Acute	3,200	NA	0.94
							Chronic	788	20	**3.81**
							Algae	3	20	**1,000.00**
							Algae (HC_{05})	52,000	0	0.06
OES: Repackaging										
Hubbard-Hall Inc, Waterbury, CT NPDES: Unknown	Off-site Waste-water Treatment	Receiving Facility: Recycle Inc.; POTW (Ind.)	Surface water	250	1.108	27.18	Acute	3,200	NA	0.01
							Chronic	788	0	0.03
							Algae	3	194	**9.06**
							Algae (HC_{05})	52,000	0	0.00
				20	13.85	339.11	Acute	3,200	NA	0.11
							Chronic	788	1	0.43
							Algae	3	20	**113.04**
							Algae (HC_{05})	52,000	0	0.01
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, and Metalworking Fluids)										
US Nasa Michoud Assembly Facility, New Orleans, LA NPDES: LA0052256	Surface Water	Surrogate NPDES LA0003280	Still body	260	1.96	765.63	Acute	3,200	NA	0.24
							Chronic	788	0	0.97
							Algae (COC)	3	260	**255.21**
							Algae (HC_{05})	52,000	0	0.01
				20	25.44	9937.5	Acute	3,200	NA	**3.11**
							Chronic	788	20	**12.61**
							Algae	3	20	**3,312.50**
							Algae (HC_{05})	52,000	0	0.19
GM Components Holdings LLC, Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	260	0.13	10.97	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	117	**3.66**
							Algae (HC_{05})	52,000	0	0.00
				20	1.71	144.47	Acute	3,200	NA	0.05
							Chronic	788	0	0.18
							Algae	3	20	**48.16**
							Algae (HC_{05})	52,000	0	0.00
Akebono Elizabethtown Plant,			Surface water	260	0.07	4.87	Acute	3,200	NA	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	COC Type	COC (ppb)	Days of Exceedance (days/year) [h]	Risk Quotient
Elizabethtown, KY NPDES: KY0089672	Surface Water	Surrogate NPDES KY0022039					Chronic	788	0	0.01
							Algae (COC)	3	27	**1.62**
							Algae (HC_{05})	52,000	0	0.00
				20	0.897	62.38	Acute	3,200	NA	0.02
							Chronic	788	0	0.08
							Algae	3	16	**20.79**
							Algae (HC_{05})	52,000	0	0.00
OES: Adhesives, Sealants, Paints, and Coatings										
Raytheon Company, Portsmouth, RI NPDES: RI0000281	Surface Water	NPDES RI0000281	Still body	250	0.013	10.83	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	250	**3.61**
							Algae (HC_{05})	52,000	0	0.00
				20	0.160	133.33	Acute	3,200	NA	0.04
							Chronic	788	0	0.17
							Algae (COC)	3	20	**44.44**
							Algae (HC_{05})	52,000	0	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	Acute	3,200	NA	0.00
							Chronic	788	0	0.00
							Algae (COC)	3	0	0.11
							Algae (HC_{05})	52,000	0	0.00
OES: Other Industrial Uses										
Eli Lilly And Company-Lilly Tech Ctr, Indianapolis, IN NPDES: IN0003310	Surface Water	NPDES IN0003310	Surface water	250	1.553	9.03	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	35	**3.01**
							Algae (HC_{05})	52,000	0	0.00
				20	19.410	113.09	Acute	3,200	NA	0.04
							Chronic	788	0	0.14
							Algae	3	17	**37.70**
							Algae (HC_{05})	52,000	0	0.00
Washington Penn Plastics, Frankfort, KY NPDES: KY0097497	Surface Water	Surrogate NPDES KY0028410	Surface water	250	0.032	7.53	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	22	**2.51**
							Algae (HC_{05})	52,000	0	0.00
				20	0.399	94.12	Acute	3,200	NA	0.03
							Chronic	788	0	0.12
							Algae	3	13	**31.37**

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	COC Type	COC (ppb)	Days of Exceedance (days/year) [h]	Risk Quotient
							Algae (HC_{05})	52,000	0	0.00
Keeshan and Bost Chemical Co., Inc., Manvel, TX NPDES: TX0072168	Surface Water	NPDES TX0072168	Still body	350	0.000095	9.50	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae	3	350	**3.17**
							Algae (HC_{05})	52,000	0	0.00
				20	0.002	200.00	Acute	3,200	NA	0.06
							Chronic	788	0	0.25
							Algae	3	20	**66.67**
							Algae (HC_{05})	52,000	0	0.00
OES: Industrial Processing Aid										
Entek International LLC, Lebanon, OR NPDES: N/A	Off-site Waste-water Treatment	No info on receiving facility; POTW (Ind.)	Surface water	300	0.38	9.3	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	140	**3.10**
							Algae (HC_{05})	52,000	0	0.00
				20	5.65	138.34	Acute	3,200	0	0.04
							Chronic	788	0	0.18
							Algae (COC)	3	20	**46.11**
							Algae (HC_{05})	52,000	0	0.00
OES: Other Commercial Uses										
Park Place Mixed Use Development, Annapolis, MD NPDES: MD0068861	Surface Water	Surrogate NPDES MD0052868	Still body	250	0.00027	9	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	250	**3.00**
							Algae (HC_{05})	52,000	0	0.00
				20	0.00334	110	Acute	3,200	NA	0.03
							Chronic	788	0	0.14
							Algae (COC)	3	20	**36.67**
							Algae (HC_{05})	52,000	0	0.00
OES: Process Solvent Recycling and Worker Handling of Wastes										
Clean Water Of New York Inc, Staten Island, NY NPDES: NY0200484	Surface Water	Surrogate NPDES NJ0000019	Still body	250	0.004	11.76	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	250	**3.92**
							Algae (HC_{05})	52,000	0	0.00
				20	0.047	138.24	Acute	3,200	NA	0.04
							Chronic	788	0	0.18
							Algae	3	20	**46.08**
							Algae (HC_{05})	52,000	0	0.00
			Still body	250	24.1	2.85	Acute	3,200	NA	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	COC Type	COC (ppb)	Days of Exceedance (days/year) [h]	Risk Quotient
Veolia Es Technical Solutions LLC, Middlesex, NJ NPDES: NJ0020141	Off-site Waste-water Treatment	Receiving Facility: Middlesex Cnty UA; NPDES NJ0020141					Chronic	788	0	0.00
							Algae (COC)	3	0	0.95
							Algae (HC_{05})	52,000	0	0.00
				20	301.78	35.72	Acute	3,200	NA	0.01
							Chronic	788	0	0.05
							Algae	3	20	**11.91**
							Algae (HC_{05})	52,000	0	0.00
Clean Harbors Deer Park LLC, La Porte, TX NPDES: TX0005941	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.35	8.57	Acute	3,200	NA	0.00
							Chronic	788	0	0.01
							Algae (COC)	3	110	**2.86**
							Algae (HC_{05})	52,000	0	0.00
				20	4.36	106.75	Acute	3,200	NA	0.03
							Chronic	788	0	0.14
							Algae	3	19	**35.58**
							Algae (HC_{05})	52,000	0	0.00
OES: Wastewater Treatment Plants (WWTP)										
New Rochelle STP, New Rochelle, NY NPDES: NY0026697	Surface Water	NPDES NY0026697	Still body	365	0.043	0.7	Acute	3,200	NA	0.00
							Chronic	788	0	0.00
							Algae (COC)	3	0	0.23
							Algae (HC_{05})	52,000	0	0.00
				20	0.786	12.79	Acute	3,200	NA	0.00
							Chronic	788	0	0.02
							Algae (COC)	3	20	**4.26**
							Algae (HC_{05})	52,000	0	0.00

a. Facilities actively releasing trichloroethylene 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 81% is applied to all indirect releases, as well as direct releases from WWTPs.

c. If a valid NPDES of the direct or indirect releaser was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.

d. EFAST uses ether the "surface water" model, for rivers and streams, or the "still water" model, for lakes, bays, and oceans.

e. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.

f. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.

g. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.

h. To determine the PDM days of exceedance for still bodies of water, the release days provided by the EPA Engineers should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.

EPA also used surface water monitoring data from the Water Quality Portal (WQP) and from the published literature to characterize the risk of TCE to aquatic organisms. For the most part these monitored surface water concentrations reflect concentrations of TCE in ambient water. There was one US study (U.S. EPA, 1977) that had measurements reflecting near-facility monitoring data. The other monitored data collected in the US reflect ambient concentrations.

Monitored data from one US study (U.S. EPA, 1977) in the published literature reporting near-facility concentrations of TCE collected between 1976 and 1977 ranging from 0.4 to 447 µg/L. While these data reflect historical levels of TCE, they are helpful to compare measured near-facility concentrations to the modeled near-facility concentrations from E-FAST. The measured concentrations in this study encompases the range of the modeled estimates across all OES with the exception of two sites, that release to still water bodies.

EPA also had monitored data reflecting ambient water concentrations. EPA's Storage and Retrieval (STORET) data and USGS's National Water Information System (NWIS) data were extracted on Oct 3rd, 2018 from the WQX/WQP. These data show an average concentration for TCE of 0.33 ± 0.29 µg/L or ppb in surface water from 2,273 measurements taken throughout the US between 2013 and 2017. The highest value recorded during these years was 2 µg/L or ppb, which was measured in 2017. Table 4-2 shows that none of the RQs for aquatic species are greater than or equal to 1. The RQs for algae range from 0 to 0.67. Acute and chronic RQs for other aquatic species are all very close to 0.

Table 4-2. RQs Calculated using Monitored Environmental Concentrations from WQX/WQP

Monitored Surface Water Concentrations (ppb) from 2013-2017	Algae RQ		RQ using Acute COC of 3,200 ppb	RQ using Chronic COC of 788 ppb
	using COC of 3 ppb	using HC_{05} of 52,000 ppb		
Mean (Standard Deviation): 0.33 (0.29) ppb	0.11	0.0	0.0	0.0
Maximum: 2 ppb	0.67	0.0	0.0	0.0

The published literature show monitored data in six U.S. studies encompassing 1,177 surface water samples collected from river and oceans throughout the nation between 1979 and 2001. Reported concentrations of TCE ranged from below the detection limit (0.0001 to 0.08) to 17.3 µg/L or ppb, with reported central tendency values ranging from 0.0002 to 1.17 µg/L (USGS, 2006; Sauer, 1981; Singh et al., 1983; USGS, 2003; Robinson et al., 2004). The maximum concentration was collected from the Charles River in Boston, Massachusetts (an urban area) between 1998 and 2000 (Robinson et al., 2004). The next highest TCE concentration was 2.0 µg/L, collected during a large nationwide survey of surface water for drinking water sources (rivers and reservoirs) between 1999 and 2000 (USGS, 2003). Table 4-3 shows an RQs for algae range from 0 to 5.77 using monitored surface water concentrations from the published literature. Acute RQs for other aquatic organisms range from 0 to 0.01, and chronic RQs range from 0 to 0.02.

Table 4-3. RQs Calculated using Monitored Environmental Concentrations from Published Literature

Monitored Surface Water Concentrations (ppb) from 2013-2017	Algae RQ		RQ using Acute COC of 3,200 ppb	RQ using Chronic COC of 788 ppb
	using COC of 3 ppb	using HC_{05} of 52,000 ppb		
Central tendency values: 0.0002 – 1.17 ppb	0.00 – 0.39	0.00	0.00	0.00
Maximum: 17.3 ppb	5.77	0.00	0.01	0.02

To compare the modeled data with the monitored data, EPA conducted a watershed analysis by combining monitored data from WQX/WQP with predicted concentrations from E-FAST modeled facility releases, using the geospatial analysis outlined in Section 2.2. A geographic distribution of the concentrations is shown in Figure 4-1 and Figure 4-2 (east and west US) for the maximum days of release scenario, and in Figure 4-3 and Figure 4-4 (east and west US) for the 20-days of release scenario. The co-location of TCE releasing facilities and monitoring stations in a HUC is shown in Figure 4-5. for HUCs in North Carolina and in Figure 4-5 for the HUC in New Mexico. The modeled estimates are only shown in Figure 4-5 and Figure 4-6 for the higher release frequency scenarios, which are associated with lower predicted surface water concentrations. The surface water concentrations were compared to the COCs in these maps.

Figure 4-1 to Figure 4-6 compare WQX Monitoring Stations from 2016 to TCE-releasing facilities modeled in E-FAST. The figures show that while some facilities releasing TCE to surface water were co-located with monitoring locations in WQX, none were downstream from facilities. The monitored data, which represents localized concentrations of TCE in ambient water, generally show lower concentrations than the modeled surface water concentrations from E-FAST, which represents concentrations near facilities releasing TCE. The modeled and monitored data together indicate that risk to aquatic organisms from TCE exposure is more likely in areas near the facilities, rather than in ambient water; however the monitored data was limited geographically and temporally.

Figure 4-1. Concentrations of Trichloroethylene from Releasing Facilities (Higher Release Frequency Scenarios) and WQX Monitoring Stations: Year 2016, East US.
[Note: All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.]

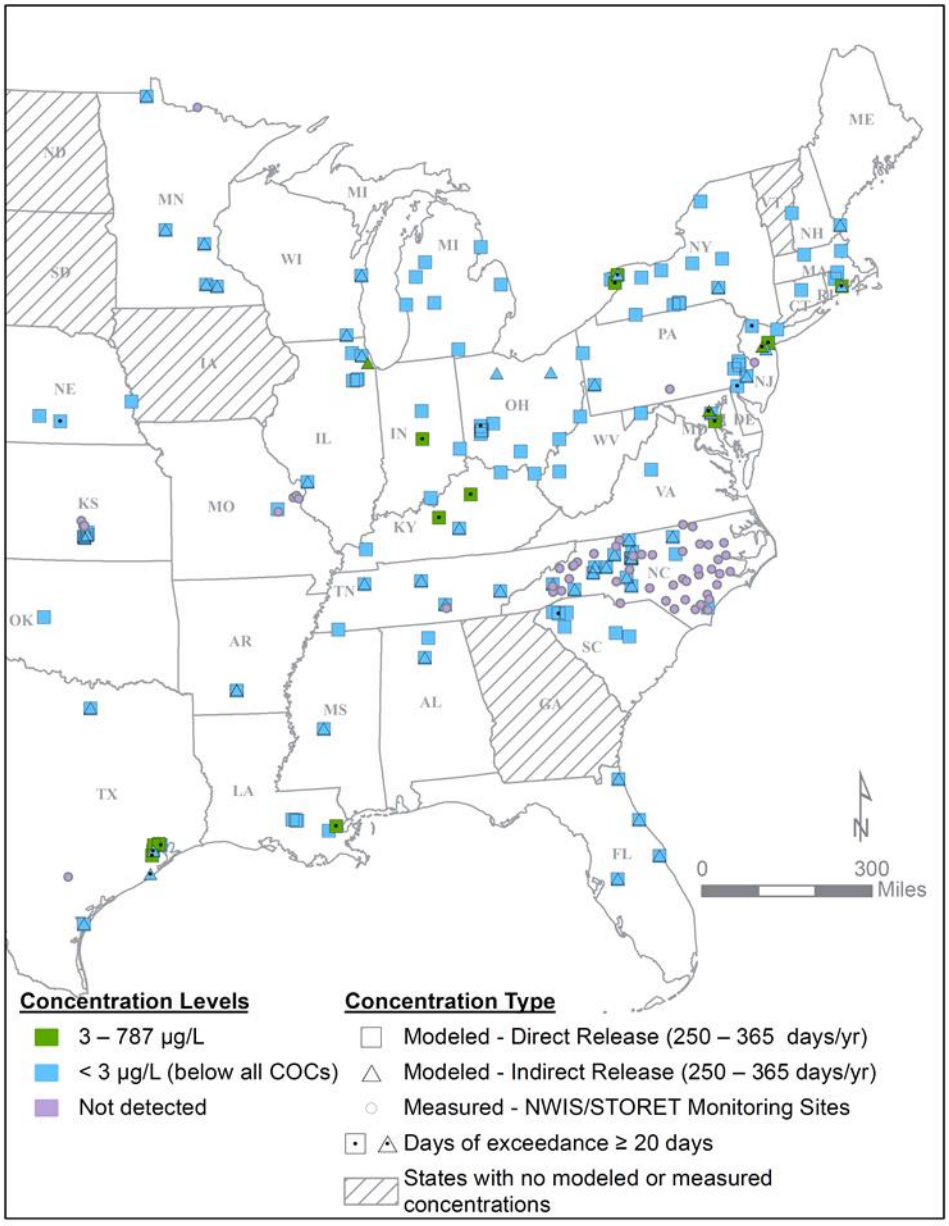


Figure 4-2. Concentrations of Trichloroethylene from Releasing Facilities (Higher Release Frequency Scenarios) and WQX Monitoring Stations: Year 2016, West US.

[Note: All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.]

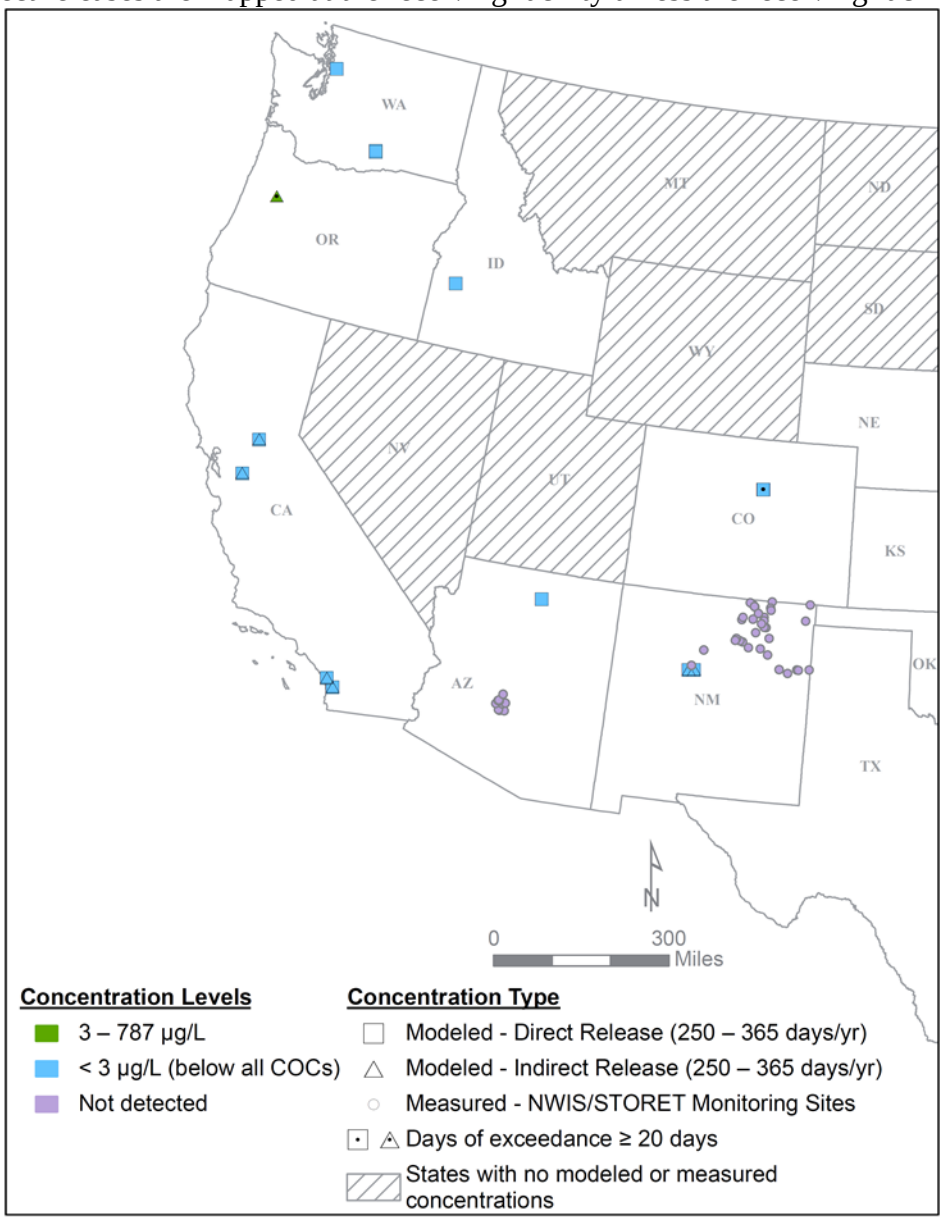


Figure 4-3. Concentrations of Trichloroethylene from Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, East US.

[Note: All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.]

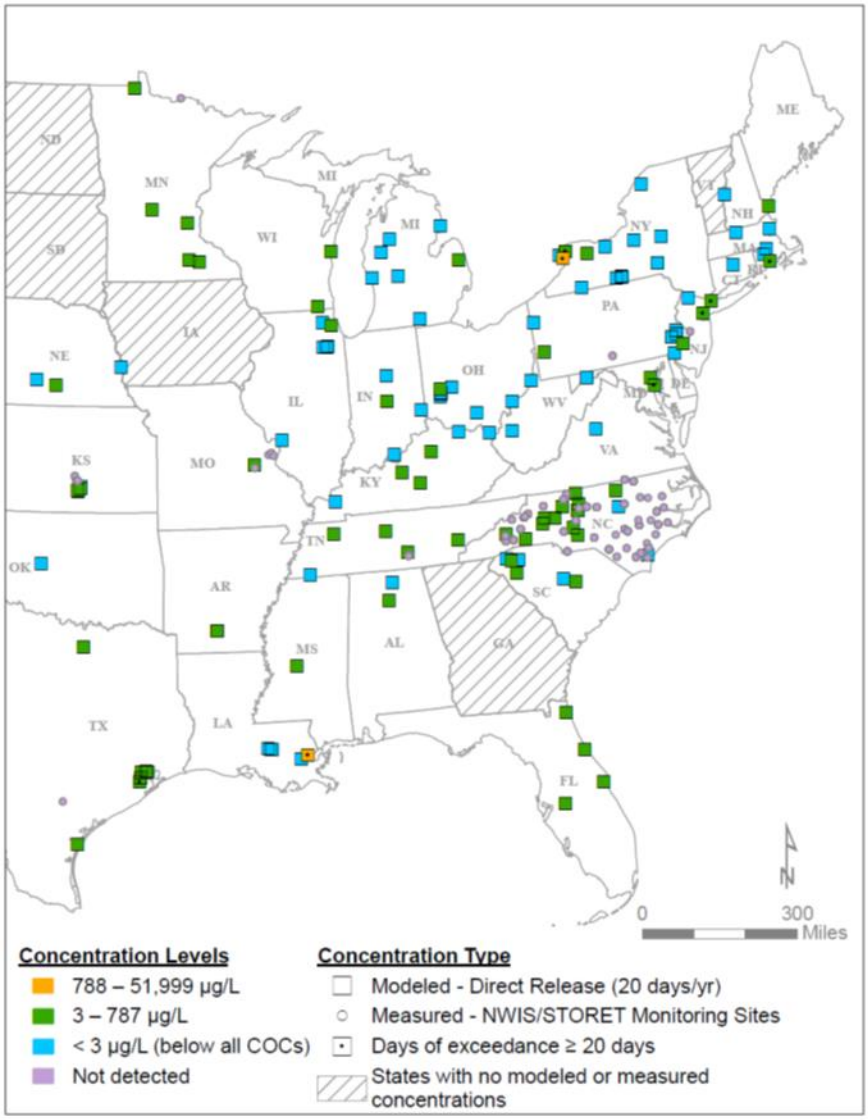


Figure 4-4. Concentrations of Trichloroethylene Releasing Facilities (20 Days of Release Scenario) and WQX Monitoring Stations: Year 2016, West US.

[Note: All indirect releases are mapped at the receiving facility unless the receiving facility is unknown.]

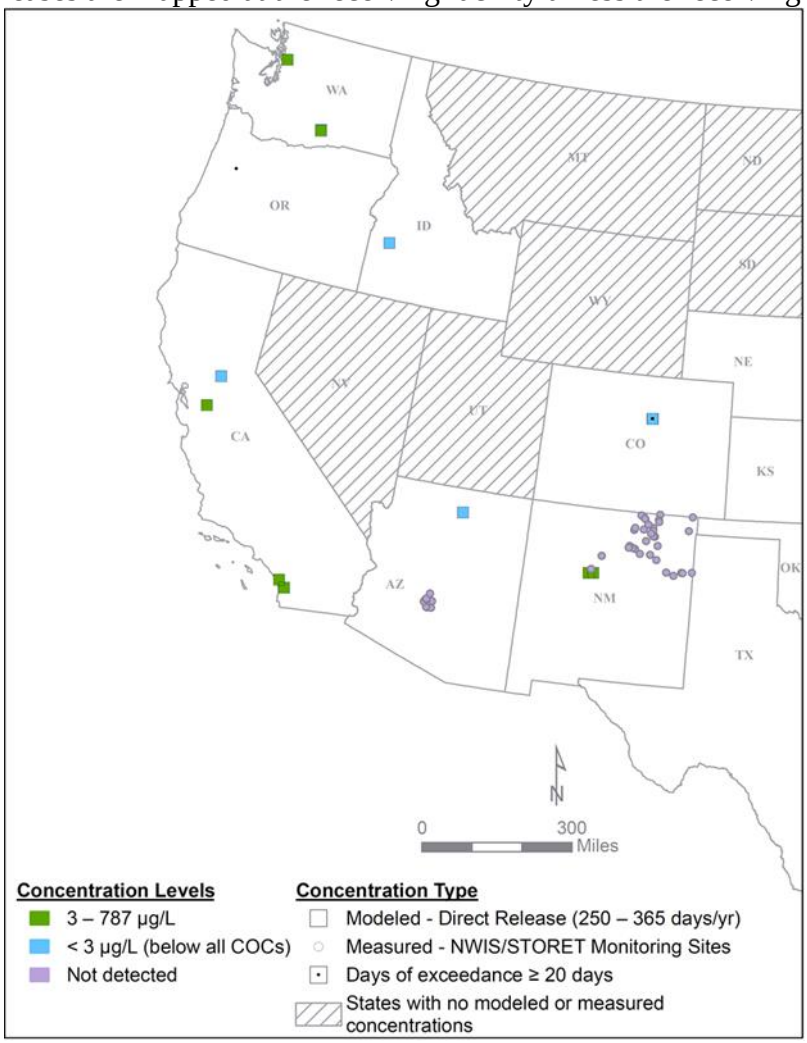


Figure 4-5. Co-location of Trichloroethylene-Releasing Facilities and WQX Monitoring Stations at the HUC 8 Level in NC

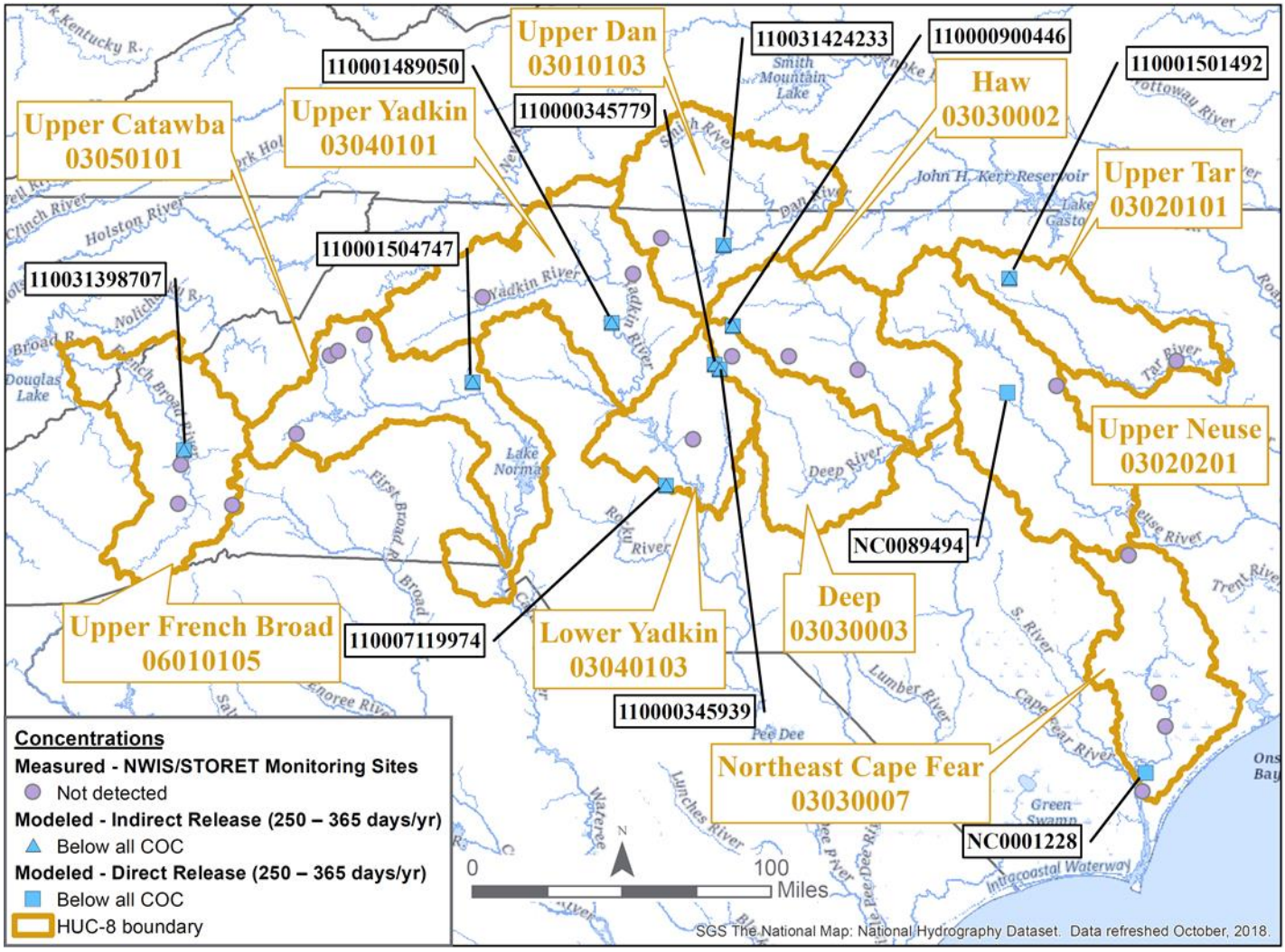
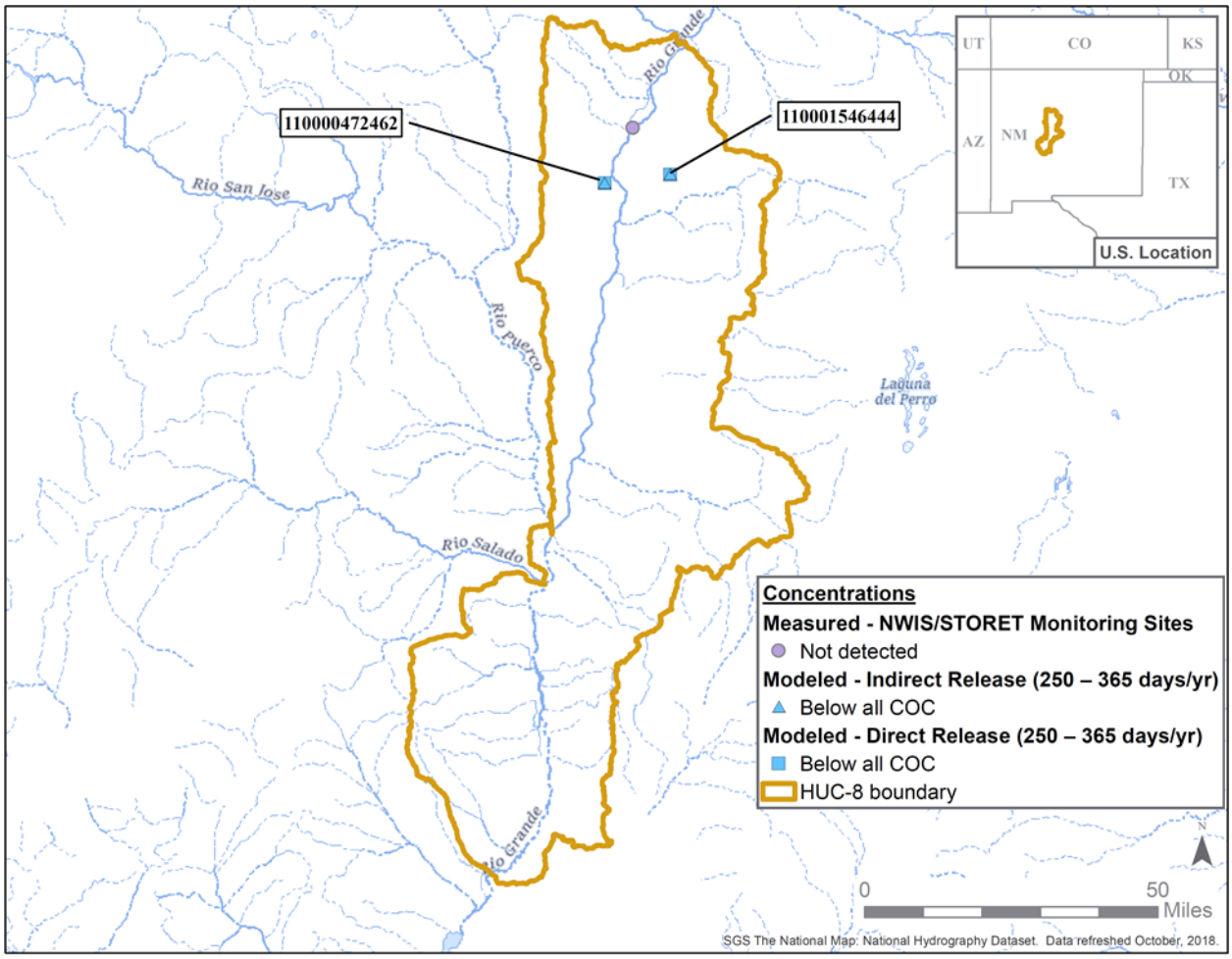


Figure 4-6. Co-location of Trichloroethylene-Releasing Facilities and WQX Monitoring Stations at the HUC 8 Level in NM



4.1.3 Risk Estimation for Sediment

EPA did not quantitatively assess exposure to sediment organisms, because TCE is not expected to partition to sediment, based on physical-chemical properties. TCE is expected to remain in aqueous phases and not adsorb to sediment due to its water solubility (> 1280 m g/L) and low partitioning to organic matter (log K_{OC} = 1.8-2.17). Limited sediment monitoring data for TCE that are available suggest that TCE is present in sediments, but because TCE has relatively low partition to organic matter (log K_{OC} = 1.802.17) and biodegrades slowly [19% biodegradation in 28 days (ECB2004)], TCE concentrations in sediment pore water are expected to be similar to the concentrations in the overlying water or lower in the deeper part of sediment which anaerobic condition prevails. Thus, the TCE detected in sediments is likely from the pore water.

4.1.4 Risk Estimation for Terrestrial

EPA did not quantitatively assess exposure to terrestrial organisms through soil, water, or biosolids. TCE is not expected to partition to soil but is expected to volatilize to air, based on its physical-chemical properties. Review of hazard data for terrestrial organisms shows potential hazard; however, physical-chemical properties do not support an exposure pathway through water and soil pathways to terrestrial organisms.

TCE is not anticipated to partition to biosolids during wastewater treatment. TCE has a predicted 81% wastewater treatment removal efficiency, predominately due to volatilization during aeration. Any TCE present in the water portion of biosolids following wastewater treatment and land application would be expected to rapidly volatilize into air. To further support this analysis, TCE was not detected in EPA's Targeted National Sewage Sludge Survey (TNSSS) nor was it reported in biosolids during EPA's Biennial Reviews for Biosolids, a robust biennial literature review conducted by EPA's Office of Water {U.S. EPA, 2019, 5933985}. Furthermore, TCE is not anticipated to remain in soil, as it is expected to either volatilize into air or migrate through soil into groundwater.

TCE is expected to volatilize to air, based on physicochemical properties. However, the emission pathways to ambient air from commercial and industrial stationary sources or associated inhalation exposure of terrestrial species were out of the scope of the risk evaluation because stationary source releases of TCE to ambient air are adequately assessed and any risks effectively managed when under the jurisdiction of the Clean Air Act (CAA).

4.2 Human Health Risk

4.2.1 Risk Estimation Approach

The use scenarios, populations of interest and toxicological endpoints used for acute and chronic exposures are are presented in Table 4-4.

Table 4-4. Use Scenarios, Populations of Interest and Toxicological Endpoints Used for Acute and Chronic Exposures

Population of Interest and Exposure Scenario	***Workers:*** [1] Acute- Adolescent (≥16 years old) and adult workers exposed to TCE for a single 8-hr exposure Chronic- Adolescent (≥16 years old) and adult workers exposed to TCE for the entire 8-hr workday for 260 days per year for 40 working years ***Occupational Non-User:*** Acute or Chronic- Adolescent (≥16 years old) and adult worker exposed to TCE indirectly by being in the same work area of the building ***Consumers*** [2] Acute- Children (≥11 years old) and adult consumers exposed to TCE for a short period of time during use [3] ***Bystanders:*** Acute- Individuals of all ages exposed to TCE through consumer use of another individual.
Health Effects, Concentration and Time Duration	***Non-Cancer Point of Departures (POD)*:** HEC- ppm; POD HECs represent 24hr values and exposure concentrations have been adjusted to match the time duration for inhalation exposure. Note: Selgrade 2010 POD is a 3h acute value that has been adjusted to match the 24hr exposure value for workers (3h exposure values were used for consumers to match available 3hr exposure estimates from CEM). HED- mg/kg; for dermal risk estimates ***Non-Cancer Health Effects:*** [4] Acute- Developmental effects and immunotoxicity Chronic- Liver effects, kidney effects, neurological effects, immune effects, reproductive effects, and developmental effects
Uncertainty Factors (UF) used in Non-Cancer Margin of Exposure (MOE) calculations	***Benchmark MOEs:*** Vary by endpoint ***Benchmark MOE*** = (UF_S) x (UF_A) x (UF_H) x $(UF_L)^5$

[1]Adult workers (>16 years old) include both healthy female and male workers.
[2] EPA believes that the users of these products are generally adults, but young teenagers and even younger children may be users or be in the same room with the user while engaging in various conditions of use. Since there are not survey data for consumer behavior patterns or a way to create varying behavior patterns for different age groups, the indoor air concentrations shown in Table 4-4. Use could be extended to all users.

[3] EPA believes that the users of these products are generally adults, but young teenagers and even younger children may be users or be in the same room with the user while engaging in various conditions of use. Since there are not survey data for consumer behavior patterns or a way to create varying behavior patterns for different age groups, the indoor air concentrations shown in Table 4-5 could be extended to all users.
[4] Female workers of childbearing age are the population of interest for reproductive and developmental effects. For other health effects (e.g., liver, kidney, etc.), healthy female or male workers were assumed to be the population of interest.
[5] UF_S=subchronic to chronic UF; UF_A=interspecies UF; UF_H=intraspecies UF; UF_L=LOAEL to NOAEL UF

The EPA uses a Margin of Exposure (MOE) approach to assessing non-cancer risk. The MOE is the ratio of the point of departure (POD) dose divided by the human exposure dose. The MOE is compared to the benchmark MOE. If the MOE exceeds the benchmark MOE, this indicates the potential for risk to human health.

Acute or chronic MOEs (MOE_{acute} or $MOE_{chronic}$) were used in this assessment to estimate non- cancer risks using Equation 4-1.

Equation 4-1. Equation to Calculate Non-Cancer Risks Following Acute or Chronic Exposures Using Margin of Exposures

$$\text{MOE}_{\text{acute or chronic}} = \frac{\text{Non} - \text{cancer Hazard value (POD)}}{\text{Human Exposure}}$$

Where:

MOE = **Margin of exposure (unitless)**
Hazard Value (POD) = HEC (ppm) or HED (mg/kg)
Human Exposure = Exposure estimate (in ppm or mg/kg) from occupational exposure assessment
= Exposure estimate (in ppm or mg/kg) from consumer exposure assessment

Acute Concentrations (ACs) in ppm and acute Average Daily Doses (ADDs) were used to calculate occupational non-cancer risks following acute inhalation or dermal exposure, respectively. Average Daily Concentrations (ADC) and non-cancer chronic ADDs were used for calculating occupational non-cancer risks following inhalation or dermal chronic exposure, respectively. ADD values accounted for modeled evaporation, representing an estimated absorbed dose. Lifetime Average Daily Concentrations (LADC) and cancer Chronic Retained Doses (CRDs) were used for calculating occupational cancer risks. See Appendix J for more details on the derivation of chronic exposure values from acute concentrations/doses.

Consumer risks via inhalation were calculated based on maximum Time-Weighted Average (TWAs) for either 3h or 24h periods and consumer risks via dermal exposure were calculated based on Acute Dose Rate (ADR). See Section 2.3.1.3.1 for more details on consumer exposure).

EPA used margin of exposures (MOEs) to estimate acute or chronic risks for non-cancer based on the following:

- the most sensitive and robust HEDs within each health effects domain reported in the literature;
- the endpoint/study-specific UFs applied to the HEDs per EPA RfD Guidance (U.S. EPA, 2002); and
- the exposure estimates calculated for TCE uses examined in this risk assessment (see Section 2.3 - Human Exposures).

MOEs allow for the presentation of a range of risk estimates. The occupational exposure scenarios considered both acute and chronic exposures, while consumer exposure scenarios considered only acute exposures. In general, the frequency of product use was considered to be too low to create chronic risk concerns. Although Westat (1987) survey data indicate that use frequencies for high-end product users (i.e., those reflecting 95th percentile annual use frequencies) may use products up to 50 times per year, available toxicological data is based on either single or continuous TCE exposure and it is unknown whether these use patterns are expected to be clustered or intermittent (e.g. one time per week). There is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated intermittent human exposures. Therefore, EPA cannot fully rule out that consumers at the high-end frequency of use could possibly be at risk for chronic hazard effects (Section 3.2), however it is expected to be unlikely.

Different adverse endpoints were used based on the expected exposure durations. For non-cancer effects, risks for developmental effects were evaluated for acute (short-term) exposures, whereas risks for other adverse effects (liver toxicity, kidney toxicity, neurotoxicity, immunotoxicity, reproductive effects, and developmental effects) were evaluated for repeated (chronic) exposures to TCE.

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 cumulative 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 relative to the benchmark MOE for that endpoint, the more unlikely it is that a non-cancer adverse effect would occur.

Extra cancer risks for chronic exposures to TCE were estimated using Equation 4-2. Estimates of extra cancer risks should be interpreted as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e., incremental or extra individual lifetime cancer risk). For purposes of this risk evaluation, EPA considers extra risk of 1 x 10^{-4} (or 1E-4 in shorthand) to be the benchmark for occupational risk estimation.

Equation 4-2. Equation to Calculate Extra Cancer Risks

$$\boldsymbol{Risk = Human\ Exposure}\ \textit{(LADC)} \times \boldsymbol{POD}\ \textit{(IUR or OSF)}$$

Where:

Risk = Extra cancer risk (unitless)
Human exposure = Exposure estimate (ppm or mg/kg/day) from occupational exposure assessment
POD = Inhalation unit risk (0.022 per ppm) or oral slope factor (0.0464 per mg/kg-day)

Risk estimates were calculated for all of the studies per health effects domain that EPA considered suitable for the risk evaluation of acute and chronic exposure scenarios in this risk evaluation for TCE. EPA used a previously developed peer-reviewed PBPK model in order to obtain both HECs and HEDs from animal toxicological studies involving either oral or inhalation administration of TCE. The PBPK model does not account for dermal exposure, so EPA relied on traditional route-to-route extrapolation from oral HED values. EPA conservatively assumes 100% absorption through all routes based on reasonably available toxicokinetic data. EPA did not evaluate TCE exposure through the oral route because the route is out of scope for this evaluation (U.S. EPA, 2017d). The volatile properties of TCE

suggest that the majority of dermally deposited TCE would quickly evaporate except in occluded scenarios. Therefore, inhalation is expected to be the predominant route of human exposure for most conditions of use. Dermal exposure was considered for occupational scenarios while accounting for evaporation according to modeling from (Kasting and Miller, 2006) (see Section 2.3.1.2.5). For consumers, dermal exposure was only considered for scenarios resulting in dermal contact with impeded evaporation (See Section 2.3.2.2.2).

4.2.1.1 Representative Points of Departure for Use in Risk Estimation

All PODs listed in Table 3-13 will be used for risk estimation of acute exposure scenarios. For chronic exposure scenarios, due to the large number of relevant endpoints, risks will be assessed using a single endpoint representative of each health domain. EPA considers all of the endpoints identified in Table 3-14 to be similarly relevant to human health hazard from TCE exposure. Therefore risk estimates for chronic exposure scenarios will be presented for only those endpoints representing the most sensitive and robust data within each health domain, with the presumption that evaluation of risks for these endpoints would also account for all other less sensitive yet relevant endpoints. These PODs are presented in Table 4-5. For complete MOE tables displaying risk estimates for all chronic endpoints, see [*Risk Calculator for Occupational Exposures. Docket: EPA-HQ-OPPT-2019-0500]*.

As described in (Section 3.2.6.4), EPA considers the POD for immunosuppression from (Selgrade and Gilmour, 2010) to be the best overall representative endpoint for acute scenarios and autoimmunity from (Keil et al., 2009) to be the best overall representative non-cancer endpoint for chronic scenarios. However, EPA presents risk estimates for all acute endpoints and chronic health domains in Section 4.2.2 and 4.2.3 in order to more accurately describe the range of risk associated with TCE exposure.

Table 4-5: Most Sensitive Endpoints from Each Health Domain for Risk Estimation of Chronic Exposure Scenarios

Target Organ / System	POD Type	Effect	HEC_{99} (ppm)	HED_{99} (mg/kg)	Uncertainty Factors (UFs)	Reference	Data Quality
Developmental Effects	$BMDL_{01}$ = 0.0207mg/kg-bw/day	Congenital heart defects	0.0037	0.0052	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; Total UF=10	(Johnson et al., 2003)	Medium
Kidney	$BMDL_{10}$ = 34 mg/kg-bw/day	Pathology changes in renal tubule	0.025	0.015	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Maltoni et al., 1986)	Medium
Immune System	LOAEL = 0.35 mg/kg-bw/day	Autoimmunity (increased anti-dsDNA and -ssDNA antibodies)	0.033	0.048	UF_S=1; UF_A= 3; UF_H=3; UF_L=3; *Total UF=30*	(Keil et al., 2009)	High
Reproductive System	$BMDL_{10}$ = 1.4 ppm	Decreased normal sperm morphology and hyper-zoospermia	0.5	0.73	UF_S=10; UF_A= 1; UF_H=3; UF_L=1; *Total UF=30*	(Chia et al., 1996)	Medium
Nervous System	LOAEL = 12 ppm	Significant decreases in wakefulness	4.8	6.5	UF_S=3; UF_A= 3; UF_H=3; UF_L=10; *Total UF=300*	(Arito et al., 1994)	Medium
Liver	$BMDL_{10}$= 21.6 ppm	Increased liver/body weight ratio and cytotoxicity/hypertrophy	9.1	7.9	UF_S=1; UF_A= 3; UF_H=3; UF_L=1; *Total UF=10*	(Kjellstrand et al., 1983)	Medium

HEC/HED_{99} values will be used for risk estimation. These upper-end outputs from the PBPK model are expected to be protective of susceptible subpopulations, accounting for the majority of identified toxicokinetic human variability. The toxicokinetic metric of the interspecies and intraspecies uncertainty

factors has been eliminated based on the use of these data-derived values, resulting in a reduced UF_A and UF_H of 3.

4.2.2 Risk Estimation for Occupational Exposures by Exposure Scenario

Risk estimates via inhalation and dermal exposure are provided below for workers and ONUs following acute (single day), chronic (40-year), or lifetime (78 year) TCE exposure. Inhalation risk estimates are based on either monitoring or modeling exposure data. Non-cancer endpoints were applied to acute and chronic exposures while cancer risk estimates are provided for adjusted lifetime exposure. Both are presented for exposure scenarios where both data types are reasonably available. All dermal risk estimates are based on modeling data as discussed in Section 2.3.1.2.5. Although generally ONU exposures are expected to be less than workers, when sufficient data was not reasonably available for quantifying ONU exposures EPA provided risk estimates for ONUs based on assuming that ONU exposure may be comparable to worker central-tendency values. This is a health-protective assumption. When reasonably available, inhalation risk estimates are presented based on both monitoring and modeling data. Otherwise, risk estimates are presented for the type of inhalation exposure data that was reasonably available. All dermal risk estimates are based on exposure modeling data. For details on the exposure estimates for each exposure scenario, see Section 2.3.1.

For occupational scenarios, EPA evaluated the impact of potential respirator use based on respirator APF of 10 and 50 in the below tables. The calculated non-cancer MOE or extra cancer risk with respirator use is then compared to the benchmark MOE to determine the level of APF required to mitigate risk for all health domains. EPA does not evaluate respirator use for occupational non-users because they do not directly handle TCE and EPA assumes that they are unlikely to consistently wear respirators. In addition, EPA believes small commercial facilities performing spot cleaning, wipe cleaning, and other related commercial uses as well as commercial printing and copying are unlikely to have a respiratory protection program. For dermal protection, EPA evaluated the impact of glove use up to the maximum possible PF of 20 for industrial scenarios and PF of 10 for commercial scenarios (see Table 2-20). For complete MOE tables displaying risk estimates for all endpoints and all PPE options, see [*Risk Calculator for Occupational Exposures. Docket: EPA-HQ-OPPT-2019-0500]*.

498 **Table 4-6. Occupational Risk Estimation - Manufacturing**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.3E-03**	**4.3E-02**	**0.21**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.0E-02**	**0.30**	**1.5**	**3.0E-02**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.5**	**34.8**	173.9	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**24.0**	239.9	1,199.4	**24.0**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	26.7	266.6	1,333.0	-	12.2	60.8	121.5	243.0
		Central Tendency	183.9	1,839.1	9,195.6	183.9	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**2.0**	**20.2**	100.8	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**13.9**	139.1	695.7	**13.9**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	15.4	154.0	770.0	-	**5.0**	25.0	50.1	100.1
		Central Tendency	106.2	1,062.4	5,311.8	106.2	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**4.2E-02**	**0.42**	**2.1**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**0.29**	**2.9**	14.6	**0.29**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**8.1**	**81.2**	406.2	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**56.0**	560.4	2,801.8	**56.0**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**5.6E-02**	**0.56**	**2.8**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**0.39**	**3.9**	**19.3**	**0.39**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.85**	**8.5**	42.3	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**5.8**	58.4	291.9	**5.8**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**6.3E-03**	**6.3E-02**	**0.31**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.3E-02**	**0.43**	**2.2**	**4.3E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**6.7E-03**	**6.7E-04**	**1.3E-04**	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**7.5E-04**	7.5E-05	1.5E-05	**7.5E-04**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are considered plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE results for *Manufacturing* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-6.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and for multiple endpoints at both high-end and central tendency inhalation exposure even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. Risk estimates remained above the benchmark for cancer at high-end inhalation exposure even when assuming the highest plausible APF. Risk estimates remained above the benchmark for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

521 **Table 4-7. Occupational Risk Estimation - Processing as a Reactant**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.3E-03**	**4.3E-02**	**0.21**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.0E-02**	**0.30**	**1.5**	**3.0E-02**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.5**	**34.8**	173.9	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**24.0**	239.9	1,199.4	**24.0**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	26.7	266.6	1,333.0	-	12.2	60.8	121.5	243.0
		Central Tendency	183.9	1,839.1	9,195.6	183.9	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**2.0**	**20.2**	100.8	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**13.9**	139.1	695.7	**13.9**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	15.4	154.0	770.0	-	**5.0**	25.0	50.1	100.1
		Central Tendency	106.2	1,062.4	5,311.8	106.2	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**4.2E-02**	**0.42**	**2.1**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**0.29**	**2.9**	14.6	**0.29**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**8.1**	**81.2**	406.2	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**56.0**	560.4	2,801.8	**56.0**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**5.6E-02**	**0.56**	**2.8**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**0.39**	**3.9**	**19.3**	**0.39**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.85**	**8.5**	42.3	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**5.8**	58.4	291.9	**5.8**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**6.3E-03**	**6.3E-02**	**0.31**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.3E-02**	**0.43**	**2.2**	**4.3E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**6.7E-03**	**6.7E-04**	**1.3E-04**	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**7.5E-04**	7.5E-05	1.5E-05	**7.5E-04**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are considered plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE results for *Processing as a Reactant* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-7.

Acute Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and for multiple endpoints at both high-end and central tendency inhalation exposure even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

Cancer Risk Estimates:
Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. Risk estimates remained above the benchmark for cancer at high-end inhalation exposure even when assuming the highest plausible APF. Risk estimates remained above the benchmark for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

545 **Table 4-8. Occupational Risk Estimation - Batch Open Top Vapor Degreasing - Inhalation Monitoring Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**1.4E-04**	**1.4E-03**	**7.1E-03**	**1.2E-03**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**8.0E-04**	**8.0E-03**	**4.0E-02**	**1.0E-02**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.12**	**1.2**	**5.8**	**0.99**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**0.65**	**6.5**	**32.6**	**8.1**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**0.89**	**8.9**	44.4	**7.6**	12.2	60.8	121.5	243.0
		Central Tendency	**5.0**	50.0	250.0	62.3	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**6.7E-02**	**0.67**	**3.4**	**0.57**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.38**	**3.8**	**18.9**	**4.7**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**0.51**	**5.1**	25.6	**4.4**	**5.0**	25.0	50.1	100.1
		Central Tendency	**2.9**	28.9	144.4	36.0	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**1.4E-03**	**1.4E-02**	**7.0E-02**	**1.2E-02**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**7.9E-03**	**7.9E-02**	**0.40**	**9.9E-02**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.27**	**2.7**	**13.5**	**2.3**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**1.5**	**15.2**	**76.2**	**19.0**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**1.9E-03**	**1.9E-02**	**9.3E-02**	**1.6E-02**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**1.0E-02**	**0.10**	**0.52**	**0.13**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**2.8E-02**	**0.28**	**1.4**	**0.24**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**0.16**	**1.6**	**7.9**	**2.0**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**2.1E-04**	**2.1E-03**	**1.0E-02**	**1.8E-03**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**1.2E-03**	**1.2E-02**	**5.9E-02**	**1.5E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.20**	**2.0E-02**	**4.0E-03**	**2.3E-02**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**2.8E-02**	**2.8E-03**	**5.5E-04**	**2.2E-03**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

Table 4-9. Occupational Risk Estimation - Batch Open Top Vapor Degreasing - Inhalation Modeling Data

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**2.9E-05**	**2.9E-04**	**1.4E-03**	**4.7E-05**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.2E-04**	**3.2E-03**	**1.6E-02**	**6.1E-04**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**2.3E-02**	**0.23**	**1.2**	**3.8E-02**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**0.26**	**2.6**	**12.9**	**0.50**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**0.18**	**1.8**	**8.9**	**0.29**	12.2	60.8	121.5	243.0
		Central Tendency	**2.0**	19.8	99.1	**3.8**	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**1.3E-02**	**0.13**	**0.67**	**2.2E-02**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.15**	**1.5**	**7.5**	**0.29**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**0.10**	**1.0**	**5.1**	**0.17**	**5.0**	25.0	50.1	100.1
		Central Tendency	**1.1**	11.4	57.2	**2.2**	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**2.8E-04**	**2.8E-03**	**1.4E-02**	**4.6E-04**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**3.1E-03**	**3.1E-02**	**0.16**	**6.0E-03**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**5.4E-02**	**0.54**	**2.7**	**8.9E-02**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**0.60**	**6.0**	**30.2**	**1.2**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**3.7E-04**	**3.7E-03**	**1.9E-02**	**6.1E-04**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**4.1E-03**	**4.1E-02**	**0.21**	**8.0E-03**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**5.6E-03**	**5.6E-02**	**0.28**	**9.3E-03**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**6.3E-02**	**0.63**	**3.1**	**0.12**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**4.2E-05**	**4.2E-04**	**2.1E-03**	**6.9E-05**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.6E-04**	**4.6E-03**	**2.3E-02**	**8.9E-04**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.78**	**7.8E-02**	**1.6E-02**	**0.46**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**6.5E-02**	**6.5E-03**	**1.3E-03**	**3.4E-02**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are considered plausible for this exposure scenario.

MOE results for *Batch Open Top Vapor Degreasing* utilized both monitoring and modeling inhalation exposure data (with dermal modeling). Results are presented in Table 4-8 and Table 4-9.

Acute Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints based on monitoring and for all endpoints based on modeling at both high-end and central tendency inhalation exposure levels. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for multiple endpoints at both inhalation exposure levels even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints based on monitoring and for all endpoints based on modeling at both high-end and central tendency inhalation exposure levels. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Based on both monitoring and modeling data, extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Based on both monitoring and modeling data, risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Based on both monitoring and modeling data, risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

OSHA PEL considerations

The OSHA PEL for TCE is 100 ppm (8hr TWA). The monitoring dataset for this OES included some data points above the PEL value. In an alternative approach, EPA calculated central tendency and high end values for the measurements lower than the PEL. This resulted in a reduction of the high-end acute exposure estimate from 25.92ppm to 19.23 ppm and the central tendency acute exposure estimate from 4.60 ppm to 4.26 ppm. Chronic high-end and central tendency exposures are reduced from 17.75 ppm and 3.15 ppm to 13.17 ppm and 2.92 ppm, respectively. Lifetime exposures are reduced from 9.10 ppm and 1.25 ppm to 6.75 ppm and 1.15 ppm, respectively. The reduced exposures do not significantly affect the risk estimates, since exposures were only reduced by up to ~30%. Based on PEL-capped exposure estimates, the acute and chronic central tendency MOEs for the congenital heart defects endpoint (with benchmark MOE = 10) are 8.7E-04 and 1.3E-03, respectively. The central tendency cancer extra risk (benchmark = 1E-04) is 2.6E-02. Therefore, the MOEs remains orders of magnitude below the benchmark MOE (or above the benchmark for cancer risk) when using only PEL-capped exposure estimates. Full details are provided in [*Occupational Risk Estimate Calculator. Docket # EPA-HQ-OPPT-2019-0500]*.

586 **Table 4-10. Occupational Risk Estimation - Batch Closed-Loop Vapor Degreasing**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**7.6E-03**	**7.6E-02**	**0.38**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**2.4E-02**	**0.24**	**1.2**	**2.4E-02**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**6.2**	**61.9**	309.5	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**19.7**	196.6	983.0	**19.7**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	47.5	474.5	2,372.5	-	12.2	60.8	121.5	243.0
		Central Tendency	150.7	1,507.3	7,536.5	150.7	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**3.6**	35.9	179.5	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**11.4**	114.0	570.1	**11.4**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	27.4	274.1	1,370.5	-	**5.0**	25.0	50.1	100.1
		Central Tendency	87.1	870.7	4,353.5	87.1	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**7.5E-02**	**0.75**	**3.8**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**0.24**	**2.4**	12.0	**0.24**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**14.5**	**144.6**	722.9	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**45.9**	459.3	2,296.3	**45.9**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**9.9E-02**	**0.99**	**5.0**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**0.32**	**3.2**	**15.8**	**0.32**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**1.5**	**15.1**	75.3	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**4.8**	47.8	239.2	**4.8**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**1.1E-02**	**0.11**	**0.56**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**3.5E-02**	**0.35**	**1.8**	**3.5E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**3.7E-03**	**3.7E-04**	7.5E-05	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**9.1E-04**	9.1E-05	1.8E-05	**9.1E-04**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE results for *Batch Closed-Loop Vapor Degreasing* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-10.

Acute Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and for immunotoxicity at both high-end and central tendency inhalation exposure even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

Cancer Risk Estimates:
Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. Risk estimates were not above the benchmark for high-end inhalation exposure when assuming APF = 50 or for central tendency inhalation exposure when assuming APF = 10. Risk estimates remained above the benchmark for multiple endpoints at both dermal exposure levels even when assuming the highest plausible glove PF.

613 **Table 4-11. Occupational Risk Estimation - Conveyorized Vapor Degreasing - Inhalation Monitoring Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**2.3E-04**	**2.3E-03**	**1.1E-02**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.4E-04**	**3.4E-03**	**1.7E-02**	**3.4E-04**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.19**	**1.9**	**9.3**	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**0.28**	**2.8**	**13.9**	**0.28**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**1.4**	14.3	71.4	-	12.2	60.8	121.5	243.0
		Central Tendency	**2.1**	21.3	106.5	**2.1**	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.11**	**1.1**	**5.4**	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.16**	**1.6**	**8.1**	**0.16**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**0.83**	**8.3**	41.3	-	**5.0**	25.0	50.1	100.1
		Central Tendency	**1.2**	12.3	61.5	**1.2**	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**2.3E-03**	**2.3E-02**	**0.11**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**3.4E-03**	**3.4E-02**	**0.17**	**3.4E-03**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.44**	**4.4**	**21.8**	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**0.65**	**6.5**	**32.5**	**0.65**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**3.0E-03**	**3.0E-02**	**0.15**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**4.5E-03**	**4.5E-02**	**0.22**	**4.5E-03**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**4.5E-02**	**0.45**	**2.3**	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**6.8E-02**	**0.68**	**3.4**	**6.8E-02**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**3.4E-04**	**3.4E-03**	**1.7E-02**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**5.0E-04**	**5.0E-03**	**2.5E-02**	**5.0E-04**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.12**	**1.2E-02**	**2.5E-03**	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**6.5E-02**	**6.5E-03**	**1.3E-03**	**6.5E-02**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

614 **Table 4-12. Occupational Risk Estimation - Conveyorized Vapor Degreasing - Inhalation Modeling Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**3.6E-06**	**3.6E-05**	**1.8E-04**	**5.9E-06**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**2.7E-04**	**2.7E-03**	**1.4E-02**	**4.8E-04**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.0E-03**	**3.0E-02**	**0.15**	**4.8E-03**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**0.22**	**2.2**	**11.0**	**0.39**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**2.3E-02**	**0.23**	**1.1**	**3.7E-02**	12.2	60.8	121.5	243.0
		Central Tendency	**1.7**	16.9	84.6	**3.0**	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**1.7E-03**	**1.7E-02**	**8.6E-02**	**2.8E-03**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.13**	**1.3**	**6.4**	**0.22**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**1.3E-02**	**0.13**	**0.65**	**2.1E-02**	**5.0**	25.0	50.1	100.1
		Central Tendency	**0.98**	**9.8**	48.8	**1.7**	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**3.6E-05**	**3.6E-04**	**1.8E-03**	**5.8E-05**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**2.7E-03**	**2.7E-02**	**0.13**	**4.7E-03**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**6.9E-03**	**6.9E-02**	**0.35**	**1.1E-02**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**0.52**	**5.2**	**25.8**	**0.90**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**4.7E-05**	**4.7E-04**	**2.4E-03**	**7.7E-05**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**3.5E-03**	**3.5E-02**	**0.18**	**6.2E-03**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**7.2E-04**	**7.2E-03**	**3.6E-02**	**1.2E-03**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**5.4E-02**	**0.54**	**2.7**	**9.4E-02**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**5.3E-06**	**5.3E-05**	**2.7E-04**	**8.6E-06**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.0E-04**	**4.0E-03**	**2.0E-02**	**6.9E-04**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**6.1**	**0.61**	**0.12**	**3.7**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**0.12**	**1.2E-02**	**2.3E-03**	**7.9E-02**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

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MOE results for *Conveyorized Vapor Degreasing* utilized both monitoring and modeling inhalation exposure data (with dermal modeling). Results are presented in Table 4-11 and Table 4-12.

Acute Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via inhalation and for most endpoints via the dermal route. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were below the benchmark MOE for all endpoints at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for multiple endpoints at both inhalation exposure levels even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were below the benchmark MOE for all endpoints at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Based on both monitoring and modeling data, extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were above the benchmark at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

647 **Table 4-13. Occupational Risk Estimation - Web Vapor Degreasing**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**7.9E-04**	**7.9E-03**	**3.9E-02**	**1.2E-03**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**1.9E-03**	**1.9E-02**	**9.3E-02**	**3.5E-03**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.64**	**6.4**	**31.8**	**0.94**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**1.5**	**15.1**	**75.7**	**2.9**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**4.9**	48.8	244.0	**7.2**	12.2	60.8	121.5	243.0
		Central Tendency	11.6	116.1	580.4	22.1	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.37**	**3.7**	**18.5**	**0.55**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.88**	**8.8**	43.9	**1.7**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**2.8**	28.2	140.9	**4.2**	**5.0**	25.0	50.1	100.1
		Central Tendency	**6.7**	67.1	335.3	12.7	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**7.7E-03**	**7.7E-02**	**0.39**	**1.1E-02**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**1.8E-02**	**0.18**	**0.92**	**3.5E-02**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**1.5**	**14.9**	**74.3**	**2.2**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**3.5**	**35.4**	**176.8**	**6.7**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**1.0E-02**	**0.10**	**0.51**	**1.5E-02**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**2.4E-02**	**0.24**	**1.2**	**4.6E-02**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.15**	**1.5**	**7.7**	**0.23**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**0.37**	**3.7**	**18.4**	**0.70**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**1.1E-03**	**1.1E-02**	**5.7E-02**	**1.7E-03**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**2.7E-03**	**2.7E-02**	**0.14**	**5.2E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**2.9E-02**	**2.9E-03**	**5.8E-04**	**1.9E-02**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**1.1E-02**	**1.1E-03**	**2.3E-04**	**5.9E-03**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

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MOE results for *Web Vapor Degreasing* utilized modeling inhalation exposure data (with dermal modeling) and are presented in Table 4-13.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at the central tendency inhalation exposure level. MOEs remained below the benchmark MOE for multiple endpoints at both inhalation exposure levels even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at the central tendency inhalation exposure level. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at the central tendency inhalation exposure level. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

675 **Table 4-14. Occupational Risk Estimation - Cold Cleaning**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**1.9E-04**	**1.9E-03**	**9.7E-03**	**3.2E-04**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.3E-03**	**3.3E-02**	**0.17**	**6.0E-03**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.16**	**1.6**	**7.9**	**0.26**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**2.7**	**27.0**	135.1	**4.9**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**1.2**	12.1	60.3	**2.0**	12.2	60.8	121.5	243.0
		Central Tendency	20.7	207.2	1,036.0	37.5	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**9.1E-02**	**0.91**	**4.6**	**0.15**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**1.6**	**15.7**	78.4	**2.8**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**0.69**	**6.9**	34.7	**1.2**	**5.0**	25.0	50.1	100.1
		Central Tendency	12.0	119.7	598.7	21.7	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**1.9E-03**	**1.9E-02**	**9.5E-02**	**3.2E-03**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**3.3E-02**	**0.33**	**1.6**	**6.0E-02**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.37**	**3.7**	**18.3**	**0.61**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**6.3**	**63.2**	315.8	**11.4**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**2.5E-03**	**2.5E-02**	**0.13**	**4.2E-03**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**4.3E-02**	**0.43**	**2.2**	**7.9E-02**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**3.8E-02**	**0.38**	**1.9**	**6.3E-02**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**0.66**	**6.6**	32.9	**1.2**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**2.8E-04**	**2.8E-03**	**1.4E-02**	**4.7E-04**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.9E-03**	**4.9E-02**	**0.24**	**8.8E-03**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.11**	**1.1E-02**	**2.3E-03**	**6.9E-02**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**6.2E-03**	**6.2E-04**	**1.2E-04**	**3.3E-03**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

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MOE results for *Cold Cleaning* utilized modeling inhalation exposure data (with dermal modeling) and are presented in Table 4-14.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

700 **Table 4-15. Occupational Risk Estimation - Aerosol Applications**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.6E-04**	**4.6E-03**	**2.3E-02**	**1.1E-02**	**1.4E-03**	**7.2E-03**	**1.4E-02**	**2.9E-02**
		Central Tendency	**1.5E-03**	**1.5E-02**	**7.3E-02**	**7.9E-02**	**4.3E-03**	**2.2E-02**	**4.3E-02**	**8.6E-02**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.38**	**3.8**	**18.8**	**8.7**	**1.1**	**5.7**	**11.3**	**22.7**
		Central Tendency	**1.2**	**11.8**	**59.0**	**64.3**	**3.4**	**17.0**	**34.0**	**68.0**
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**2.9**	28.8	143.9	66.3	**7.7**	38.7	77.4	154.8
		Central Tendency	**9.0**	90.4	452.2	492.9	23.2	116.1	232.2	464.3
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.22**	**2.2**	**10.9**	**5.0**	**0.76**	**3.8**	**7.6**	**15.1**
		Central Tendency	**0.68**	**6.8**	34.2	37.3	**2.3**	**11.4**	**22.7**	45.4
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**1.7**	16.6	83.1	38.2	**3.2**	15.9	31.9	63.8
		Central Tendency	**5.2**	52.3	261.3	284.4	**9.6**	47.8	95.6	191.3
Kidney (Maltoni et al., 1986)	10	High End	**4.6E-03**	**4.6E-02**	**0.23**	**0.11**	**6.1E-03**	**3.0E-02**	**6.1E-02**	**0.12**
		Central Tendency	**1.4E-02**	**0.14**	**0.72**	**0.78**	**1.8E-02**	**9.1E-02**	**0.18**	**0.36**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.88**	**8.8**	**43.8**	**20.2**	**2.6**	**13.1**	**26.2**	**52.5**
		Central Tendency	**2.8**	**27.6**	**137.9**	**150.0**	**7.9**	**39.3**	**78.7**	**157.4**
Immunotoxicity (Keil et al., 2009)	30	High End	**6.0E-03**	**6.0E-02**	**0.30**	**0.14**	**1.9E-02**	**9.7E-02**	**0.19**	**0.39**
		Central Tendency	**1.9E-02**	**0.19**	**0.95**	**1.0**	**5.8E-02**	**0.29**	**0.58**	**1.2**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**9.1E-02**	**0.91**	**4.6**	**2.1**	**0.29**	**1.5**	**2.9**	**5.9**
		Central Tendency	**0.29**	**2.9**	**14.4**	**15.6**	**0.88**	**4.4**	**8.8**	**17.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**6.8E-04**	**6.8E-03**	**3.4E-02**	**1.6E-02**	**2.1E-03**	**1.0E-02**	**2.1E-02**	**4.2E-02**
		Central Tendency	**2.1E-03**	**2.1E-02**	**0.11**	**0.12**	**6.3E-03**	**3.1E-02**	**6.3E-02**	**0.13**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**4.9E-02**	**4.9E-03**	**9.7E-04**	**2.0E-03**	**5.9E-02**	**1.2E-02**	**5.9E-03**	**2.9E-03**
		Central Tendency	**1.4E-02**	**1.4E-03**	**2.9E-04**	**2.6E-04**	**1.5E-02**	**3.0E-03**	**1.5E-03**	**7.6E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

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MOE results for *Aerosol Applications* utilized modeling inhalation exposure data (with dermal modeling) and are presented in Table 4-15.

Acute Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:
Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

724 **Table 4-16. Occupational Risk Estimation - Spot Cleaning and Wipe Cleaning (and Other Commercial Uses) - Inhalation Monitoring Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**3.9E-03**	**3.9E-02**	**0.19**	-	**1.4E-03**	**7.2E-03**	**1.4E-02**	N/A[2]
		Central Tendency	**2.9E-02**	**0.29**	**1.4**	**2.9E-02**	**4.3E-03**	**2.2E-02**	**4.3E-02**	
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.2**	**31.6**	157.8	-	**1.1**	**5.7**	**11.3**	
		Central Tendency	**23.5**	235.1	1,175.3	**23.5**	**3.4**	**17.0**	**34.0**	
Developmental - Mortality (Narotsky et al., 1995)	10	High End	24.2	242.0	1,210.1	-	**7.7**	38.7	77.4	
		Central Tendency	180.2	1,802.2	9,010.9	180.2	23.2	116.1	232.2	
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**1.8**	**18.3**	91.5	-	**0.76**	**3.8**	**7.6**	
		Central Tendency	**13.6**	136.3	681.7	**13.6**	**2.3**	**11.4**	**22.7**	
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	13.5	135.5	677.3	-	**2.7**	13.6	27.2	N/A[2]
		Central Tendency	100.9	1,008.7	5,043.7	100.9	**9.3**	46.3	92.7	
Kidney (Maltoni et al., 1986)	10	High End	**3.7E-02**	**0.37**	**1.9**	-	**5.2E-03**	**2.6E-02**	**5.2E-02**	
		Central Tendency	**0.28**	**2.8**	13.9	**0.28**	**1.8E-02**	**8.8E-02**	**0.18**	
Neurotoxicity (Arito et al., 1994)	300	High End	**7.1**	**71.5**	357.3	-	**2.2**	**11.2**	**22.4**	
		Central Tendency	**53.2**	532.1	2,660.4	**53.2**	**7.6**	**38.1**	**76.3**	
Immunotoxicity (Keil et al., 2009)	30	High End	**4.9E-02**	**0.49**	**2.5**	-	**1.7E-02**	**8.3E-02**	**0.17**	
		Central Tendency	**0.37**	**3.7**	**18.3**	**0.37**	**5.6E-02**	**0.28**	**0.56**	
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.74**	**7.4**	37.2	-	**0.25**	**1.3**	**2.5**	
		Central Tendency	**5.5**	55.4	277.1	**5.5**	**0.86**	**4.3**	**8.6**	
Developmental Toxicity (Johnson et al., 2003)	10	High End	**5.5E-03**	**5.5E-02**	**0.28**	-	**1.8E-03**	**9.0E-03**	**1.8E-02**	
		Central Tendency	**4.1E-02**	**0.41**	**2.1**	**4.1E-02**	**6.1E-03**	**3.1E-02**	**6.1E-02**	
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**7.6E-03**	**7.6E-04**	**1.5E-04**	-	**6.9E-02**	**1.4E-02**	**6.9E-03**	N/A[2]
		Central Tendency	**7.9E-04**	7.9E-05	1.6E-05	**7.9E-04**	**1.6E-02**	**3.1E-03**	**1.6E-03**	

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.
[2] Glove PF =20 is only applicable to industrial settings (See Section 2.3.1).

725 **Table 4-17. Occupational Risk Estimation - Spot Cleaning and Wipe Cleaning (and Other Commercial Uses) - Inhalation Modeling Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.0E-03**	**4.0E-02**	**0.20**	**6.3E-03**	**1.4E-03**	**7.2E-03**	**1.4E-02**	N/A[1]
		Central Tendency	**1.2E-02**	**0.12**	**0.58**	**2.3E-02**	**4.3E-03**	**2.2E-02**	**4.3E-02**	
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.2**	**32.5**	162.5	**5.1**	**1.1**	**5.7**	**11.3**	
		Central Tendency	**9.4**	**93.7**	468.3	**18.8**	**3.4**	**17.0**	**34.0**	
Developmental - Mortality (Narotsky et al., 1995)	10	High End	24.9	249.1	1,245.5	39.4	**7.7**	38.7	77.4	
		Central Tendency	71.8	718.0	3,590.0	144.1	23.2	116.1	232.2	
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**1.9**	**18.8**	94.2	**3.0**	**0.76**	**3.8**	**7.6**	
		Central Tendency	**5.4**	54.3	271.6	**10.9**	**2.3**	**11.4**	**22.7**	
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	14.0	139.6	697.9	22.1	**2.7**	13.6	27.2	N/A[1]
		Central Tendency	40.3	402.7	2,013.3	80.5	**9.3**	46.3	92.7	
Kidney (Maltoni et al., 1986)	10	High End	**3.8E-02**	**0.38**	**1.9**	**6.1E-02**	**5.2E-03**	**2.6E-02**	**5.2E-02**	
		Central Tendency	**0.11**	**1.1**	**5.5**	**0.22**	**1.8E-02**	**8.8E-02**	**0.18**	
Neurotoxicity (Arito et al., 1994)	300	High End	**7.4**	**73.6**	368.1	**11.7**	**2.2**	**11.2**	**22.4**	
		Central Tendency	**21.2**	**212.4**	1,061.9	**42.5**	**7.6**	**38.1**	**76.3**	
Immunotoxicity (Keil et al., 2009)	30	High End	**5.1E-02**	**0.51**	**2.5**	**8.0E-02**	**1.7E-02**	**8.3E-02**	**0.17**	
		Central Tendency	**0.15**	**1.5**	**7.3**	**0.29**	**5.6E-02**	**0.28**	**0.56**	
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.77**	**7.7**	38.3	**1.2**	**0.25**	**1.3**	**2.5**	
		Central Tendency	**2.2**	**22.1**	110.6	**4.4**	**0.86**	**4.3**	**8.6**	
Developmental Toxicity (Johnson et al., 2003)	10	High End	**5.7E-03**	**5.7E-02**	**0.28**	**9.0E-03**	**1.8E-03**	**9.0E-03**	**1.8E-02**	
		Central Tendency	**1.6E-02**	**0.16**	**0.82**	**3.3E-02**	**6.1E-03**	**3.1E-02**	**6.1E-02**	
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**5.8E-03**	**5.8E-04**	**1.2E-04**	**3.6E-03**	**6.9E-02**	**1.4E-02**	**6.9E-03**	N/A[1]
		Central Tendency	**1.8E-03**	**1.8E-04**	3.7E-05	**9.2E-04**	**1.6E-02**	**3.1E-03**	**1.6E-03**	

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] Glove PF =20 is only applicable to industrial settings (See Section 2.3.1).

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MOE calculations for *Spot Cleaning and Wipe Cleaning* utilized both monitoring and modeling inhalation exposure data (with dermal modeling). This data also applies to the exposure scenario of *Other Commercial Uses.* Results are presented in Table 4-16 and Table 4-17.

Acute Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via inhalation and for multiple endpoints via the dermal route even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via both inhalation and dermal routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Based on both monitoring and modeling data, extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers based on monitoring data. ONU risk estimates were above the benchmark at both high-end and central tendency inhalation exposure levels based on modeling data. Based on both monitoring and modeling data, risk estimates remained above the benchmark for cancer at high-end inhalation exposure levels and both dermal exposure levels even when assuming the highest plausible APF and glove PF protection. Risk estimates were not above the benchmark for central tendency inhalation exposure when assuming APF = 10 based on monitoring data or when assuming APF = 50 based on modeling data.

PPE Considerations

EPA is presenting risk estimates for respiratory protection up to APF = 50 as a what-if scenario, however EPA believes that small commercial facilities performing spot cleaning, wipe cleaning, and other related commercial uses are unlikely to have a respiratory protection program. Therefore, the use of respirators is unlikely for workers in these facilities.

761 **Table 4-18. Occupational Risk Estimation - Formulation of Aerosol and Non-Aerosol Products**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**9.7E-03**	**9.7E-02**	**0.49**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	22.4	224.3	1,121.3	22.4	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**7.9**	**78.9**	394.7	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	18,182.7	181,827.5	909,137.3	18,182.7	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	60.5	605.3	3,026.3	-	12.2	60.8	121.5	243.0
		Central Tendency	139,401.1	1,394,010.5	6,970,052.6	139,401.1	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**4.6**	45.8	228.9	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	10,546.0	105,459.9	527,299.6	10,546.0	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	35.0	349.6	1,748.2	-	**5.0**	25.0	50.1	100.1
		Central Tendency	80,525.3	805,253.2	4,026,266.0	80,525.3	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**9.6E-02**	**0.96**	**4.8**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	221.2	2,212.2	11,061.2	221.2	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**18.4**	**184.4**	922.1	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	42,474.9	424,748.9	2,123,744.7	42,474.9	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**0.13**	**1.3**	**6.3**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	292.0	2,920.1	14,600.7	292.0	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**1.9**	**19.2**	96.1	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	4,424.5	44,244.7	221,223.4	4,424.5	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**1.4E-02**	**0.14**	**0.71**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	32.7	327.4	1,637.1	32.7	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**2.9E-03**	**2.9E-04**	5.9E-05	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	9.9E-07	9.9E-08	2.0E-08	9.9E-07	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

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MOE results for *Formulation of Aerosol and Non-Aerosol Products* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-18.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for congenital heart defects at high-end inhalation exposure even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and at both dermal exposure levels even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at at high-end inhalation exposures, but risk estimates were below the benchmark for cancer at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. Risk estimates were above the benchmark at both dermal exposure levels. Risk estimates were not above the benchmark for high-end inhalation exposure when assuming APF = 50. Risk estimates remained above the benchmark for cancer at both dermal exposure levels even when assuming the highest plausible glove PF protection.

791 **Table 4-19. Occupational Risk Estimation - Repackaging**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**9.7E-03**	**9.7E-02**	**0.49**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	22.4	224.3	1,121.3	22.4	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**7.9**	**78.9**	394.7	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	18,182.7	181,827.5	909,137.3	18,182.7	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	60.5	605.3	3,026.3	-	12.2	60.8	121.5	243.0
		Central Tendency	139,401.1	1,394,010.5	6,970,052.6	139,401.1	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**4.6**	45.8	228.9	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	10,546.0	105,459.9	527,299.6	10,546.0	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	35.0	349.6	1,748.2	-	**5.0**	25.0	50.1	100.1
		Central Tendency	80,525.3	805,253.2	4,026,266.0	80,525.3	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**9.6E-02**	**0.96**	**4.8**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	221.2	2,212.2	11,061.2	221.2	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**18.4**	**184.4**	922.1	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	42,474.9	424,748.9	2,123,744.7	42,474.9	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**0.13**	**1.3**	**6.3**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	292.0	2,920.1	14,600.7	292.0	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**1.9**	**19.2**	96.1	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	4,424.5	44,244.7	221,223.4	4,424.5	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**1.4E-02**	**0.14**	**0.71**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	32.7	327.4	1,637.1	32.7	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**2.9E-03**	**2.9E-04**	5.9E-05	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	9.9E-07	9.9E-08	2.0E-08	9.9E-07	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

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MOE results for *Repackaging* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-19.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and at both dermal exposure levels even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at at high-end inhalation exposures, but risk estimates were below the benchmark for cancer at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. Risk estimates were above the benchmark at both dermal exposure levels. Risk estimates were not above the benchmark for high tendency inhalation exposure when assuming APF = 50. Risk estimates remained above the benchmark for cancer at both dermal exposure levels even when assuming the highest plausible glove PF protection.

828 **Table 4-20. Occupational Risk Estimation - Metalworking Fluids - Inhalation Monitoring Data**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**1.5E-04**	**1.5E-03**	**7.4E-03**	-	**2.8E-03**	**1.4E-02**	**2.8E-02**	**5.6E-02**
		Central Tendency	**1.6E-04**	**1.6E-03**	**8.0E-03**	**1.6E-04**	**8.5E-03**	**4.2E-02**	**8.5E-02**	**0.17**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.12**	**1.2**	**6.0**	-	**2.2**	**11.1**	**22.2**	**44.5**
		Central Tendency	**0.13**	**1.3**	**6.5**	**0.13**	**6.7**	**33.4**	**66.7**	133.4
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**0.92**	**9.2**	45.8	-	15.2	75.9	151.9	303.8
		Central Tendency	**0.99**	**9.9**	49.5	**0.99**	45.6	227.8	455.6	911.3
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**6.9E-02**	**0.69**	**3.5**	-	**1.5**	**7.4**	**14.9**	**29.7**
		Central Tendency	**7.5E-02**	**0.75**	**3.7**	**7.5E-02**	**4.5**	**22.3**	44.6	89.2
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**0.53**	**5.3**	26.4	-	**6.3**	31.3	62.6	125.1
		Central Tendency	**0.57**	**5.7**	28.6	**0.57**	18.8	93.8	187.7	375.4
Kidney (Maltoni et al., 1986)	10	High End	**1.5E-03**	**1.5E-02**	**7.3E-02**	-	**1.2E-02**	**5.9E-02**	**0.12**	**0.24**
		Central Tendency	**1.6E-03**	**1.6E-02**	**7.9E-02**	**1.6E-03**	**3.6E-02**	**0.18**	**0.36**	**0.71**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.28**	**2.8**	**13.9**	-	**5.1**	**25.7**	**51.5**	**103.0**
		Central Tendency	**0.30**	**3.0**	**15.1**	**0.30**	**15.4**	**77.2**	**154.4**	308.9
Immunotoxicity (Keil et al., 2009)	30	High End	**1.9E-03**	**1.9E-02**	**9.6E-02**	-	**3.8E-02**	**0.19**	**0.38**	**0.76**
		Central Tendency	**2.1E-03**	**2.1E-02**	**0.10**	**2.1E-03**	**0.11**	**0.57**	**1.1**	**2.3**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**2.9E-02**	**0.29**	**1.5**	-	**0.58**	**2.9**	**5.8**	**11.6**
		Central Tendency	**3.1E-02**	**0.31**	**1.6**	**3.1E-02**	**1.7**	**8.7**	**17.3**	34.7
Developmental Toxicity (Johnson et al., 2003)	10	High End	**2.2E-04**	**2.2E-03**	**1.1E-02**	-	**4.1E-03**	**2.1E-02**	**4.1E-02**	**8.2E-02**
		Central Tendency	**2.3E-04**	**2.3E-03**	**1.2E-02**	**2.3E-04**	**1.2E-02**	**6.2E-02**	**0.12**	**0.25**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.19**	**1.9E-02**	**3.9E-03**	-	**3.0E-02**	**6.0E-03**	**3.0E-03**	**1.5E-03**
		Central Tendency	**0.14**	**1.4E-02**	**2.8E-03**	**0.14**	**7.8E-03**	**1.6E-03**	**7.8E-04**	**3.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

Table 4-21. Occupational Risk Estimation - Metalworking Fluids - Inhalation Modeling Data

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Modeling)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.3E-02**	**0.43**	**2.1**	-	**2.8E-03**	**1.4E-02**	**2.8E-02**	**5.6E-02**
		Central Tendency	**0.16**	**1.6**	**7.9**	**0.16**	**8.5E-03**	**4.2E-02**	**8.5E-02**	**0.17**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**34.6**	346.2	1,730.8	-	**2.2**	**11.1**	**22.2**	**44.5**
		Central Tendency	128.6	1,285.7	6,428.6	128.6	**6.7**	**33.4**	**66.7**	133.4
Developmental - Mortality (Narotsky et al., 1995)	10	High End	265.4	2,653.8	13,269.2	-	15.2	75.9	151.9	303.8
		Central Tendency	985.7	9,857.1	49,285.7	985.7	45.6	227.8	455.6	911.3
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**20.1**	200.8	1,003.8	-	**1.5**	**7.4**	**14.9**	**29.7**
		Central Tendency	74.6	745.7	3,728.6	74.6	**4.5**	**22.3**	44.6	89.2
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	151.7	1,516.7	7,583.3	-	**6.3**	31.3	62.6	125.1
		Central Tendency	568.8	5,687.5	28,437.5	568.8	18.8	93.8	187.7	375.4
Kidney (Maltoni et al., 1986)	10	High End	**0.42**	**4.2**	20.8	-	**1.2E-02**	**5.9E-02**	**0.12**	**0.24**
		Central Tendency	**1.6**	15.6	78.1	**1.6**	**3.6E-02**	**0.18**	**0.36**	**0.71**
Neurotoxicity (Arito et al., 1994)	300	High End	**80.0**	800.0	4,000.0	-	**5.1**	**25.7**	**51.5**	**103.0**
		Central Tendency	300.0	3,000.0	15,000.0	300.0	**15.4**	**77.2**	**154.4**	308.9
Immunotoxicity (Keil et al., 2009)	30	High End	**0.55**	**5.5**	**27.5**	-	**3.8E-02**	**0.19**	**0.38**	**0.76**
		Central Tendency	**2.1**	**20.6**	103.1	**2.1**	**0.11**	**0.57**	**1.1**	**2.3**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**8.3**	83.3	416.7	-	**0.58**	**2.9**	**5.8**	**11.6**
		Central Tendency	31.3	312.5	1,562.5	31.3	**1.7**	**8.7**	**17.3**	34.7
Developmental Toxicity (Johnson et al., 2003)	10	High End	**6.2E-02**	**0.62**	**3.1**	-	**4.1E-03**	**2.1E-02**	**4.1E-02**	**8.2E-02**
		Central Tendency	**0.23**	**2.3**	11.6	**0.23**	**1.2E-02**	**6.2E-02**	**0.12**	**0.25**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**6.6E-04**	6.6E-05	1.3E-05	-	**3.0E-02**	**6.0E-03**	**3.0E-03**	**1.5E-03**
		Central Tendency	**1.3E-04**	1.3E-05	2.6E-06	**1.3E-04**	**7.8E-03**	**1.6E-03**	**7.8E-04**	**3.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE calculations for *Metalworking Fluids* utilized both monitoring and modeling inhalation exposure data (with dermal modeling). Results are presented in Table 4-20 and Table 4-21.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints based on monitoring and for congenital heart defects based on modeling at both high-end and central tendency exposure levels via inhalation. Based on both monitoring and modeling data, EPA is unable to estimate ONU exposures separately from workers. Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for multiple endpoints via dermal exposure. MOEs remained below the benchmark MOE for multiple endpoints based on monitoring and for congenital heart defects based on modeling at both exposure levels via inhalation and for congenital heart defects at both dermal exposure levels even when assuming the highest plausible APF and glove PF protection based on monitoring data.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints based on monitoring and for multiple endpoints based on modeling at both high-end and central tendency exposure levels via inhalation. Based on both monitoring and modeling data, EPA is unable to estimate ONU exposures separately from workers. Based on both monitoring and modeling data, MOEs for workers were below the benchmark MOE for all endpoints via dermal exposure. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection based on monitoring data. For modeling data, MOEs were not below the benchmark MOE at central tendency exposure level when assuming APF = 50, although MOEs were below the benchmark MOE for multiple endpoints via the dermal route even when assuming the highest plausible glove PF protection.

Cancer Risk Estimates:

Based on both monitoring and modeling data, extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Based on both monitoring and modeling data, EPA is unable to estimate ONU exposures separately from workers. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection based on monitoring data. For modeling data, risk estimates were not above the benchmark at either inhalation exposure level when assuming APF = 10, although risk estimates were above the benchmark via the dermal route even when assuming the highest plausible glove PF protection.

861 **Table 4-22. Occupational Risk Estimation - Adhesives, Sealants, Paints, and Coatings (Industrial Setting)**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**2.8E-04**	**2.8E-03**	**1.4E-02**	**1.1E-02**	**2.5E-03**	**1.3E-02**	**2.5E-02**	**5.0E-02**
		Central Tendency	**2.4E-03**	**2.4E-02**	**0.12**	**1.2E-02**	**7.5E-03**	**3.8E-02**	**7.5E-02**	**0.15**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.23**	**2.3**	**11.4**	**9.0**	**2.0**	**9.9**	**19.8**	**39.5**
		Central Tendency	**1.9**	**19.4**	**97.1**	**9.6**	**5.9**	**29.7**	**59.3**	118.6
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**1.7**	17.5	87.4	69.0	13.5	67.5	135.0	270.0
		Central Tendency	14.9	148.8	744.1	73.3	40.5	202.5	405.0	810.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.13**	**1.3**	**6.6**	**5.2**	**1.3**	**6.6**	**13.2**	**26.4**
		Central Tendency	**1.1**	**11.3**	56.3	**5.5**	**4.0**	**19.8**	39.6	79.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**1.0**	10.1	50.5	39.9	**5.6**	27.8	55.6	111.2
		Central Tendency	**8.6**	86.0	429.9	42.4	16.7	83.4	166.8	333.7
Kidney (Maltoni et al., 1986)	10	High End	**2.8E-03**	**2.8E-02**	**0.14**	**0.11**	**1.1E-02**	**5.3E-02**	**0.11**	**0.21**
		Central Tendency	**2.4E-02**	**0.24**	**1.2**	**0.12**	**3.2E-02**	**0.16**	**0.32**	**0.63**
Neurotoxicity (Arito et al., 1994)	300	High End	**0.53**	**5.3**	**26.6**	**21.0**	**4.6**	**22.9**	**45.8**	**91.5**
		Central Tendency	**4.5**	**45.3**	**226.7**	**22.3**	**13.7**	**68.6**	**137.3**	**274.5**
Immunotoxicity (Keil et al., 2009)	30	High End	**3.7E-03**	**3.7E-02**	**0.18**	**0.14**	**3.4E-02**	**0.17**	**0.34**	**0.68**
		Central Tendency	**3.1E-02**	**0.31**	**1.6**	**0.15**	**0.10**	**0.51**	**1.0**	**2.0**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**5.5E-02**	**0.55**	**2.8**	**2.2**	**0.51**	**2.6**	**5.1**	**10.3**
		Central Tendency	**0.47**	**4.7**	**23.6**	**2.3**	**1.5**	**7.7**	**15.4**	30.8
Developmental Toxicity (Johnson et al., 2003)	10	High End	**4.1E-04**	**4.1E-03**	**2.1E-02**	**1.6E-02**	**3.7E-03**	**1.8E-02**	**3.7E-02**	**7.3E-02**
		Central Tendency	**3.5E-03**	**3.5E-02**	**0.17**	**1.7E-02**	**1.1E-02**	**5.5E-02**	**0.11**	**0.22**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.10**	**1.0E-02**	**2.0E-03**	**2.6E-03**	**3.4E-02**	**6.8E-03**	**3.4E-03**	**1.7E-03**
		Central Tendency	**9.3E-03**	**9.3E-04**	**1.9E-04**	**1.9E-03**	**8.7E-03**	**1.7E-03**	**8.7E-04**	**4.4E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

MOE results for *Adhesives, Sealants, Paints, and Coatings (Industrial Setting)* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-22. Inhalation exposures are estimated to be identical for industrial and commercial workers.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both inhalation exposure levels even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

888 **Table 4-23. Occupational Risk Estimation - Adhesives, Sealants, Paints, and Coatings (Commercial Setting)**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**2.8E-04**	**2.8E-03**	**1.4E-02**	**1.1E-02**	**1.6E-03**	**8.0E-03**	**1.6E-02**	N/A[1]
		Central Tendency	**2.4E-03**	**2.4E-02**	**0.12**	**1.2E-02**	**4.8E-03**	**2.4E-02**	**4.8E-02**	
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.23**	**2.3**	**11.4**	**9.0**	**1.3**	**6.3**	**12.6**	
		Central Tendency	**1.9**	**19.4**	**97.1**	**9.6**	**3.8**	**18.9**	**37.8**	
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**1.7**	17.5	87.4	69.0	**8.6**	43.0	86.0	
		Central Tendency	14.9	148.8	744.1	73.3	25.8	129.0	258.0	
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.13**	**1.3**	**6.6**	**5.2**	**0.84**	**4.2**	**8.4**	
		Central Tendency	**1.1**	**11.3**	56.3	**5.5**	**2.5**	**12.6**	**25.2**	
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**1.0**	10.1	50.5	39.9	**3.5**	17.7	35.4	N/A[1]
		Central Tendency	**8.6**	86.0	429.9	42.4	10.6	53.1	106.3	
Kidney (Maltoni et al., 1986)	10	High End	**2.8E-03**	**2.8E-02**	**0.14**	**0.11**	**6.7E-03**	**3.4E-02**	**6.7E-02**	
		Central Tendency	**2.4E-02**	**0.24**	**1.2**	**0.12**	**2.0E-02**	**0.10**	**0.20**	
Neurotoxicity (Arito et al., 1994)	300	High End	**0.53**	**5.3**	**26.6**	**21.0**	**2.9**	**14.6**	**29.1**	
		Central Tendency	**4.5**	**45.3**	**226.7**	**22.3**	**8.7**	**43.7**	**87.4**	
Immunotoxicity (Keil et al., 2009)	30	High End	**3.7E-03**	**3.7E-02**	**0.18**	**0.14**	**2.2E-02**	**0.11**	**0.22**	
		Central Tendency	**3.1E-02**	**0.31**	**1.6**	**0.15**	**6.5E-02**	**0.32**	**0.65**	
Reproductive Toxicity (Chia et al., 1996)	30	High End	**5.5E-02**	**0.55**	**2.8**	**2.2**	**0.33**	**1.6**	**3.3**	
		Central Tendency	**0.47**	**4.7**	**23.6**	**2.3**	**0.98**	**4.9**	**9.8**	
Developmental Toxicity (Johnson et al., 2003)	10	High End	**4.1E-04**	**4.1E-03**	**2.1E-02**	**1.6E-02**	**2.3E-03**	**1.2E-02**	**2.3E-02**	
		Central Tendency	**3.5E-03**	**3.5E-02**	**0.17**	**1.7E-02**	**7.0E-03**	**3.5E-02**	**7.0E-02**	
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**0.10**	**1.0E-02**	**2.0E-03**	**2.6E-03**	**5.3E-02**	**1.1E-02**	**5.3E-03**	N/A[1]
		Central Tendency	**9.3E-03**	**9.3E-04**	**1.9E-04**	**1.9E-03**	**1.4E-02**	**2.7E-03**	**1.4E-03**	

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario under a rigorous PPE program.
[1] Glove PF =20 is only applicable to industrial settings (See Section 2.3.1).

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MOE results for *Adhesives, Sealants, Paints, and Coatings (Commercial Setting)* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-23. Inhalation exposures are estimated to be identical for industrial and commercial settings.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Table 4-24. Occupational Risk Estimation - Industrial Processing Aid (12 hr)

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**5.8E-04**	**5.8E-03**	**2.9E-02**	**2.5E-03**	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**1.7E-03**	**1.7E-02**	**8.7E-02**	**5.6E-03**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**0.47**	**4.7**	**23.4**	**2.1**	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**1.4**	**14.1**	**70.6**	**4.6**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	**3.6**	35.9	179.6	15.8	12.2	60.8	121.5	243.0
		Central Tendency	10.8	108.2	540.9	35.1	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**0.27**	**2.7**	**13.6**	**1.2**	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**0.82**	**8.2**	40.9	**2.7**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	**2.1**	20.7	103.7	**9.2**	**5.0**	25.0	50.1	100.1
		Central Tendency	**6.2**	62.5	312.5	20.3	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**5.7E-03**	**5.7E-02**	**0.28**	**2.5E-02**	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**1.7E-02**	**0.17**	**0.86**	**5.6E-02**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**1.1**	**10.9**	**54.7**	**4.8**	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**3.3**	**33.0**	**164.8**	**10.7**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**7.5E-03**	**7.5E-02**	**0.38**	**3.3E-02**	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**2.3E-02**	**0.23**	**1.1**	**7.3E-02**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.11**	**1.1**	**5.7**	**0.50**	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**0.34**	**3.4**	**17.2**	**1.1**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**8.4E-04**	**8.4E-03**	**4.2E-02**	**3.7E-03**	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**2.5E-03**	**2.5E-02**	**0.13**	**8.2E-03**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**4.9E-02**	**4.9E-03**	**9.9E-04**	**1.1E-02**	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**1.3E-02**	**1.3E-03**	**2.5E-04**	**3.9E-03**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.

MOE results for *Industrial Processing Aid* utilized 12hr monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-24.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for most endpoints at both high-end and central tendency exposure levels via inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both inhalation exposure levels even when assuming the highest plausible APF. MOEs remained below the benchmark MOE for congenital heart defects at both dermal exposure levels even when assuming the highest plausible glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for all endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. MOEs for ONUs were also below the benchmark MOE for multiple endpoints at both high-end and central tendency inhalation exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. Risk estimates for ONUs were also above the benchmark for cancer at both high-end and central tendency inhalation exposure levels. Risk estimates remained above the benchmark for cancer at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

946 **Table 4-25. Occupational Risk Estimation - Commercial Printing and Copying**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**5.3E-03**	**5.3E-02**	**0.26**	-	**4.1E-03**	**2.1E-02**	**4.1E-02**	NA[2]
		Central Tendency	**0.13**	**1.3**	**6.5**	**0.13**	**1.2E-02**	**6.2E-02**	**0.12**	
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**4.3**	**42.9**	214.7	-	**3.2**	**16.2**	**32.4**	
		Central Tendency	105.9	1,058.8	5,294.1	105.9	**9.7**	**48.6**	**97.1**	
Developmental - Mortality (Narotsky et al., 1995)	10	High End	32.9	329.3	1,646.4	-	22.1	110.6	221.1	
		Central Tendency	811.8	8,117.6	40,588.2	811.8	66.3	331.7	663.4	
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	30	High End	**2.5**	**24.9**	124.6	-	**2.2**	**10.8**	**21.6**	
		Central Tendency	61.4	614.1	3,070.6	61.4	**6.5**	32.5	64.9	
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	19.0	190.2	951.0	-	**9.1**	45.5	91.1	NA[2]
		Central Tendency	468.9	4,689.2	23,445.9	468.9	27.3	136.6	273.3	
Kidney (Maltoni et al., 1986)	10	High End	**5.2E-02**	**0.52**	**2.6**	-	**1.7E-02**	**8.6E-02**	**0.17**	
		Central Tendency	**1.3**	12.9	64.4	**1.3**	**5.2E-02**	**0.26**	**0.52**	
Neurotoxicity (Arito et al., 1994)	300	High End	**10.0**	**100.3**	501.6	-	**7.5**	**37.5**	**74.9**	
		Central Tendency	**247.3**	2,473.4	12,367.1	**247.3**	**22.5**	**112.4**	**224.8**	
Immunotoxicity (Keil et al., 2009)	30	High End	**6.9E-02**	**0.69**	**3.4**	-	**5.5E-02**	**0.28**	**0.55**	
		Central Tendency	**1.7**	**17.0**	85.0	**1.7**	**0.17**	**0.83**	**1.7**	
Reproductive Toxicity (Chia et al., 1996)	30	High End	**1.0**	**10.5**	52.3	-	**0.84**	**4.2**	**8.4**	
		Central Tendency	**25.8**	257.6	1,288.2	**25.8**	**2.5**	**12.6**	**25.2**	
Developmental Toxicity (Johnson et al., 2003)	10	High End	**7.7E-03**	**7.7E-02**	**0.39**	-	**6.0E-03**	**3.0E-02**	**6.0E-02**	
		Central Tendency	**0.19**	**1.9**	**9.5**	**0.19**	**1.8E-02**	**9.0E-02**	**0.18**	
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**5.4E-03**	**5.4E-04**	**1.1E-04**	-	**2.1E-02**	**4.1E-03**	**2.1E-03**	NA[2]
		Central Tendency	**1.7E-04**	1.7E-05	3.4E-06	**1.7E-04**	**5.3E-03**	**1.1E-03**	**5.3E-04**	

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario under a rigorous PPE program.
[1] EPA is unable to estimate ONU exposures separately from workers.
[2] Glove PF =20 is only applicable to industrial settings (See Section 2.3.1).

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MOE results for *Commercial Printing and Copying* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-25.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE congenital heart defects at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects via inhalation and for multiple endpoints via dermal exposure at both exposure levels even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via inhalation and for all endpoints via the dermal route. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects via inhalation and for multiple endpoints via dermal exposure at both exposure levels even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. Risk estimates remained above the benchmark at high-end inhalation exposure but were not above the benchmark at central tendency inhalation exposure when assuming APF = 10. Risk estimates remained above the benchmark at both dermal exposure levels even when assuming the highest plausible glove PF protection.

PPE Considerations

EPA is presenting risk estimates for respiratory protection up to APF = 50 as a what-if scenario, however EPA believes that small commercial facilities performing commercial printing and copying are unlikely to have a respiratory protection program. Therefore, the use of respirators is unlikely for workers in these facilities.

977 **Table 4-26. Occupational Risk Estimation - Other Industrial Uses**

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**4.3E-03**	**4.3E-02**	**0.21**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	**3.0E-02**	**0.30**	**1.5**	**3.0E-02**	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**3.5**	**34.8**	173.9	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	**24.0**	239.9	1,199.4	**24.0**	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	26.7	266.6	1,333.0	-	12.2	60.8	121.5	243.0
		Central Tendency	183.9	1,839.1	9,195.6	183.9	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	100	High End	**2.0**	**20.2**	100.8	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	**13.9**	139.1	695.7	**13.9**	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	15.4	154.0	770.0	-	**5.0**	25.0	50.1	100.1
		Central Tendency	106.2	1,062.4	5,311.8	106.2	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**4.2E-02**	**0.42**	**2.1**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	**0.29**	**2.9**	14.6	**0.29**	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**8.1**	**81.2**	406.2	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	**56.0**	560.4	2,801.8	**56.0**	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**5.6E-02**	**0.56**	**2.8**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	**0.39**	**3.9**	**19.3**	**0.39**	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**0.85**	**8.5**	42.3	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	**5.8**	58.4	291.9	**5.8**	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**6.3E-03**	**6.3E-02**	**0.31**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	**4.3E-02**	**0.43**	**2.2**	**4.3E-02**	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**6.7E-03**	**6.7E-04**	**1.3E-04**	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	**7.5E-04**	7.5E-05	1.5E-05	**7.5E-04**	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE results for *Other Industrial Uses* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-26.

Acute Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for congenital heart defects at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:

MOEs for workers were below the benchmark MOE for multiple endpoints at both high-end and central tendency exposure levels via inhalation and for all endpoints via the dermal route. EPA is unable to estimate ONU exposures separately from workers. MOEs remained below the benchmark MOE for multiple endpoints at both exposure levels via dermal and inhalation routes even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:

Extra risk estimates for workers were above the benchmark level for cancer at both high-end and central tendency exposure levels via both inhalation and dermal routes. EPA is unable to estimate ONU exposures separately from workers. Risk estimates remained above the benchmark at high-end inhalation exposure but were not above the benchmark at central tendency inhalation exposure when assuming APF = 10. Risk estimates remained above the benchmark at both dermal exposure levels even when assuming the highest plausible glove PF.

Table 4-27. Occupational Risk Estimation - Process Solvent Recycling and Worker Handling of Wastes

Endpoint	Benchmark MOE	Exposure Level	Inhalation (Monitoring)				Dermal (Modeling)			
			No PPE Worker MOE	APF = 10 Worker MOE	APF = 50 Worker MOE	No PPE ONU MOE [1]	No PPE Worker MOE	Glove PF=5 Worker MOE	Glove PF=10 Worker MOE	Glove PF=20 Worker MOE
ACUTE NON-CANCER										
Developmental - Congenital Heart Defects (Johnson et al., 2003)	10	High End	**9.7E-03**	**9.7E-02**	**0.49**	-	**2.3E-03**	**1.1E-02**	**2.3E-02**	**4.5E-02**
		Central Tendency	22.4	224.3	1,121.3	22.4	**6.8E-03**	**3.4E-02**	**6.8E-02**	**0.14**
Developmental - Neurotoxicity (Fredriksson et al., 1993)	100	High End	**7.9**	**78.9**	394.7	-	**1.8**	**8.9**	**17.8**	**35.6**
		Central Tendency	18,182.7	181,827.5	909,137.3	18,182.7	**5.3**	**26.7**	**53.4**	106.7
Developmental - Mortality (Narotsky et al., 1995)	10	High End	60.5	605.3	3,026.3	-	12.2	60.8	121.5	243.0
		Central Tendency	139,401.1	1,394,010.5	6,970,052.6	139,401.1	36.5	182.3	364.5	729.0
Immunotoxicity - Immunosuppression (Selgrade and Gilmour, 2010)	100	High End	**4.6**	45.8	228.9	-	**1.2**	**5.9**	**11.9**	**23.8**
		Central Tendency	10,546.0	105,459.9	527,299.6	10,546.0	**3.6**	**17.8**	35.7	71.3
CHRONIC NON-CANCER										
Liver (Kjellstrand et al., 1983)	10	High End	35.0	349.6	1,748.2	-	**5.0**	25.0	50.1	100.1
		Central Tendency	80,525.3	805,253.2	4,026,266.0	80,525.3	15.0	75.1	150.2	300.3
Kidney (Maltoni et al., 1986)	10	High End	**9.6E-02**	**0.96**	**4.8**	-	**9.5E-03**	**4.8E-02**	**9.5E-02**	**0.19**
		Central Tendency	221.2	2,212.2	11,061.2	221.2	**2.9E-02**	**0.14**	**0.29**	**0.57**
Neurotoxicity (Arito et al., 1994)	300	High End	**18.4**	**184.4**	922.1	-	**4.1**	**20.6**	**41.2**	**82.4**
		Central Tendency	42,474.9	424,748.9	2,123,744.7	42,474.9	**12.4**	**61.8**	**123.5**	**247.1**
Immunotoxicity (Keil et al., 2009)	30	High End	**0.13**	**1.3**	**6.3**	-	**3.0E-02**	**0.15**	**0.30**	**0.61**
		Central Tendency	292.0	2,920.1	14,600.7	292.0	**9.1E-02**	**0.46**	**0.91**	**1.8**
Reproductive Toxicity (Chia et al., 1996)	30	High End	**1.9**	**19.2**	96.1	-	**0.46**	**2.3**	**4.6**	**9.2**
		Central Tendency	4,424.5	44,244.7	221,223.4	4,424.5	**1.4**	**6.9**	**13.9**	**27.7**
Developmental Toxicity (Johnson et al., 2003)	10	High End	**1.4E-02**	**0.14**	**0.71**	-	**3.3E-03**	**1.6E-02**	**3.3E-02**	**6.6E-02**
		Central Tendency	32.7	327.4	1,637.1	32.7	**9.9E-03**	**4.9E-02**	**9.9E-02**	**0.20**
LIFETIME CANCER RISK										
Combined Cancer Risk - Kidney, NHL, Liver	1×10^{-4}	High End	**2.9E-03**	**2.9E-04**	5.9E-05	-	**3.8E-02**	**7.5E-03**	**3.8E-03**	**1.9E-03**
		Central Tendency	9.9E-07	9.9E-08	2.0E-08	9.9E-07	**9.7E-03**	**1.9E-03**	**9.7E-04**	**4.9E-04**

Bold text/pink shading indicates MOE < benchmark MOE. The highest PPE scenarios displayed are plausible for this exposure scenario.
[1] EPA is unable to estimate ONU exposures separately from workers.

MOE results for *Process Solvent Recycling and Worker Handling of Wastes* utilized monitoring inhalation exposure data (with dermal modeling) and are presented in Table 4-27.

Acute Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for congenital heart defects at high-end inhalation exposure and at both dermal exposure levels even when assuming the highest plausible APF and glove PF protection.

Chronic Non-Cancer Risk Estimates:
MOEs for workers were below the benchmark MOE for multiple endpoints at high-end inhalation exposures, but MOEs were above the benchmark MOE for all endpoints at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. MOEs were below the benchmark MOE for multiple endpoints at both dermal exposure levels. MOEs remained below the benchmark MOE for multiple endpoints at high-end inhalation exposure and at both dermal exposure levels even when assuming the highest plausible APF and glove PF protection.

Cancer Risk Estimates:
Extra risk estimates for workers were above the benchmark level for cancer at at high-end inhalation exposures, but risk estimates were above the benchmark MOE for cancer at central tendency inhalation exposures. EPA is unable to estimate ONU exposures separately from workers. Risk estimates were not above the benchmark at central tendency inhalation exposure when assuming APF = 50. Risk estimates remained above the benchmark at both dermal exposure levels even when assuming the highest plausible glove PF protection.

4.2.3 Risk Estimation for Consumer Exposures by Exposure Scenario

Risk estimates via inhalation and dermal routes are provided below for consumers and bystanders following acute exposure. Risk estimates were presented for differing 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. See Section 2.3.2.6.1 for more details on the characterization of consumer exposure and [*CEM Modeling Results and Risk Estimates. Docket # EPA-HQ-OPPT-2019-0500]* for MOE estimates of all modeled scenarios.

As discussed in Section 2.3.2.2, in general, the frequency of product use was considered to be too low to create chronic risk concerns. Although high-end frequencies of consumer use are up to 50 times per year, available toxicological data is based on either single or continuous TCE exposure and it is unknown whether these use patterns are expected to be clustered or intermittent (e.g. one time per week). There is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures. Therefore, EPA cannot fully rule out that consumers at the high-end frequency of use could possibly be at risk for chronic hazard effects, however it is expected to be unlikely. Therefore, based on reasonably available information, EPA did not develop risk estimates for this population.

Table 4-28. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Brake and Parts Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**6.4E-05**	**5.2E-02**	**0.40**	**3.5E-02**
	Bystander	**2.2E-04**	**1.8E-01**	**1.4**	**0.14**
Moderate-Intensity User	User	**4.1E-04**	**0.33**	**2.5**	**0.21**
	Bystander	**1.6E-03**	**1.3**	10	**0.94**
Low-Intensity User	User	**5.2E-03**	**4.2**	32	**2.7**
	Bystander	**2.0E-02**	**17**	127	**12**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**6.8E-05**	**5.4E-02**	**0.37**	**3.6E-02**
	Children (16-20 years)	**7.3E-05**	**5.7E-02**	**0.39**	**3.8E-02**
	Children (11-15 years)	**6.7E-05**	**5.3E-02**	**0.36**	**3.5E-02**
Moderate-Intensity User	Adult (≥21 years)	**9.1E-04**	**0.72**	**4.9**	**0.48**
	Children (16-20 years)	**9.7E-04**	**0.77**	**5.2**	**0.51**
	Children (11-15 years)	**8.9E-04**	**0.70**	**4.8**	**0.47**
Low-Intensity User	Adult (≥21 years)	**4.1E-02**	**32**	220	**22**
	Children (16-20 years)	**4.4E-02**	**34**	235	**23**
	Children (11-15 years)	**4.0E-02**	**32**	215	**21**

MOE results for *Brake and Parts Cleaner* are presented in Table 4-28.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-29. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Aerosol Electronic Degreaser/Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**9.8E-05**	**8.0E-02**	**0.61**	**5.0E-02**
	Bystander	**4.9E-04**	**0.40**	**3.0**	**0.28**
Moderate-Intensity User	User	**2.3E-03**	**1.9**	15	**1.2**
	Bystander	**1.3E-02**	**10**	78	**7.1**
Low-Intensity User	User	**6.7E-02**	**54**	414	**33**
	Bystander	**0.34**	277	2123	193
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Aerosol Electronic Degreaser/Cleaner* are presented in Table 4-29.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity user inhalation exposure levels and for multiple endpoints at high and medium-intensity exposure levels.

Table 4-30. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Liquid Electronic Degreaser/Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.0E-04**	**8.3E-02**	**0.64**	**5.2E-02**
	Bystander	**5.1E-04**	**0.41**	**3.2**	**0.29**
Moderate-Intensity User	User	**1.6E-03**	**1.3**	**9.9**	**0.79**
	Bystander	**8.5E-03**	**6.9**	53	**4.8**
Low-Intensity User	User	**2.1E-02**	**17**	132	**11**
	Bystander	**0.11**	**88**	674	61
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**1.2E-04**	**9.5E-03**	**0.65**	**6.4E-02**
	Children (16-20 years)	**1.3E-04**	**0.10**	**0.70**	**6.8E-02**
	Children (11-15 years)	**1.2E-04**	**9.3E-02**	**0.64**	**6.2E-02**
Moderate-Intensity User	Adult (≥21 years)	**1.8E-03**	**1.4**	**9.7**	**9.5E-01**
	Children (16-20 years)	**1.9E-03**	**1.5**	10	**1.0**
	Children (11-15 years)	**1.8E-03**	**1.4**	**9.6**	**9.4E-01**
Low-Intensity User	Adult (≥21 years)	**7.3E-03**	**5.7**	39	**3.8**
	Children (16-20 years)	**7.8E-03**	**6.1**	42	**4.1**
	Children (11-15 years)	**7.1E-03**	**5.6**	38	**3.7**

MOE results for *Liquid Electronic Degreaser/Cleaner* are presented in Table 4-30.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-31. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Aerosol Spray Degreaser/Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**2.3E-05**	**1.8E-02**	**0.14**	**1.2E-02**
	Bystander	**7.9E-05**	**6.4E-02**	**0.49**	**4.9E-02**
Moderate-Intensity User	User	**9.0E-05**	**7.3E-02**	**0.56**	**4.6E-02**
	Bystander	**3.6E-04**	**0.29**	**2.2**	**0.21**
Low-Intensity User	User	**6.0E-04**	**0.48**	**3.7**	**0.31**
	Bystander	**2.5E-03**	**2.0**	15	**1.4**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**7.3E-05**	**5.7E-02**	**0.39**	**3.8E-02**
	Children (16-20 years)	**7.8E-05**	**6.1E-02**	**0.42**	**4.1E-02**
	Children (11-15 years)	**7.1E-05**	**5.6E-02**	**0.38**	**3.7E-02**
Moderate-Intensity User	Adult (≥21 years)	**5.8E-04**	**0.46**	**3.1**	**0.31**
	Children (16-20 years)	**6.2E-04**	**0.49**	**3.3**	**0.33**
	Children (11-15 years)	**5.7E-04**	**0.45**	**3.1**	**0.30**
Low-Intensity User	Adult (≥21 years)	**2.9E-03**	**2.3**	16	**1.5**
	Children (16-20 years)	**3.1E-03**	**2.4**	17	**1.6**
	Children (11-15 years)	**2.8E-03**	**2.2**	15	**1.5**

MOE results for *Aerosol Spray Degreaser/Cleaner* are presented in Table 4-31.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-32. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Liquid Degreaser/Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**2.5E-05**	**2.0E-02**	**0.16**	**1.3E-02**
	Bystander	**1.0E-04**	**8.3E-02**	**0.64**	**6.1E-02**
Moderate-Intensity User	User	**2.4E-04**	**0.19**	**1.5**	**0.12**
	Bystander	**1.2E-03**	**1.0**	**7.8**	**0.70**
Low-Intensity User	User	**1.4E-03**	**1.2**	**8.8**	**0.71**
	Bystander	**7.6E-03**	**6.2**	47	**4.3**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**3.0E-05**	**2.4E-02**	**0.16**	**1.6E-02**
	Children (16-20 years)	**3.2E-05**	**2.6E-02**	**0.17**	**1.7E-02**
	Children (11-15 years)	**3.0E-05**	**2.3E-02**	**0.16**	**1.6E-02**
Moderate-Intensity User	Adult (≥21 years)	**2.4E-04**	**0.19**	**1.3**	**0.13**
	Children (16-20 years)	**2.6E-04**	**0.20**	**1.4**	**0.14**
	Children (11-15 years)	**2.4E-04**	**0.19**	**1.3**	**0.13**
Low-Intensity User	Adult (≥21 years)	**1.8E-03**	**1.4**	**9.8**	**0.96**
	Children (16-20 years)	**1.9E-03**	**1.5**	10	**1.0**
	Children (11-15 years)	**1.8E-03**	**1.4**	**9.6**	**0.94**

MOE results for *Liquid Degreaser/Cleaner* are presented in Table 4-32.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-33. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Aerosol Gun Scrubber

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**5.0E-02**	**40**	309	**26**
	Bystander	**0.20**	164	1255	120
Moderate-Intensity User	User	**4.7E-02**	**38**	294	**24**
	Bystander	**0.25**	202	1551	141
Low-Intensity User	User	**8.1E-02**	**66**	506	41
	Bystander	**0.44**	354	2715	247
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**7.5E-05**	**5.9E-02**	**0.41**	**4.0E-02**
	Children (16-20 years)	**8.1E-05**	**6.4E-02**	**0.43**	**4.2E-02**
	Children (11-15 years)	**7.4E-05**	**5.8E-02**	**0.40**	**3.9E-02**
Moderate-Intensity User	Adult (≥21 years)	**6.0E-04**	**0.48**	**3.2**	**0.32**
	Children (16-20 years)	**6.4E-04**	**0.51**	**3.5**	**0.34**
	Children (11-15 years)	**5.9E-04**	**0.46**	**3.2**	**0.31**
Low-Intensity User	Adult (≥21 years)	**7.5E-03**	**5.9**	41	**4.0**
	Children (16-20 years)	**8.0E-03**	**6.3**	43	**4.2**
	Children (11-15 years)	**7.3E-03**	**5.8**	40	**3.9**

MOE results for *Aerosol Gun Scrubber* are presented in Table 4-33.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity user inhalation exposure levels.

Table 4-34. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Liquid Gun Scrubber

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**5.8E-02**	**47**	361	**30**
	Bystander	**0.24**	191	1465	140
Moderate-Intensity User	User	**5.5E-02**	**45**	343	**28**
	Bystander	**0.29**	236	1809	164
Low-Intensity User	User	**5.9E-02**	**48**	370	30
	Bystander	**0.30**	247	1893	172
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**3.3E-05**	**2.6E-02**	**0.18**	**1.7E-02**
	Children (16-20 years)	**3.5E-05**	**2.7E-02**	**0.19**	**1.8E-02**
	Children (11-15 years)	**3.2E-05**	**2.5E-02**	**0.17**	**1.7E-02**
Moderate-Intensity User	Adult (≥21 years)	**2.6E-04**	**0.21**	**1.4**	**0.14**
	Children (16-20 years)	**2.8E-04**	**0.22**	**1.5**	**0.15**
	Children (11-15 years)	**2.5E-04**	**0.20**	**1.4**	**0.13**
Low-Intensity User	Adult (≥21 years)	**1.9E-03**	**1.5**	10	**1.0**
	Children (16-20 years)	**2.1E-03**	**1.6**	11	**1.1**
	Children (11-15 years)	**1.9E-03**	**1.5**	10	**1.0**

MOE results for *Liquid Gun Scrubber* are presented in Table 4-34.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity user inhalation exposure levels.

Table 4-35. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Mold Release

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**2.3E-04**	**0.18**	**1.4**	**0.11**
	Bystander	**1.1E-03**	**0.91**	**7.0**	**0.64**
Moderate-Intensity User	User	**2.1E-03**	**1.7**	13	**1.1**
	Bystander	**1.1E-02**	**9.2**	71	**6.4**
Low-Intensity User	User	**2.1E-02**	**17**	130	**11**
	Bystander	**0.11**	**87**	667	61
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Mold Release* are presented in Table 4-35.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-36. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Aerosol Tire Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**2.4E-04**	**0.19**	**1.5**	**0.13**
	Bystander	**5.4E-04**	**0.44**	**3.4**	**0.32**
Moderate-Intensity User	User	**8.9E-04**	**0.72**	**5.5**	**0.46**
	Bystander	**3.6E-03**	**2.9**	22	**2.0**
Low-Intensity User	User	**6.4E-03**	**5.2**	40	**3.3**
	Bystander	**2.6E-02**	**21**	164	**15**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**3.3E-04**	**0.26**	**1.8**	**0.17**
	Children (16-20 years)	**3.5E-04**	**0.28**	**1.9**	**0.19**
	Children (11-15 years)	**3.2E-04**	**0.26**	**1.7**	**0.17**
Moderate-Intensity User	Adult (≥21 years)	**1.3E-03**	**1.0**	**7.1**	**0.70**
	Children (16-20 years)	**1.4E-03**	**1.1**	**7.6**	**0.74**
	Children (11-15 years)	**1.3E-03**	**1.0**	**6.9**	**0.68**
Low-Intensity User	Adult (≥21 years)	**5.7E-03**	**4.5**	31	**3.0**
	Children (16-20 years)	**6.0E-03**	**4.8**	33	**3.2**
	Children (11-15 years)	**5.5E-03**	**4.4**	30	**2.9**

MOE results for *Aerosol Tire Cleaner* are presented in Table 4-36.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-37. Consumer Risk Estimation - Solvents for Cleaning and Degreasing - Liquid Tire Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**7.8E-05**	**6.3E-02**	**0.48**	**4.2E-02**
	Bystander	**2.4E-04**	**0.20**	**1.5**	**0.14**
Moderate-Intensity User	User	**4.0E-04**	**0.32**	**2.5**	**0.21**
	Bystander	**1.6E-03**	**1.3**	**9.9**	**0.92**
Low-Intensity User	User	**2.0E-03**	**1.6**	12	**1.0**
	Bystander	**8.3E-03**	**6.7**	51	**4.7**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**5.9E-05**	**4.7E-02**	**0.32**	**3.1E-02**
	Children (16-20 years)	**6.3E-05**	**5.0E-02**	**0.34**	**3.3E-02**
	Children (11-15 years)	**5.8E-05**	**4.6E-02**	**0.31**	**3.0E-02**
Moderate-Intensity User	Adult (≥21 years)	**2.4E-04**	**0.19**	**1.3**	**0.12**
	Children (16-20 years)	**2.5E-04**	**0.20**	**1.4**	**0.13**
	Children (11-15 years)	**2.3E-04**	**0.18**	**1.2**	**0.12**
Low-Intensity User	Adult (≥21 years)	**7.1E-04**	**0.56**	**3.8**	**0.37**
	Children (16-20 years)	**7.6E-04**	**0.60**	**4.1**	**0.40**
	Children (11-15 years)	**6.9E-04**	**0.55**	**3.7**	**0.37**

MOE results for *Liquid Tire Cleaner* are presented in Table 4-37.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-38. Consumer Risk Estimation - Lubricants and Greases - Tap and Die Fluid

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**2.5E-04**	**0.20**	**1.6**	**0.13**
	Bystander	**1.3E-03**	**1.0**	**7.8**	**0.71**
Moderate-Intensity User	User	**2.4E-03**	**1.9**	15	**1.2**
	Bystander	**1.3E-02**	**10**	79	**7.1**
Low-Intensity User	User	**1.3E-02**	**11**	83	**6.8**
	Bystander	**4.3E-02**	**35**	270	**28**
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Tap and Die Fluid* are presented in Table 4-38.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-39. Consumer Risk Estimation - Lubricants and Greases - Penetrating Lubricant

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**3.2E-04**	**0.26**	**2.0**	**0.16**
	Bystander	**1.6E-03**	**1.3**	**9.8**	**0.89**
Moderate-Intensity User	User	**5.4E-03**	**4.4**	33	**2.7**
	Bystander	**2.9E-02**	**23**	179	**16**
Low-Intensity User	User	**0.17**	139	1065	86
	Bystander	**0.88**	712	5460	496
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Penetrating Lubricant* are presented in Table 4-39.

MOEs for consumer users were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity inhalation exposure levels and for multiple endpoints at high and medium-intensity exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints for high and medium-intensity users and for congenital heart defects at all user intensity inhalation exposure levels.

Table 4-40. Consumer Risk Estimation - Adhesives and Sealants - Solvent-Based Adhesive and Sealant

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.1E-04**	**9.3E-02**	**0.71**	**5.6E-02**
	Bystander	**9.1E-04**	**0.74**	**5.7**	**0.52**
Moderate-Intensity User	User	**3.7E-03**	**3.0**	23	**1.8**
	Bystander	**3.6E-02**	**29**	223	**20**
Low-Intensity User	User	**0.42**	340	2604	207
	Bystander	**2.8**	2300	17636	1602
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Solvent-Based Adhesive and Sealant* are presented in Table 4-40.

MOEs for consumer users were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity inhalation exposure levels and for multiple endpoints at high and medium-intensity exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints for high and medium-intensity users and for congenital heart defects at all user intensity inhalation exposure levels.

Table 4-41. Consumer Risk Estimation - Adhesives and Sealants - Mirror Edge Sealant

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.1E-03**	**0.90**	**6.9**	**0.57**
	Bystander	**4.7E-03**	**3.8**	29	**2.7**
Moderate-Intensity User	User	**3.3E-03**	**2.7**	21	**1.7**
	Bystander	**1.8E-02**	**15**	114	**10**
Low-Intensity User	User	**0.17**	134	1028	83
	Bystander	**0.91**	737	5651	513
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Mirror Edge Sealant* are presented in Table 4-41.

MOEs for consumer users were below the benchmark MOE for congenital heart defects at high, medium, and low-intensity inhalation exposure levels and for multiple endpoints at high and medium-intensity exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints for high and medium-intensity users and for congenital heart defects at all user intensity inhalation exposure levels.

Table 4-42. Consumer Risk Estimation - Adhesives and Sealants - Tire Repair Cement / Sealer

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**3.1E-04**	**0.25**	**1.9**	**0.17**
	Bystander	**9.7E-04**	**0.79**	**6.1**	**0.57**
Moderate-Intensity User	User	**5.6E-03**	**4.5**	35	**2.9**
	Bystander	**2.3E-02**	**18**	141	**13**
Low-Intensity User	User	**6.2E-02**	**50**	385	32
	Bystander	**0.23**	188	1444	133
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Tire Repair Cement/Sealer* are presented in Table 4-42.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints for high and medium-intensity users and for congenital heart defects at all user intensity inhalation exposure levels.

Table 4-43. Consumer Risk Estimation - Cleaning and Furniture Care Products - Carpet Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**7.0E-05**	**5.7E-02**	**0.44**	**3.6E-02**
	Bystander	**3.2E-04**	**0.26**	**2.0**	**0.18**
Moderate-Intensity User	User	**5.8E-04**	**0.47**	**3.6**	**0.29**
	Bystander	**2.9E-03**	**2.4**	18	**1.7**
Low-Intensity User	User	**3.4E-03**	**2.7**	21	**1.7**
	Bystander	**1.6E-02**	**13**	99	**9.0**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**1.1E-04**	**8.8E-02**	**0.60**	**5.9E-02**
	Children (16-20 years)	**1.2E-04**	**9.4E-02**	**0.64**	**6.3E-02**
	Children (11-15 years)	**1.1E-04**	**8.6E-02**	**0.59**	**5.7E-02**
Moderate-Intensity User	Adult (≥21 years)	**6.7E-04**	**0.53**	**3.6**	**0.35**
	Children (16-20 years)	**7.1E-04**	**0.56**	**3.8**	**0.38**
	Children (11-15 years)	**6.6E-04**	**0.52**	**3.5**	**0.35**
Low-Intensity User	Adult (≥21 years)	**1.3E-02**	**11**	72	**7.1**
	Children (16-20 years)	**1.4E-02**	**11**	77	**7.5**
	Children (11-15 years)	**1.3E-02**	**10**	70	**6.9**

MOE results for *Carpet Cleaner* are presented in Table 4-43.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-44. Consumer Risk Estimation - Cleaning and Furniture Care Products - Aerosol Spot Remover

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.1E-04**	**9.3E-02**	**0.71**	**5.6E-02**
	Bystander	**1.1E-03**	**0.87**	**6.7**	**0.61**
Moderate-Intensity User	User	**9.8E-04**	**0.79**	**6.1**	**0.47**
	Bystander	**9.9E-03**	**8.0**	61	**5.6**
Low-Intensity User	User	**6.5E-03**	**5.3**	41	**3.2**
	Bystander	**5.4E-02**	**43**	333	30
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**9.4E-04**	**0.74**	**5.1**	**0.50**
	Children (16-20 years)	**1.0E-03**	**0.79**	**5.4**	**0.53**
	Children (11-15 years)	**9.2E-04**	**0.73**	**5.0**	**0.49**
Moderate-Intensity User	Adult (≥21 years)	**5.7E-03**	**4.5**	**31**	**3.0**
	Children (16-20 years)	**6.0E-03**	**4.8**	**33**	**3.2**
	Children (11-15 years)	**5.5E-03**	**4.4**	**30**	**2.9**
Low-Intensity User	Adult (≥21 years)	**5.7E-02**	**45**	305	**30**
	Children (16-20 years)	**6.0E-02**	**48**	325	32
	Children (11-15 years)	**5.5E-02**	**44**	297	**29**

MOE results for *Aerosol Spot Remover* are presented in Table 4-44.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-45. Consumer Risk Estimation - Cleaning and Furniture Care Products - Liquid Spot Remover

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**9.3E-05**	**7.5E-02**	**0.58**	**4.7E-02**
	Bystander	**4.6E-04**	**0.37**	**2.9**	**0.26**
Moderate-Intensity User	User	**7.8E-04**	**0.63**	**4.9**	**0.39**
	Bystander	**4.2E-03**	**3.4**	26	**2.4**
Low-Intensity User	User	**6.8E-03**	**5.5**	42	**3.4**
	Bystander	**3.4E-02**	**28**	214	**19**
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**1.6E-04**	**0.13**	**0.87**	**8.5E-02**
	Children (16-20 years)	**1.7E-04**	**0.14**	**0.93**	**9.1E-02**
	Children (11-15 years)	**1.6E-04**	**0.13**	**0.85**	**8.4E-02**
Moderate-Intensity User	Adult (≥21 years)	**9.8E-04**	**0.77**	**5.3**	**0.51**
	Children (16-20 years)	**1.0E-03**	**0.82**	**5.6**	**0.55**
	Children (11-15 years)	**9.5E-04**	**0.75**	**5.1**	**0.50**
Low-Intensity User	Adult (≥21 years)	**1.5E-02**	**12**	79	**7.7**
	Children (16-20 years)	**1.6E-02**	**12**	84	**8.2**
	Children (11-15 years)	**1.4E-02**	**11**	77	**7.5**

MOE results for *Liquid Spot Remover* are presented in Table 4-45.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-46. Consumer Risk Estimation - Arts, Crafts, and Hobby Materials - Fixatives and Finishing Spray Coatings

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**4.0E-04**	**0.32**	**2.5**	**0.20**
	Bystander	**1.6E-03**	**1.3**	10	**0.92**
Moderate-Intensity User	User	**2.5E-03**	**2.0**	15	**1.2**
	Bystander	**1.3E-02**	**11**	83	**7.6**
Low-Intensity User	User	**1.3E-02**	**10**	79	**6.4**
	Bystander	**6.5E-02**	**53**	407	37
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Fixatives and Finishing Spray Coatings* are presented in Table 4-46.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-47. Consumer Risk Estimation - Apparel and Footwear Care Products - Shoe Polish

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.1E-03**	**0.89**	**6.8**	**0.55**
	Bystander	**5.5E-03**	**4.4**	34	**3.1**
Moderate-Intensity User	User	**1.1E-02**	**8.8**	67	**5.4**
	Bystander	**5.9E-02**	**48**	366	33
Low-Intensity User	User	**6.2E-02**	**50**	386	31
	Bystander	**3.2E-01**	258	1977	180
Dermal Exposure					
High-Intensity User	Adult (≥21 years)	**1.7E-03**	**1.4**	**9.3**	**0.91**
	Children (16-20 years)	**1.8E-03**	**1.45**	**9.9**	**0.97**
	Children (11-15 years)	**1.7E-03**	**1.3**	**9.1**	**0.89**
Moderate-Intensity User	Adult (≥21 years)	**1.0E-02**	**8.2**	56	**5.5**
	Children (16-20 years)	**1.1E-02**	**8.7**	60	**5.8**
	Children (11-15 years)	**1.0E-02**	**8.0**	54	**5.3**
Low-Intensity User	Adult (≥21 years)	**0.10**	**82**	560	55
	Children (16-20 years)	**0.11**	**87**	596	58
	Children (11-15 years)	**0.10**	**80**	545	53

MOE results for *Shoe Polish* are presented in Table 4-47.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity exposure levels via both inhalation and dermal routes. Dermal MOEs were below the benchmark MOE for multiple endpoints and all age groups. MOEs for bystanders were below the benchmark MOE for multiple endpoints for high and medium-intensity users and for congenital heart defects at all user intensity inhalation exposure levels.

Table 4-48. Consumer Risk Estimation - Other Consumer Uses - Fabric Spray

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**5.8E-05**	**0.12**	**0.91**	**7.2E-02**
	Bystander	**2.4E-04**	**0.94**	**7.2**	**0.66**
Moderate-Intensity User	User	**3.6E-04**	**0.72**	**5.5**	**0.43**
	Bystander	**1.9E-03**	**7.3**	**56**	**5.1**
Low-Intensity User	User	**1.9E-03**	**4.1**	31	**2.5**
	Bystander	**9.5E-03**	**33**	251	**23**
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Fabric Spray* are presented in Table 4-48.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-49. Consumer Risk Estimation - Other Consumer Uses - Film Cleaner

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**5.8E-05**	**4.7E-02**	**0.36**	**3.0E-02**
	Bystander	**2.4E-04**	**0.19**	**1.5**	**0.13**
Moderate-Intensity User	User	**3.6E-04**	**0.29**	**2.2**	**0.18**
	Bystander	**1.9E-03**	**1.6**	12	**1.1**
Low-Intensity User	User	**1.9E-03**	**1.5**	12	**0.93**
	Bystander	**9.5E-03**	**7.7**	59	**5.4**
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Fabric Spray* are presented in Table 4-49.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

Table 4-50. Consumer Risk Estimation - Other Consumer Uses - Hoof Polish

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**1.7E-03**	**1.4**	10	**0.79**
	Bystander	**0.34**	272	2084	157
Moderate-Intensity User	User	**1.7E-02**	**14**	106	**8.0**
	Bystander	**7.8**	6307	48351	3653
Low-Intensity User	User	**0.12**	**97**	747	56
	Bystander	48	38519	295309	22309
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Hoof Polish* are presented in Table 4-50.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for congenital heart defects only for high and medium-intensity users. MOEs for bystanders were not below the benchmark MOE for any endpoint at low-intensity inhalation exposure levels.

Table 4-51. Consumer Risk Estimation - Other Consumer Uses - Pepper Spray

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Immunosuppression (Selgrade and Gilmour, 2010)
Inhalation Exposure					
Single Scenario	User	**0.21**	169	1297	98
	Bystander	Not modeled due to simulated outdoor scenario - can be considered equal to user.			
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Pepper Spray* are presented in Table 4-51.

MOEs for consumer users were below the benchmark MOE for congenital heart defects. Dermal exposure was not quantified. MOEs for bystanders are expected to be equivalent to users.

Table 4-52. Consumer Risk Estimation - Other Consumer Uses - Toner Aid

Scenario	Consumer Receptor	Benchmark			
		10	100	10	30
		Developmental Effects Congenital Heart Defects (Johnson et al., 2003)	**Developmental Effects** Developmental Neurotoxicity (Fredriksson et al., 1993)	**Developmental Effects** Increased Resorptions (Narotsky et al., 1995)	**Acute Immunotoxicity** Response to Infection (Selgrade and Gilmour, 2010)
Inhalation Exposure					
High-Intensity User	User	**4.2E-04**	**0.34**	**2.6**	**0.21**
	Bystander	**1.7E-03**	**1.4**	11	**0.97**
Moderate-Intensity User	User	**2.6E-03**	**2.1**	16	**1.3**
	Bystander	**1.4E-02**	**11**	88	**8.0**
Low-Intensity User	User	**1.4E-02**	**11**	84	**6.8**
	Bystander	**6.9E-02**	**56**	431	39
Dermal exposures were not quantified for this scenario, as dermal exposure with impeded evaporation is not expected.					

MOE results for *Toner Aid* are presented in Table 4-52.

MOEs for consumer users were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity inhalation exposure levels. Dermal exposure was not quantified. MOEs for bystanders were below the benchmark MOE for multiple endpoints at high, medium, and low-intensity user inhalation exposure levels.

4.3 Assumptions and Key Sources of Uncertainty for Risk Characterization

4.3.1 Environmental Risk Characterization

There were some uncertainties related to environmental risk for TCE, with some leading to potentially underestimating risk and some leading to potentially overestimating risk. As mentioned in Section 3.1.7, there were uncertainties regarding the hazard data for aquatic species; however, some of the uncertainty was mitigated by the use of multiple lines of evidence supporting the assessment of hazard.

There were also uncertainties around surface water concentrations used to determine the environmental risk. EPA used E-FAST, monitored data, and data from reasonably available literature to characterize acute and chronic exposures of TCE to aquatic organisms. In some ways the E-FAST estimates are underestimating exposure, because data used in E-FAST include TRI and DMR data. TRI does not include smaller facilities with fewer than 10 full time employees, nor does it cover certain sectors, which may lead to underestimates in total TCE releases to the environment. DMR data are submitted by NPDES permit holders to states or directly to the EPA according to the monitoring requirements of the facility's permit. States are only required to load major discharger data into DMR and may or may not load minor discharger data. The definition of major vs. minor discharger is set by each state and could be based on discharge volume or facility size. Due to these limitations, some sites that discharge may not be included in the DMR dataset.

In other ways the E-FAST estimates are overestimating exposure, because TCE is a volatile chemical, and E-FAST doesn't take volatilization into consideration; and, for static water bodies, E-FAST uses a dilution factor as low as one. This may have led to an over estimation of surface water concentrations for the two facilities with environmental risks, as both release to still water bodies. Additionally, both facilities with risk showed 20 days of exceeding the chronic COC. (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.) However, there is uncertainty about whether those 20 days would be consecutive, because the days of exceedance modeled in E-FAST occur sporadically throughout the year. Because TCE is a volatile chemical, it is more likely that a chronic exposure duration will occur when there are more days of exceedances.

The reasonably available monitored data was limited temporally and geographically. Aquatic environmental conditions such as temperature and composition (i.e., total organic carbon, water hardness, dissolve oxygen, and pH) can fluctuate with the seasons, which could affect TCE concentrations in water and sediment pore water. In addition, TCE monitoring data was collected only in certain areas, and within a limited number of states in the U.S. There were no measurements reasonably available immediately downstream from facilities releasing TCE to surface water; these data are only a limited representation of ambient water.

4.3.2 Human Health Risk Characterization

4.3.2.1 Occupational Exposure Considerations

Air concentrations. In most scenarios where data were reasonably available, EPA did not find enough reasonably available data to determine complete statistical distributions of actual air concentrations for the workers exposed to TCE. Ideally, EPA would like to know 50th and 95th percentiles for each exposed population. In the absence of percentile data for monitoring, the air concentration means and medians (means are preferred over medians) of the data sets served as substitutes for 50th percentiles (central tendencies) of the actual distributions, whereas high ends of ranges served as substitutes for 95th

percentiles of the actual distributions. However, these substitutes are uncertain and are not as reliable as the true percentiles. For instance, in the few cases where enough data were found to determine statistical means and 95th percentiles, the associated substitutes (i.e., medians and high ends of ranges) were shown to overestimate exposures, sometimes significantly. While it most air concentration data represent real exposure levels, EPA cannot determine whether these concentrations are representative of the statistical distributions of actual air concentrations to which workers are exposed. It is unknown whether these uncertainties overestimate or underestimate exposures. The range of air concentration estimates from central tendency to high-end was generally not large (e.g., less than 20-fold for most exposure scenarios). Because of this the results of risk characterization were generally not sensitive to the individual estimates of the central tendency and high-end separately but rather were based on considering both central tendency and high-end exposure estimates which increase the overall confidence in the risk characterization.

Exposures for ONUs can vary substantially. Most data sources do not sufficiently describe the proximity of these employees to the exposure source. As such, exposure levels for the "occupational non-user" category will have high variability depending on the specific work activity performed. It is possible that some employees categorized as "occupational non-user" have exposures similar to those in the "worker" category depending on their specific work activity pattern. Therefore, in the absence of specific monitoring or modeling data, worker risk estimates were applied to ONUs. In many instances, this is likely to overestimate exposures, although the central tendency worker values may be a reasonable approximation of ONU estimates.

Additionally, some data sources may be inherently biased. For example, bias may be present if exposure monitoring was conducted to address concerns regarding adverse human health effects reported following exposures during use. These sources may cause exposures to be overestimated.

Where data were not reasonably available, the modeling approaches used to estimate air concentrations also involve uncertainties. Model parameter values did not all contain distributions known to represent the modeled scenario. It is also uncertain whether the model equations generate results that represent actual workplace air concentrations. It is unknown whether these uncertainties overestimate or underestimate exposures.

Averaging Times. EPA cannot determine how accurately the assumptions of exposure frequencies (days/yr exposed) and exposed working years may represent actual exposure frequencies and exposed working years. For example, tenure is used to represent exposed working years, but many workers may not be exposed during their entire tenure. It is unknown whether these uncertainties overestimate or underestimate exposures, although the high-end values may result in overestimates when used in combination with high-end values of other parameters.

See Section 2.3.1.3 for more details on uncertainties and assumptions underlying the occupational exposure assessment.

4.3.2.2 Consumer/Bystander Exposure Considerations

Inhalation and dermal exposures are evaluated for acute exposure scenarios, i.e., those resulting from short-term or daily exposures. Chronic exposure scenarios resulting from long-term use of household consumer products are not evaluated. As discussed in Section 2.3.2.2, in general, the frequency of product use was considered to be too low to create chronic risk concerns. Although high-end frequencies of consumer use are up to 50 times per year, reasonably available toxicological data is based on either single or continuous TCE exposure and it is unknown whether these use patterns are expected to be

clustered or intermittent (e.g. one time per week). There is uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures. Therefore, EPA cannot fully rule out that consumers at the high-end frequency of use could possibly be at risk for chronic hazard effects, however it is expected to be unlikely.

The output of the consumer exposure model is fully determined by the choices of parameter values and initial conditions. Stochastic approaches feature inherent randomness, such that a given set of parameter values and initial conditions can lead to an ensemble of different model outputs. Because EPA's largely deterministic approach involves choices regarding low, medium, and high values for highly influential factors such as chemical mass and frequency/duration of product use, it likely captures the range of potential exposure levels although it does not necessarily enable characterization of the full probabilistic distribution of all possible outcomes.

Certain inputs to which model outputs are sensitive, such as zone volumes and airflow rates, were not varied across product-use scenarios. As a result, model outcomes for extreme circumstances such as a relatively large chemical mass in a relatively low-volume environment likely are not represented among the model outcomes. Such extreme outcomes are believed to lie near the upper end (e.g., at or above the 90th percentile) of the exposure distribution.

See Section 2.3.2.7 for more details on uncertainties and assumptions underlying the consumer exposure assessment.

4.3.2.3 Dermal Absorption Considerations

The occupational and consumer assessment approaches utilize different models for estmating dermal absorption. As discussed in Section 2.3.2.5.1, the occupational exposure assessment used a fractional absorption model that accounts for evaporation of volatile chemicals such as TCE. In contrast, the consumer assessment used a permeability model that incorporates duration of use and was only applied to exposure scenarios where evaporation was believed to be impeded. There are several parameters that must be estimated for each of the respective models, including quantity deposited on skin, surface area of contact, evaporative flux, film thickness, and exposure duration. Many of these are likely to vary not only by condition of use but also the particulars of the individual activity patterns on a daily basis. Therefore, these parameters can only be approximated and the absorption estimates may either underestimate or overestimate the actual exposure of any particular worker or consumer on a given day, however they serve as a reasonable generalized approximation if not a higher-end bound.

The choice of one model over the other is primarily driven by the exposure scenario that needs to be assessed and the information that is reasonably available. For example, EPA does not know the exact duration of exposure for occupational loading and unloading hence EPA used the engineering model for occupational exposure assessment since it is event based and does not require a duration input. In contrast, for consumer applications there is reasonably available information for duration of use, hence the permeability model can be used for these exposure scenarios with greater confidence. Overall, the two models are considered appropriate for their respective uses based on the reasonably available information.

4.3.2.4 Confidence in Risk Estimates

Occupational Exposure Scenarios

There is varying confidence in inhalation exposure estimates from different occupational risk scenarios, ranging from low-to-medium to medium-to-high (see Table 2-12). Despite some OES with low to medium overall confidence, many of these are further supported by the availability of both monitoring

and modeling data, despite the uncertainties within each (see Table 2-26). Additionally, the data quality scores for monitoring data ranged from medium to high, and the inhalation modeling approach was peer reviewed during the 2014 TCE risk assessment process (U.S. EPA, 2014b) (for a subset of COUs). EPA acknowledges the uncertainty and lower confidence in applying worker estimates to represent ONUs in the absence of reasonably available ONU data for certain OES. Therefore, EPA has low confidence in risk estimates for ONUs based on this assumption. There is medium confidence in the occupational dermal modeling approach, which was developed from a peer-reviewed publication (Kasting and Miller, 2006).

Consumer Exposure Scenarios

There is medium to high confidence in consumer inhalation exposure modeling (see Section 2.3.2.8), however there is low to medium confidence in consumer dermal exposure modeling due to uncertainties related to absorption (as discussed above) and assumptions regarding impeded evaporation for particular conditions of use.

Human Health Hazard

The human health database covers a wide range of endpoints, with most health effects supported by animal, epidemiological, and mechanistic evidence. There is medium confidence in the integration of human health data for both acute non-cancer, medium to high confidence for cancer, and high confidence for chronic non-cancer endpoints, although there is additional uncertainty in the dose-response analysis for the congenital heart defects endpoint (see Section 3.2.6 for more details).

Risk Conclusions

For all exposure scenarios, the confidence in the risk estimates is raised due to the presence of both central tendency and high end estimates for occupational scenarios and low-, moderate-, and high-intensity user estimates for consumer scenarios. Any reduced confidence in individual exposure estimates is mitigated by the use of a range of exposure estimates, which cover a variety of different assumptions to account for any uncertainty and variability. Therefore, while there is lower confidence in various occupational inhalation estimates and for consumer dermal exposure estimates, there is high confidence in the overall approach and it is unlikely that any refinement of risk estimates would result in variation of more than a few fold in either direction.

In considering risk estimates relative to the benchmark MOE/extra risk, identified risks are typically present for multiple endpoints, at both high-end and central tendency (or high and medium-intensity user scenarios for consumers) exposure levels, for both inhalation and dermal exposure, and based on both monitoring and modeling data, when available (Sections 4.5.2.1 and 4.5.2.2). In accounting for the totality of uncertainties, including confidence levels for each exposure scenario/COU, strength of the human health hazard information, and range of risk estimates provided for the different aspects of the risk evaluation relative to the benchmark, confidence in the risk estimates for each of the receptors and exposure durations is as follows:

Acute Non-Cancer Inhalation Occupational Risk (workers): Medium
Acute Non-Cancer Dermal Occupational Risk (workers): Medium
Acute Non-Cancer Inhalation Occupational Risk (ONUs): Medium (Low[19] when based on central tendency of workers without ONU-specific data)

[19] EPA notes that while there is Low confidence in the accuracy of the risk estimates due to Low confidence in the exposure estimates in these instances, the risk conclusions (i.e. risk estimate below or above benchmark) does not change if ONU chronic exposure values are varied by 10x in either direction.

Chronic Inhalation Non-Cancer Occupational Risk (workers): High
Chronic Dermal Non-Cancer Occupational Risk (workers): Medium-High
Chronic Inhalation Non-Cancer Occupational Risk (ONUs): Medium-High (Low[19] when based on central tendency of workers without ONU-specific data)

Lifetime Cancer Inhalation Occupational Risk (workers): Medium-High
Lifetime Cancer Dermal Occupational Risk (workers): Medium-High
Lifetime Cancer Inhalation Occupational Risk (ONUs): Medium-High (Low[19] when based on central tendency of workers without ONU-specific data)

Acute Non-Cancer Inhalation Consumer Risk (users): Medium-High
Acute Non-Cancer Dermal Consumer Risk (users): Low-Medium
Acute Non-Cancer Inhalation Consumer Risk (bystanders): Medium-High

4.4 Other Risk Related Considerations

4.4.1 Potentially Exposed or Susceptible Populations

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. Dermal risk estimates were calculated for both average workers and women of childbearing age [*Occupational Risk Estimate Calculator*. *Docket # EPA-HQ-OPPT-2019-0500*], based on differences in delivered dose accounting for differing body weight and hand size. Exposures differ by only ~10% between these groups, so this difference is relatively insignificant considering the magnitude of risk estimates relative to the benchmark MOE. Accordingly, the risk characterization section only presents dermal risk estimates for average adult workers (Section 4.2.2). Similarly, risk estimates were provided for each of the three lifestages that are expected to potentially be directly exposed through consumer use, namely 11-15 year olds, 16-20 year olds, and adults 21 and over (Section 4.2.3). These risk estimates also only varied by a small percentage relative to the magnitude of risk estimates relative to the benchmark MOE. EPA determined that bystanders may include lifestages of any age.

For inhalation exposures, risk estimates did not differ between genders 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 voume) which may affect internal delivered concentration or dose is sufficiently accounted for in the PBPK model, although some differences among lifestages or between working and at-rest individuals may not have been accounted for. The use of HEC/HED_{99} values is expected to account for the vast majority of physiological differences among individuals.

EPA identified lifestage, gender, genetic polymorphisms, race/ethnicity, preexisting health status, and lifestyle factors and nutrition status as factors affecting biological susceptibility. The use of HEC/HED_{99} POD values derived from relevant PBPK dose metrics accounts for the vast majority of toxicokinetic variation across the population. By relying on the 99th percentile output of the PBPK model, these values are expected to be protective of particularly susceptible subpopulations, including those with genetic polymorphisms resulting in increased activity of bioactivating enzymes. The (Selgrade and Gilmour, 2010) study accounts for pre-existing infection concurrent with TCE exposure, representing a susceptible status that applies intermittently to the entire population. Cardiac malformations are most strongly associated with offspring of older mothers (Brender et al., 2014; Yauck et al., 2004). While inconsistencies in the data on cardiac malformations (Appendix G.2) suggest that there may nor be a risk for all individuals, inclusion of risk estimates for cardiac malformations is protective of susceptible mothers (Jenkins et al., 2007) and their offspring.

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*" (40 CFR § 702.33). In this risk evaluation, EPA determined that aggregating dermal and inhalation exposure for risk characterization was not appropriate due to uncertainties in quantifying the relative contribution of dermal vs inhalation exposure, since dermally applied dose could evaporate and then be inhaled. Aggregating exposures from multiple routes could therefore inappropriately overestimate total exposure,

as simply adding exposures from different routes without an available PBPK model for those routes would compound uncertainties. EPA also did not consider aggregate exposure among individuals who may be exposed both in an occupational and consumer context because there is insufficient information reasonably available as to the likelihood of this scenario or the relative distribution of exposures from each pathway.

EPA defines sentinel exposure as "*the exposure to a single chemical substance that represents the plausible upper bound of exposure relative to all other exposures within a broad category of similar or related exposures*" (40 CFR § 702.33). In terms of this risk evaluation, EPA considered sentinel exposures by considering risks to populations who may have upper bound exposures – for example, workers and ONUs who perform activities with higher exposure potential, or consumers who have higher exposure potential (e.g., those involved with do-it-yourself projects) or certain physical factors like body weight or skin surface area exposed. EPA characterized high-end exposures in evaluating exposure using both monitoring data and modeling approaches. Where statistical data are reasonably available, EPA typically uses the 95th percentile value of the reasonably available dataset to characterize high-end exposure for a given condition of use. For consumer and bystander exposures, EPA characterized sentinel exposure through a "high-intensity use" category based on both product and user-specific factors.

4.5 Risk Conclusions

4.5.1 Environmental Risk Conclusions

Risks to aquatic organisms like fish and invertebrates were identified near one open-top vapor degreasing facility and one facility that processes TCE as a reactant (See Table 4-53). These facilities had an acute RQ ≥ 1, or a chronic RQ ≥ 1 and 20 days or more of exceedance for the chronic COC. Risk to the most sensitive species of algae were identified near 521 facilities (with 20 days or more of exceedances for 461 of these facilities, and more than 100 days exceedances for 10 facilities); however, as a taxonomic group, 95% of algae species not experience risk. (They had RQs ≥ 1 using the algae COC of 3 ppb but RQs < 1 using the algae HC_{05} of 52,000 ppb.) These facilities are not included in Table 4-53 in this section, but are in Table 4-1 for reference.

EPA did not identify risks to aquatic organisms like fish and invertebrates in the ambient water where monitored data were reasonably available. Monitored data from the Water Quality Portal and the reasonably available literature show no exceedances of the acute COC, or chronic COC in ambient water. Monitored data from literature showed some exceedances of the algae COC of 3 ppb in ambient water; however, the data show no exceedances of the algae COC of 52,000 ppb.

Near-facility monitoring data report levels of TCE ranging from 0.4 to 447 µg/L (U.S. EPA, 1977). These data show that measured, near-facility concentrations compare to the modeled near-facility concentrations from E-FAST. With the exception of two sites, the measured concentrations in this study encompasses the range of the modeled estimates across all OES from E-FAST.

Open-top Vapor Degreasing:
One out of 64 open-top vapor degreasing facilities had releases of TCE to surface water that indicate risk to aquatic organisms. U.S. NASA Michoud Assembly Facility in New Orleans, LA had an acute RQ ≥ 1 (RQ = 3.11). In other words, the surface water concentration modeled for this facility was 3.11 times higher than the acute COC of 3,200 ppb, indicating risk to aquatic organisms from acute exposures. The facility also had a chronic RQ of 12.61 with 20 days of exceedance. In other words, the surface water concentration was 12.61 higher than the COC of 788 for 20 days. *Therefore, EPA identified risk to aquatic organisms at this site for acute and chronic exposures to TCE.*

Processing as a Reactant:
One out of 443 facilties (including 440 unknown sites modeled in E-FAST) that process TCE as a reactant had releases of TCE to surface water that indicate risk to aquatic organisms like fish and invertebrates. Praxair Technology Center in Tonawanda, NY had a chronic RQs of 3.81 with 20 days of exceedance. In other words, the surface water concentration modeled for this facility was 3.81 times higher than the COC for chronic exposures. *Therefore, EPA identified risk to aquatic organisms at this site for chronic exposures to TCE.*

Table 4-53. Facilities with Acute or Chronic Risk Identified for Aquatic Organisms (RQs ≥ 1 in **bold)**

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	COC Type	COC (ppb)	Days of Exceedance (days/year) [h]	Risk Quotient
OES: Processing as a Reactant										
Praxair Technology Center, Tonawanda, NY NPDES: NY0000281	Surface Water	NPDES NY0000281	Still body	350	0.00169	169	Acute	3,200	NA	0.05
							Chronic	788	0	0.21
							Algae	3	350	**56.33**
							Algae (HC_{05})	52,000	0	0.00
				20	0.03	3000	Acute	3,200	NA	0.94
							Chronic	788	20	**3.81**
							Algae	3	20	**1,000.00**
							Algae (HC_{05})	52,000	0	0.06
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, and Metalworking Fluids)										
US Nasa Michoud Assembly Facility, New Orleans, LA NPDES: LA0052256	Surface Water	Surrogate NPDES LA0003280	Still body	260	1.96	765.63	Acute	3,200	NA	0.24
							Chronic	788	0	0.97
							Algae (COC)	3	260	**255.21**
							Algae (HC_{05})	52,000	0	0.01
				20	25.44	9937.5	Acute	3,200	NA	**3.11**
							Chronic	788	20	**12.61**
							Algae	3	20	**3,312.50**
							Algae (HC_{05})	52,000	0	0.19

a. Facilities actively releasing trichloroethylene 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 81% 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 reasonably available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.
d. EFAST uses ether the "surface water" model, for rivers and streams, or the "still water" model, for lakes, bays, and oceans.
e. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.
f. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.
g. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.
h. To determine the PDM days of exceedance for still bodies of water, the release days provided by the EPA Engineers should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.

4.5.2 Human Health Risk Conclusions

4.5.2.1 Summary of Risk Estimates for Workers and ONUs

Table 4-54 summarizes the representative 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 in gray. When both monitoring and modeling inhalation exposures were available, EPA presented the most reliable data source in the table. The occupational exposure assessment and risk characterization are described in more detail in Sections 2.3.1 and 4.2.2, respectively. Specific links to the relevant risk characterization sections are listed in Table 4-54 in the Occupational Exposure Scenario column.

Of note, the risk summary below is based on the most robust and well-supported PODs selected from among the most sensitive acute and chronic non-cancer endpoints, as well as cancer. EPA selected immunosuppression (Selgrade and Gilmour, 2010) as the best overall representative acute endpoint, and autoimmunity from the immunotoxicity domain (Keil et al., 2009) was selected to best represent chronic exposure (Section 3.2.6.4). For the majority of exposure scenarios, risks were identified for multiple endpoints in both acute and chronic exposure scenarios, however risk estimates are only summarized for these particular endpoints. Risk estimates are also presented considering PPE up to respirator APF 50 and glove PF 10 or 20. When risks did not exceed the benchmark, the lowest protection factor that results in no risk is shown (i.e., if risks do not exceed the benchmark for APF 10 and above, the risk estimate for APF 10 is shown).

Inhalation Exposure

For acute and chronic exposures via inhalation without PPE (i.e. no respirators) there are risks for workers relative to the benchmarks for all the OES at the high-end exposure level for non-cancer effects from both acute and chronic exposure durations as well as for cancer. Occupational non-users (ONUs) are expected to have lower exposure levels than workers in most instances but exposures could not always be quantified. Therefore, when separate ONU exposure estimates were not reasonably available, EPA provided risk estimates for ONUs based on worker values (without PPE). These instances are indicated in Table 4-54 with “upper limit” added to the ONU cell in the Population column. Risks to ONUs were indicated at high-end exposure levels for all OES following chronic exposure and for most OES following acute exposure, although central-tendency exposure levels are considered more representative for ONUs.

When only considering central tendency inhalation exposure level, risks for any endpoint were not identified to workers or ONUs for the following exposure scenarios:

- Formulation of Aerosol and Non-Aerosol Products
- Repackaging
- Process Solvent Recycling and Worker Handling of Wastes

When respirators are worn (either APF 10 or 50) there are risks relative to the benchmarks for non-cancer effects and for cancer for workers (ONUs are assumed to not consistently wear respirators) from both acute and chronic exposure durations at high-end exposure levels for the majority of OES (risks remain with respirator use for all exposure scenarios following chronic exposure). Risks for any endpoint were not identified when assuming the maximum plausible APF (up to APF =50) and central tendency exposure levels for the same exposure scenarios that did not demonstrate risk without PPE:

- Formulation of Aerosol and Non-Aerosol Products

- Repackaging
- Process Solvent Recycling and Worker Handling of Wastes

Dermal Exposure

For acute and chronic exposures via dermal contact without PPE (i.e. no gloves) there are risks to workers for both non-cancer effects and cancer (ONUs are assumed to not have direct dermal contact with TCE) at both high-end and central-tendency exposure levels for all OES. Risks are still identified for all exposure scenarios (at high-end exposure levels following acute exposure and at both exposure levels following chronic exposure) when gloves are worn even when assuming the maximum applicable glove protection (either PF 10 or 20).

1709 **Table 4-54. Occupational Risk Summary Table**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Manufacture - Domestic manufacture	Domestic manufacture	Manufacturing - Table 4-6	Worker	Inhalation	High-End	**2.0**	**5.6E-02**	**6.7E-03**	100.8 (APF 50)	**2.8 (APF 50)**	**1.3E-04 (APF 50)**
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	139.1 (APF 10)	**19.3 (APF 50)**	7.5E-05 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	**N/A**		
Manufacture - Import	Import	Repackaging - Table 4-19	Worker	Inhalation	High-End	**4.6**	**0.13**	**2.9E-03**	45.8 (APF 10)	**6.3 (APF 50)**	5.9E-05 (APF 50)
					Central Tendency	10546	292	9.9E-07	105460 (APF 10)	2920 (APF 10)	9.9E-08 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	10546	292	9.9E-07	N/A		
Processing - Processing as a reactant/ intermediate	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as	Processing as a Reactant - Table 4-7	Worker	Inhalation	High-End	**2.0**	**5.6E-02**	**6.7E-03**	100.8 (APF 50)	**2.8 (APF 50)**	**1.3E-04 (APF 50)**
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	139.1 (APF 10)	**19.3 (APF 50)**	7.5E-05 (APF 10)

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
	refrigerants, foam blowing agents and solvents)			Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	**N/A**		
Processing - Incorporation into formulation, mixture or reaction product	Solvents (for cleaning or degreasing)	Formulation of Aerosol and Non-Aerosol Products - Table 4-18	Worker	Inhalation	High-End	**4.6**	**0.13**	**2.9E-03**	45.8 (APF 10)	**6.3 (APF 50)**	5.9E-05 (APF 50)
	Adhesives and sealant chemicals				Central Tendency	10546	292	9.9E-07	105460 (APF 10)	2920 (APF 10)	9.9E-08 (APF 10)
	Solvents (which become part of product formulation or mixture) (e.g., lubricants and greases, paints and coatings, other uses)			Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
Processing - incorporated into articles	Solvents (becomes an integral component of articles)				Central Tendency	10546	292	9.9E-07	N/A		
Processing - Repackaging	Solvents (for cleaning or degreasing)	Repackaging - Table 4-19	Worker	Inhalation	High-End	**4.6**	**0.13**	**2.9E-03**	45.8 (APF 10)	**6.3 (APF 50)**	5.9E-05 (APF 50)
					Central Tendency	10546	292	9.9E-07	105460 (APF 10)	2920 (APF 10)	9.9E-08 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	10546	292	9.9E-07	N/A		
Processing - Recycling	Recycling	Process Solvent Recycling and Worker Handling of Wastes - Table 4-27	Workers	Inhalation	High-End	**4.6**	**0.13**	**2.9E-03**	45.8 (APF 10)	**6.3 (APF 50)**	5.9E-05 (APF 50)
					Central Tendency	10546	292	9.9E-07	105460 (APF 10)	2920 (APF 10)	9.9E-08 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	10546	292	9.9E-07	N/A		
Distribution in commerce - Distribution	Distribution	Distribution	Distribution is accounted for as part of other COUs								
Industrial/ commercial use - Solvents (for cleaning or degreasing)	Batch vapor degreaser (e.g., open-top, closed-loop)	Batch Open-Top Vapor Degreasing - Table 4-8	Workers	Inhalation (Monitoring Data) [a]	High-End	**6.7E-02**	**1.9E-03**	**0.20**	**3.4 (APF 50)**	**9.3E-02 (APF 50)**	**4.0E-03 (APF 50)**
					Central Tendency	**0.38**	**1.0E-02**	**2.8E-02**	**18.9 (APF 50)**	**0.52 (APF 50)**	**5.5E-04 (APF 50)**
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Solvents (for cleaning or degreasing)			ONU	Inhalation (Monitoring Data) [a]	High-End	**0.57**	**1.6E-02**	**2.3E-02**	**N/A**		
					Central Tendency	**4.7**	**0.13**	**2.2E-03**	**N/A**		
		Batch Closed-Loop Vapor Degreasing - Table 4-10	Workers	Inhalation	High-End	**3.6**	**9.9E-02**	**3.7E-03**	35.9 (APF 10)	**5.0 (APF 50)**	7.5E-05 (APF 50)
					Central Tendency	**11.4**	**0.32**	**9.1E-04**	114.0 (APF 10)	**15.8 (APF 50)**	9.1E-05 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	**11.4**	**0.32**	**9.1E-04**	**N/A**		
	In-line vapor degreaser (e.g., conveyorized, web cleaner)	Conveyorized Vapor Degreasing - Table 4-11	Workers	Inhalation (Monitoring Data) [a]	High-End	**0.11**	**3.0E-03**	**0.12**	**5.4 (APF 50)**	**0.15 (APF 50)**	**2.5E-03 (APF 50)**
					Central Tendency	**0.16**	**4.5E-03**	**6.5E-02**	**8.1 (APF 50)**	**0.22 (APF 50)**	**1.3E-03 (APF 50)**
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation (Monitoring Data) [a]	High-End	-	-	-	-		
					Central Tendency	**0.16**	**4.5E-03**	**6.5E-02**	**N/A**		

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Solvents (for cleaning or degreasing)		Web Vapor Degreasing - Table 4-13	Workers	Inhalation	High-End	**0.37**	**1.0E-02**	**2.9E-02**	**18.5 (APF 50)**	**0.51 (APF 50)**	**5.8E-04 (APF 50)**
					Central Tendency	**0.88**	**2.4E-02**	**1.1E-02**	43.9 (APF 50)	**1.2 (APF 50)**	**2.3E-04 (APF 50)**
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU	Inhalation	High-End	**0.55**	**1.5E-02**	**1.9E-02**	**N/A**		
					Central Tendency	**1.7**	**4.6E-02**	**5.9E-03**	**N/A**		
	Cold cleaner	Cold Cleaning - Table 4-14	Worker	Inhalation	High-End	**9.1E-02**	**2.5E-03**	**0.11**	**4.6 (APF 50)**	**0.13 (APF 50)**	**2.3E-03 (APF 50)**
					Central Tendency	**1.6**	**4.3E-02**	**6.2E-03**	78.4 (APF 50)	**2.2 (APF 50)**	**1.2E-04 (APF 50)**
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
			ONU	Inhalation	High-End	**0.15**	**4.7E-04**	**6.9E-02**	**N/A**		
					Central Tendency	**2.8**	**8.8E-03**	**3.3E-03**	**N/A**		
	Aerosol spray degreaser/cleaner	Aerosol Applications - Table 4-15	Worker	Inhalation	High-End	**0.22**	**6.0E-03**	**4.9E-02**	**10.9 (APF 50)**	**0.30 (APF 50)**	**9.7E-04 (APF 50)**
					Central Tendency	**0.68**	**1.9E-02**	**1.4E-02**	34.2 (APF 50)	**0.95 (APF 50)**	**2.9E-04 (APF 50)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
	Mold release		ONU	Dermal	High-End	**0.76**	**1.9E-02**	**5.9E-02**	**15.1 (PF 20)**	**0.39 (PF 20)**	**2.9E-03 (PF 20)**
					Central Tendency	**2.3**	**5.8E-02**	**1.5E-02**	45.4 (PF 20)	**1.2 (PF 20)**	**7.6E-04 (PF 20)**
				Inhalation	High-End	**5.0**	**0.14**	**2.0E-03**	**N/A**		
					Central Tendency	37.3	**1.0**	**2.6E-04**	**N/A**		
Industrial/ commercial use - Lubricants and greases/ lubricants and lubricant additives	Tap and die fluid	Metalworking Fluids - Table 4-21	Worker	Inhalation (Modeling Data) [b]	High-End	**20.1**	**0.55**	**6.6E-04**	200.8 (APF 10)	**27.5 (APF 50)**	1.3E-05 (APF 50)
					Central Tendency	74.6	**2.1**	**1.3E-04**	745.7 (APF 10)	103.1 (APF 50)	2.6E-06 (APF 50)
				Dermal	High-End	**1.5**	**3.8E-02**	**3.0E-02**	**29.7 (PF 20)**	**0.76 (PF 20)**	**1.5E-03 (PF 20)**
					Central Tendency	**4.5**	**0.11**	**7.8E-03**	**0.17 (PF 20)**	**2.3 (PF 20)**	**3.9E-04 (PF 20)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	74.6	**2.1**	**1.3E-04**	**N/A**		
	Penetrating lubricant	Aerosol Applications - Table 4-15	Worker	Inhalation	High-End	**0.22**	**6.0E-03**	**4.9E-02**	**10.9 (APF 50)**	**0.30 (APF 50)**	**9.7E-04 (APF 50)**
					Central Tendency	**0.68**	**1.9E-02**	**1.4E-02**	34.2 (APF 50)	**0.95 (APF 50)**	**2.9E-04 (APF 50)**
				Dermal	High-End	**0.76**	**1.9E-02**	**5.9E-02**	**15.1 (PF 20)**	**0.39 (PF 20)**	**2.9E-03 (PF 20)**
					Central Tendency	**2.3**	**5.8E-02**	**1.5E-02**	45.4 (PF 20)	**1.2 (PF 20)**	**7.6E-04 (PF 20)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Lubricants and greases/ lubricants and lubricant additives			ONU	Inhalation	High-End	**5.0**	**0.14**	**2.0E-03**	**N/A**		
					Central Tendency	37.3	**1.0**	**2.6E-04**	**N/A**		
	Solvent-based adhesives and sealants	Adhesives, Sealants, Paints, and Coatings - Table 4-22 and Table 4-23	Worker	Inhalation	High-End	**0.13**	**3.7E-03**	**0.10**	**6.6 (APF 50)**	**0.18 (APF 50)**	**2.0E-03 (APF 50)**
					Central Tendency	**1.1**	**3.1E-02**	**9.3E-03**	56.3 (APF 50)	**1.6 (APF 50)**	**1.9E-04 (APF 50)**
				Dermal (Industrial)	High-End	**1.3**	**3.4E-02**	**3.4E-02**	**26.4 (PF 20)**	**0.68 (PF 20)**	**1.7E-03 (PF 20)**
	Tire repair cement/ Sealer				Central Tendency	**4.0**	**0.10**	**8.7E-03**	39.6 (PF 10)	**2.0 (PF 20)**	**4.4E-04 (PF 20)**
				Dermal (Commercial)	High-End	**0.84**	**2.2E-02**	**5.3E-02**	**8.4 (PF 10)**	**0.22 (PF 10)**	**5.3E-03 (PF 10)**
					Central Tendency	**2.5**	**6.5E-02**	**1.4E-02**	**25.2 (PF 10)**	**0.65 (PF 10)**	**1.4E-03 (PF 10)**
	Mirror edge sealant		ONU	Inhalation	High-End	**5.2**	**0.14**	**2.6E-03**	**N/A**		
					Central Tendency	**5.5**	**0.15**	**1.9E-03**	**N/A**		
Industrial/ commercial use - Functional fluids (closed systems)	Heat exchange fluid	Other Industrial Uses - Table 4-26	Worker	Inhalation	High-End	**2.0**	**5.6E-02**	**6.7E-03**	**100.8 (APF 50)**	**2.8 (APF 50)**	**1.3E-04 (APF 50)**
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	**139.1 (APF 10)**	**19.3 (APF 50)**	7.5E-05 (APF 10)
				Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	**35.7 (PF 10)**	**1.8 (PF 20)**	**4.9E-04 (PF 20)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Paints and coatings			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	**13.9**	**0.39**	**7.5E-04**	**N/A**		
	Diluent in solvent-based paints and coatings	Adhesives, Sealants, Paints, and Coatings - Table 4-22 and Table 4-23	Worker	Inhalation	High-End	**0.13**	**3.7E-03**	**0.10**	**6.6 (APF 50)**	**0.18 (APF 50)**	**2.0E-03 (APF 50)**
					Central Tendency	**1.1**	**3.1E-02**	**9.3E-03**	56.3 (APF 50)	**1.6 (APF 50)**	**1.9E-04 (APF 50)**
				Dermal (Industrial)	High-End	**1.3**	**3.4E-02**	**3.4E-02**	**26.4 (PF 20)**	**0.68 (PF 20)**	**1.7E-03 (PF 20)**
					Central Tendency	**4.0**	**0.10**	**8.7E-03**	39.6 (PF 10)	**2.0 (PF 20)**	**4.4E-04 (PF 20)**
				Dermal (Commercial)	High-End	**0.84**	**2.2E-02**	**5.3E-02**	**8.4 (PF 10)**	**0.22 (PF 10)**	**5.3E-03 (PF 10)**
					Central Tendency	**2.5**	**6.5E-02**	**1.4E-02**	**25.2 (PF 10)**	**0.65 (PF 10)**	**1.4E-03 (PF 10)**
			ONU	Inhalation	High-End	**5.2**	**0.14**	**2.6E-03**	**N/A**		
					Central Tendency	**5.5**	**0.15**	**1.9E-03**	**N/A**		
Industrial/ commercial use - Cleaning and furniture care products	Carpet cleaner	Spot Cleaning and Wipe Cleaning [c] - Table 4-17	Worker	Inhalation (Modeling Data) [b]	High-End	**1.9**	**5.1E-02**	**5.8E-03**	94.2 (APF 50) [c]	**2.5 (APF 50) [c]**	**1.2E-04 (APF 50) [c]**
					Central Tendency	**5.4**	**0.15**	**1.8E-03**	54.3 (APF 10) [c]	**7.3 (APF 50) [c]**	3.7E-05 (APF 10) [c]
	Wipe cleaning			Dermal	High-End	**0.76**	**1.7E-02**	**6.9E-02**	**7.6 (PF 10)**	**0.17 (PF 10)**	**6.9E-03 (PF 10)**
					Central Tendency	**2.3**	**5.6E-02**	**1.6E-02**	**22.7 (PF 10)**	**0.56 (PF 10)**	**1.6E-03 (PF 10)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Laundry and dishwashing products	Spot remover		ONU	Inhalation (Modeling Data) [b]	High-End	**3.0**	**8.0E-02**	**3.6E-03**	**N/A**		
					Central Tendency	**10.9**	**0.29**	**9.2E-04**	**N/A**		
Industrial/ commercial use - Arts, crafts and hobby materials	Fixatives and finishing spray coatings	Adhesives, Sealants, Paints, and Coatings - Table 4-22 and Table 4-23	Worker	Inhalation	High-End	**0.13**	**3.7E-03**	**0.10**	**6.6 (APF 50)**	**0.18 (APF 50)**	**2.0E-03 (APF 50)**
					Central Tendency	**1.1**	**3.1E-02**	**9.3E-03**	56.3 (APF 50)	**1.6 (APF 50)**	**1.9E-04 (APF 50)**
				Dermal (Industrial)	High-End	**1.3**	**3.4E-02**	**3.4E-02**	**26.4 (PF 20)**	**0.68 (PF 20)**	**1.7E-03 (PF 20)**
					Central Tendency	**4.0**	**0.10**	**8.7E-03**	39.6 (PF 10)	**2.0 (PF 20)**	**4.4E-04 (PF 20)**
				Dermal (Commercial)	High-End	**0.84**	**2.2E-02**	**5.3E-02**	**8.4 (PF 10)**	**0.22 (PF 10)**	**5.3E-03 (PF 10)**
					Central Tendency	**2.5**	**6.5E-02**	**1.4E-02**	**25.2 (PF 10)**	**0.65 (PF 10)**	**1.4E-03 (PF 10)**
			ONU	Inhalation	High-End	**5.2**	**0.14**	**2.6E-03**	**N/A**		
					Central Tendency	**5.5**	**0.15**	**1.9E-03**	**N/A**		
Industrial/ commercial use - Corrosion inhibitors and anti-scaling agents	Corrosion inhibitors and anti-scaling agents	Industrial Processing Aid - Table 4-24	Worker	Inhalation	High-End	**0.27**	**7.5E-03**	**4.9E-02**	**13.6 (APF 50)**	**3.0E-02 (APF 50)**	**9.9E-04 (APF 50)**
					Central Tendency	**0.82**	**2.3E-02**	**1.3E-02**	40.9 (APF 50)	**9.1E-02 (APF 50)**	**2.5E-04 (APF 50)**

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Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Industrial/ commercial use - Processing aids	Process solvent used in battery manufacture	Industrial Processing Aid - Table 4-24		Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
	Process solvent used in polymer fiber spinning, fluoroelastomer manufacture and Alcantara manufacture		ONU	Inhalation	High-End	**1.2**	**3.3E-02**	**1.1E-02**	**N/A**		
	Extraction solvent used in caprolactam manufacture				Central Tendency	**2.7**	**7.3E-02**	**3.9E-03**	**N/A**		
	Precipitant used in beta-cyclodextrin manufacture										
Industrial/ commercial use - Ink, toner and colorant products	Toner aid	Commercial Printing and Copying [c] - Table 4-25	Workers	Inhalation	High-End	**2.5**	**6.9E-02**	**5.4E-03**	124.6 (APF 50) [c]	**3.4 (APF 50) [c]**	**1.1E-04 (APF 50) [c]**
					Central Tendency	61.4	**1.7**	**1.7E-04**	614.1 (APF 10) [c]	85.0 (APF 50) [c]	1.7E-05 (APF 10) [c]
				Dermal	High-End	**2.2**	**5.5E-02**	**2.1E-02**	**21.6 (PF 10)**	**0.55 (PF 10)**	**2.1E-03 (PF 10)**
					Central Tendency	**6.5**	**0.17**	**5.3E-03**	32.5 (PF 5)	**1.7 (PF 10)**	**5.3E-04 (PF 10)**
			ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	61.4	**1.7**	**1.7E-04**	**N/A**		
Industrial/ commercial use - Automotive care products	Brake and parts cleaner	Aerosol Applications - Table 4-15	Workers	Inhalation	High-End	**0.22**	**6.0E-03**	**4.9E-02**	**10.9 (APF 50)**	**0.30 (APF 50)**	**9.7E-04 (APF 50)**
					Central Tendency	**0.68**	**1.9E-02**	**1.4E-02**	34.2 (APF 50)	**0.95 (APF 50)**	**2.9E-04 (APF 50)**

Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
				Dermal	High-End	**0.76**	**1.9E-02**	**5.9E-02**	**15.1 (PF 20)**	**0.39 (PF 20)**	**2.9E-03 (PF 20)**
					Central Tendency	**2.3**	**5.8E-02**	**1.5E-02**	45.4 (PF 20)	**1.2 (PF 20)**	**7.6E-04 (PF 20)**
			ONU	Inhalation	High-End	**5.0**	**0.14**	**2.0E-03**	**N/A**		
					Central Tendency	37.3	**1.0**	**2.6E-04**	**N/A**		
Industrial/ commercial use - Apparel and footwear care products	Shoe polish	Other Commercial Uses (Spot Cleaning and Wipe Cleaning) [c] - Table 4-17	Worker	Inhalation (Modeling Data) [b]	High-End	**1.9**	**5.1E-02**	**5.8E-03**	94.2 (APF 50) [c]	**2.5 (APF 50) [c]**	**1.2E-04 (APF 50) [c]**
					Central Tendency	**5.4**	**0.15**	**1.8E-03**	54.3 (APF 10) [c]	**7.3 (APF 50) [c]**	3.7E-05 (APF 10) [c]
				Dermal	High-End	**0.76**	**1.7E-02**	**6.9E-02**	**7.6 (PF 10)**	**0.17 (PF 10)**	**6.9E-03 (PF 10)**
					Central Tendency	**2.3**	**5.6E-02**	**1.6E-02**	**22.7 (PF 10)**	**0.56 (PF 10)**	**1.6E-03 (PF 10)**
Industrial/ commercial use - Other uses	Hoof polishes		ONU	Inhalation (Modeling Data) [b]	High-End	**3.0**	**8.0E-02**	**3.6E-03**	**N/A**		
	Gun Scrubber								**N/A**		
	Pepper spray				Central Tendency	**10.9**	**0.29**	**9.2E-04**	**N/A**		
	Other miscellaneous industrial and commercial uses										

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Life Cycle Stage/ Category	Subcategory	Occupational Exposure Scenario	Population	Exposure Route and Duration	Exposure Level	Risk Estimates for No PPE			Risk Estimates with PPE		
						Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})	Acute Non-Cancer (benchmark MOE = 30)	Chronic Non-Cancer (benchmark MOE = 30)	Cancer (benchmark = 10^{-4})
Disposal	Industrial pre-treatment	Process Solvent Recycling and Worker Handling of Wastes - Table 4-27	Workers	Inhalation	High-End	**4.6**	**0.13**	**2.9E-03**	45.8 (APF 10)	**6.3 (APF 50)**	5.9E-05 (APF 50)
					Central Tendency	10546	292	9.9E-07	105460 (APF 10)	2920 (APF 10)	9.9E-08 (APF 10)
	Industrial wastewater treatment			Dermal	High-End	**1.2**	**3.0E-02**	**3.8E-02**	**23.8 (PF 20)**	**0.61 (PF 20)**	**1.9E-03 (PF 20)**
					Central Tendency	**3.6**	**9.1E-02**	**9.7E-03**	35.7 (PF 10)	**1.8 (PF 20)**	**4.9E-04 (PF 20)**
	Publicly owned treatment works (POTW)		ONU (upper limit)	Inhalation	High-End	-	-	-	-		
					Central Tendency	10546	292	9.9E-07	N/A		

[a] Monitoring data was selected as most representative based on the EPA data hierarchy where high-quality monitoring data is preferred over modeling results or exposure limits.
[b] Modeling data was selected as most representative because the monitoring dataset contained a very low number of datapoints.
[c] EPA believes that small commercial facilities performing spot cleaning, wipe cleaning, and other related commercial uses as well as commercial printing and copying are unlikely to have a respiratory protection program. Therefore, the use of respirators is unlikely for workers in these facilities.
N/A = Not Applicable. ONUs are assumed to not wear respiratory protection.

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4.5.2.2 Summary of Risk Estimates for Consumers and Bystanders

Table 4-55 summarizes the risk estimates for CNS effects from acute inhalation and dermal exposures for all consumer exposure scenarios. Risk estimates that exceed the benchmark (i.e. MOEs less than the benchmark MOE) are highlighted by bolding the number and shading the cell in gray. The consumer exposure assessment and risk characterization are described in more detail in Sections 2.3.2 and 4.2.3, respectively. Specific links to the relevant risk characterization sections are listed in Table 4-55 in the Consumer Condition of Use Scenario column.

Of note, the risk summary below is based on the most robust and well-supported PODs selected from among the most sensitive acute and chronic non-cancer endpoints, as well as cancer. EPA selected immunosuppression (Selgrade and Gilmour, 2010) as the best overall representative acute endpoint (Section 3.2.6.4). For the majority of exposure scenarios, risks were identified for multiple endpoints, however risk estimates are only summarized for this particular endpoint.

Inhalation

For acute inhalation exposures there are risks for non-cancer effects for consumer users relative to the benchmarks for all COUs except Pepper Spray and for bystanders for most COUs at both medium and high-intensity user exposure levels.

Dermal

For acute dermal exposures there are risks for non-cancer effects for consumer users (bystanders are assumed to not have direct dermal contact with TCE) relative to the benchmarks for all COUs where dermal exposure is expected at both medium and high-intensity user exposure levels (and for most COUs at low-intensity).

Table 4-55. Consumer Risk Summary Table

Life Cycle Stage/ Category	Subcategory/ Consumer Condition of Use Scenario	Population	Exposure Route and Duration	Age Group	Acute Non-Cancer (benchmark MOE = 30)		
					High-Intensity User	Moderate-Intensity User	Low-Intensity User
Consumer Use - Solvents (for cleaning or degreasing)	Brake and Parts Cleaner - Table 4-28	User	Inhalation	All [a]	**3.5E-02**	**0.21**	**2.7**
			Dermal	21+	**3.6E-02**	**0.48**	**22**
				16-20	**3.8E-02**	**0.51**	**23**
				11-15	**3.5E-02**	**0.47**	**21**
		Bystander	Inhalation	All	**0.14**	**0.94**	**12**
	Aerosol electronic degreaser/cleaner - Table 4-29	User	Inhalation	All	**5.0E-02**	**1.2**	**33**
		Bystander	Inhalation	All	**0.28**	**7.1**	193
	Liquid electronic degreaser/cleaner - Table 4-30	User	Inhalation	All	**5.2E-02**	**0.79**	**11**
			Dermal	21+	**6.4E-02**	**9.5E-01**	**3.8**
				16-20	**6.8E-02**	**1.0**	**4.1**
				11-15	**6.2E-02**	**9.4E-01**	**3.7**
		Bystander	Inhalation	All	**0.29**	**4.8**	61

Life Cycle Stage/ Category	Subcategory/ Consumer Condition of Use Scenario	Population	Exposure Route and Duration	Age Group	Acute Non-Cancer (benchmark MOE = 30)		
					High-Intensity User	Moderate-Intensity User	Low-Intensity User
	Aerosol spray degreaser/cleaner - Table 4-31	User	Inhalation	All	**1.2E-02**	**4.6E-02**	**0.31**
			Dermal	21+	**3.8E-02**	**0.31**	**1.5**
				16-20	**4.1E-02**	**0.33**	**1.6**
				11-15	**3.7E-02**	**0.30**	**1.5**
		Bystander	Inhalation	All	**4.9E-02**	**0.21**	**1.4**
	Liquid degreaser/cleaner - Table 4-32	User	Inhalation	All	**1.3E-02**	**0.12**	**0.71**
			Dermal	21+	**1.6E-02**	**0.13**	**0.96**
				16-20	**1.7E-02**	**0.14**	**1.0**
				11-15	**1.6E-02**	**0.13**	**0.94**
		Bystander	Inhalation	All	**6.1E-02**	**0.70**	**4.3**
	Aerosol gun scrubber - Table 4-33	User	Inhalation	All	**26**	**24**	41
			Dermal	21+	**4.0E-02**	**0.32**	**4.0**
				16-20	**4.2E-02**	**0.34**	**4.2**
				11-15	**3.9E-02**	**0.31**	**3.9**
		Bystander	Inhalation	All	120	141	247
	Liquid gun scrubber - Table 4-34	User	Inhalation	All	**30**	**28**	**30**
			Dermal	21+	**1.7E-02**	**0.14**	**1.0**
				16-20	**1.8E-02**	**0.15**	**1.1**
				11-15	**1.7E-02**	**0.13**	**1.0**
		Bystander	Inhalation	All	140	164	172
	Mold Release - Table 4-35	User	Inhalation	All	**0.11**	**1.1**	**11**
		Bystander	Inhalation	All	**0.64**	**6.4**	61
	Aerosol Tire Cleaner - Table 4-36	User	Inhalation	All	**0.13**	**0.46**	**3.3**
			Dermal	21+	**0.17**	**0.70**	**3.0**
				16-20	**0.19**	**0.74**	**3.2**
				11-15	**0.17**	**0.68**	**2.9**
		Bystander	Inhalation	All	**0.32**	**2.0**	**15**
	Liquid Tire Cleaner - Table 4-37	User	Inhalation	All	**4.2E-02**	**0.21**	**1.0**
			Dermal	21+	**3.1E-02**	**0.12**	**0.37**
				16-20	**3.3E-02**	**0.13**	**0.40**
				11-15	**3.0E-02**	**0.12**	**0.37**
		Bystander	Inhalation	All	**0.14**	**0.92**	**4.7**
Consumer Use - Lubricants and greases	Tap and Die Fluid - Table 4-38	User	Inhalation	All	**0.13**	**1.2**	**6.8**
		Bystander	Inhalation	All	**0.71**	**7.1**	**28**
		User	Inhalation	All	**0.16**	**2.7**	86

Life Cycle Stage/ Category	Subcategory/ Consumer Condition of Use Scenario	Population	Exposure Route and Duration	Age Group	Acute Non-Cancer (benchmark MOE = 30)		
					High-Intensity User	Moderate-Intensity User	Low-Intensity User
	Penetrating lubricant - Table 4-39	Bystander	Inhalation	All	**0.89**	**16**	496
Consumer Use - Adhesives and sealants	Solvent-based adhesives and sealants - Table 4-40	User	Inhalation	All	**5.6E-02**	**1.8**	207
		Bystander	Inhalation	All	**0.52**	**20**	1602
	Mirror edge sealant - Table 4-41	User	Inhalation	All	**0.57**	**1.7**	83
		Bystander	Inhalation	All	**2.7**	**10**	513
	Tire repair cement/ sealer - Table 4-42	User	Inhalation	N/A	**0.17**	**2.9**	32
		Bystander	Inhalation	N/A	**0.57**	**13**	133
Consumer use - Cleaning and furniture care products	Carpet cleaner - Table 4-43	User	Inhalation	All	**3.6E-02**	**0.29**	**1.7**
			Dermal	21+	**5.9E-02**	**0.35**	**7.1**
				16-20	**6.3E-02**	**0.38**	**7.5**
				11-15	**5.7E-02**	**0.35**	**6.9**
		Bystander	Inhalation	All	**0.18**	**1.7**	**9.0**
	Aerosol Spot Remover - Table 4-44	User	Inhalation	All	**5.6E-02**	**0.47**	**3.2**
			Dermal	21+	**0.50**	**3.0**	**30**
				16-20	**0.53**	**3.2**	32
				11-15	**0.49**	**2.9**	**29**
		Bystander	Inhalation	All	**0.61**	**5.6**	30
	Liquid Spot Remover - Table 4-45	User	Inhalation	All	**4.7E-02**	**0.39**	**3.4**
			Dermal	21+	**8.5E-02**	**0.51**	**7.7**
				16-20	**9.1E-02**	**0.55**	**8.2**
				11-15	**8.4E-02**	**0.50**	**7.5**
		Bystander	Inhalation	All	**0.26**	**2.4**	**19**
Consumer use - Arts, crafts, and hobby materials	Fixatives and finishing spray coatings - Table 4-46	User	Inhalation	All	**0.20**	**1.2**	**6.4**
		Bystander	Inhalation	All	**0.92**	**7.6**	37
Consumer use - Apparel and footwear care products	Shoe polish - Table 4-47	User	Inhalation	All	**0.55**	**5.4**	31
			Dermal	21+	**0.91**	**5.5**	55
				16-20	**0.97**	**5.8**	58
				11-15	**0.89**	**5.3**	53
		Bystander	Inhalation	All	**3.1**	33	180
Consumer use - Other consumer uses	Fabric spray - Table 4-48	User	Inhalation	All	**7.2E-02**	**0.43**	**2.5**
		Bystander	Bystander	All	**0.66**	**5.1**	**23**
	Film cleaner -	User	Inhalation	All	**3.0E-02**	**0.18**	**0.93**

Life Cycle Stage/ Category	Subcategory/ Consumer Condition of Use Scenario	Population	Exposure Route and Duration	Age Group	Acute Non-Cancer (benchmark MOE = 30)		
					High-Intensity User	Moderate-Intensity User	Low-Intensity User
	Table 4-49	Bystander	Bystander	All	**0.13**	**1.1**	**5.4**
	Hoof polish - Table 4-50	User	Inhalation	All	**0.79**	**8.0**	56
		Bystander	Bystander	All	157	3653	22309
	Pepper spray - Table 4-51	User	Inhalation	All	98		
		Bystander					
	Toner aid - Table 4-52	User	Inhalation	All	**0.21**	**1.3**	**6.8**
		Bystander	Bystander	All	**0.97**	**8.0**	39

[a] Inhalation exposures are based on a 2-zone model of air concentrations (Section 2.3.2.4.1) that are independent of any age-specific exposure factors.

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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).[20]

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 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 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 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.

[20] 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 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 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 x 10^{-6} to 1x 10^{-4} as the appropriate benchmark for the general population, consumer users, and non-occupational PESS.[21]

For the subject chemical substance, the EPA, consistent with case law and 2017 NIOSH guidance,[22] used 1 x 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:

RQ = Environmental Concentration / 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 presumed. 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

[21] As an example, when EPA's Office of Water in 2017 updated the Human Health Benchmarks for Pesticides, the benchmark for a "theoretical upper-bound excess lifetime cancer risk" from pesticides in drinking water was identified as 1 in 1,000,000 to 1 in 10,000 over a lifetime of exposure (EPA. Human Health Benchmarks for Pesticides: Updated 2017 Technical Document. January 2017. https://www.epa.gov/sites/production/files/2015-10/documents/hh-benchmarks-techdoc.pdf). Similarly, EPA's approach under the Clean Air Act to evaluate residual risk and to develop standards is a two-step approach that includes a "presumptive limit on maximum individual lifetime [cancer] risk (MIR) of approximately 1 in 10 thousand" and consideration of whether emissions standards provide an ample margin of safety to protect public health "in consideration of all health information, including the number of persons at risk levels higher than approximately 1 in 1 million, as well as other relevant factors" (54 FR 38044, 38045, September 14, 1989).

[22] International Union, UAW v. Pendergrass, 878 F.2d 389 (D.C. Cir. 1989), citing Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607 (1980) ("Benzene decision"), in which it was found that a lifetime cancer risk of 1 in 1,000 was found to be clearly significant; and NIOSH (Whittaker et al., 2016). Current intelligence bulletin 68: NIOSH chemical carcinogen policy, available at https://www.cdc.gov/niosh/docs/2017-100/pdf/2017-100.pdf .

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 may be considered (e.g., exposure scenario, uncertainty, severity of effect) for purposes of making a risk determination.

5.2 Risk Determinations for TCE

EPA's preliminary determinations of unreasonable risk for specific conditions of use of TCE listed below are based on health risks to workers and occupational non-users (ONUs) during occupational exposures, and to consumers and bystanders during exposures to consumer uses.

As described in section 4, significant risks associated with more than one adverse effect (e.g., developmental toxicity, reproductive toxicity, liver toxicity, kidney toxicity, immunotoxicity, neurotoxicity, and cancer) were identified for particular conditions of use. While congenital heart defects were the most sensitive endpoint for TCE, for the purpose of the draft risk determination, there are uncertainties which decrease EPA's confidence in this endpoint. Section 26 of TSCA requires that EPA make decisions consistent with the "best available science." Section 26 also requires other scientific considerations including consideration of the "extent of independent verification" and "weight of the scientific evidence." As described in EPA's framework rule for risk evaluation [82 FR 33726] weight of the scientific evidence includes consideration of the "strengths, limitations and relevance of the information." Neither the statute nor the framework rule require that EPA choose the lowest number and EPA believes that public health is best served when EPA relies upon the highest quality information for which EPA has the greatest confidence. Based on these considerations, EPA is relying upon immunosuppression for acute inhalation and dermal exposures, and autoimmunity for chronic inhalation and dermal exposures. In Table 5-1 and Section 5.3 below, EPA has identified immunosuppression and autoimmunity as the critical endpoints for determining whether or not a condition of use presents unreasonable risks. EPA has the most confidence in these endpoints and it is expected that addressing risks for these effects would address other identified risks. For the majority of the occupational and consumer conditions of use, unreasonable risk determinations were consistent whether based on congenital heart defects (an endpoint for which EPA has lower confidence) or immunosuppression and autoimmunity endpoints.

- 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 immunosuppression resulting from acute inhalation and dermal exposures, autoimmunity resulting from chronic inhalation and dermal exposures, and cancer resulting from chronic inhalation and dermal exposure. The determinations reflect the severity of the effects associated with the occupational exposures to TCE and incorporate consideration of expected 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 2.3.1.2.7.

- 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. 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. The drivers for EPA's determination of unreasonable risks to ONUs are immunosuppression resulting from acute

inhalation exposures, autoimmunity resulting from chronic inhalation exposures, and cancer resulting from chronic inhalation exposure. The determinations reflect the severity of the effects associated with the occupational exposures to TCE and the expected absence of PPE for ONUs. For dermal exposures, because ONUs are not expected to be dermally exposed to TCE, dermal risks to ONUs generally 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. Estimated numbers of occupational non-users are in Section 2.3.1.2.7.

- 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 immunosuppression 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. Estimated numbers of consumers are in Section 2.3.1.2.7.

- 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 is immunosuppression 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 TCE, dermal non-cancer risks to bystanders were not evaluated. Estimated numbers of bystanders are in Section 2.3.1.2.7.

As described below, risks to the environment and general population either were not relevant for these conditions of use or were evaluated and not found to be unreasonable. For the conditions of use where EPA found no unreasonable risk, EPA describes the estimated risks in Section 4.5.2 (Table 4-54 and Table 4-55).

- **Environmental risks:** EPA concluded that environmental exposures are expected for aquatic species for the conditions of use within the scope of the evaluation. EPA identified risks from acute and chronic exposures for aquatic organisms like aquatic invertebrates and fish near two facilities releasing TCE to surface water and risks to the most sensitive algae species near over 400 facilities. EPA did not identify any additional scenarios indicating unreasonable risk for aquatic organisms from exposures to TCE in surface waters. For aquatic organisms like aquatic invertebrates and fish, one facility had an acute RQ greater than 1 (RQ = 3.11), exceeding the acute COC of 3,200 ppb and indicating risk to aquatic organisms from acute exposures. This facility is one of 59 facilities modeled by EPA that use TCE for open-top vapor degreasing (see Section 4.5.1). Another facility had an acute RQ of 0.94 indicating some uncertainty about whether it would also pose risks to aquatic organisms from acute exposures. This facility is one of 11 facilities modeled by EPA that process TCE as a reactant (see Section 4.5.1). Both facilities had chronic RQs greater than 1, exceeding the chronic COC of 788 ppb for 20 days. The over 400 facilities with potential risks to the

most sensitive algae species (exceeding the algae COC of 3 ppb) did not show risks for algae species as a whole, as they showed no risks for 95% of algae species (no exceedances of the algae COC of 52,000 ppb). Monitored data from the Water Quality Portal and grey literature show no exceedances of the acute COC and the chronic COC in ambient water. Monitored data from literature showed some exceedances of the algae COC of 3 ppb in ambient water; however, the data show no exceedances of the algae COC of 52,000 ppb. Therefore, EPA did not identify risks for acute or chronic exposure durations in ambient water for areas where monitored data were reasonably available. Given the uncertainties in the modeling data and exceedance of the acute RQ for only one data point and of the chronic RQ for only two out of 70 facilities modeled, EPA does not consider these risks unreasonable (see Section 4.5.2).

- **General population:** Exposure pathways to the general population are covered by other statutes and consist of: the ambient air pathway (*i.e.*, TCE is listed as a HAP in the Clean Air Act (CAA)), the drinking water pathway (*i.e.*, National Primary Drinking Water Regulations (NPDWRs) are promulgated for TCE under the Safe Drinking Water Act), ambient water pathways (*i.e.*, TCE is a priority pollutant with recommended water quality criteria for protection of human health under the CWA), the biosolids pathway (*i.e.*, the biosolids pathway for TCE is currently being addressed in the CWA regulatory analytical process), disposal pathways (TCE 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 (U.S. EPA, 2018d).

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.

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 and occupational non-users)**
Manufacture – Import (includes repackaging and loading/unloading)	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)
Processing – Processing as a reactant/intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants, foam blowing agents and solvents)	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Processing – Incorporation into formulation, mixture or reaction product – Solvents (for cleaning or degreasing); adhesives and sealant chemicals; solvents (which become part of product formulation or mixture) (e.g., lubricants and greases, paints and coatings, other uses)	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)

Condition of Use	Unreasonable Risk Determination
Processing – Incorporation into articles – Solvents (becomes an integral components of articles)	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)
Processing – Repackaging – Solvents (for cleaning or degreasing)	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)
Processing – Recycling	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)
Distribution in Commerce	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (open-top)	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (closed-loop)	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (conveyorized)	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial 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)**
Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Cold cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner; mold release	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Lubricants and greases/lubricants and lubricant additives – Tap and die fluid	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Lubricants and greases/lubricants and lubricant additives – Penetrating lubricant	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Adhesives and sealants – Solvent-based adhesives and sealants; tire repair cement/sealer; mirror edge sealant	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Functional fluids (closed systems) – Heat exchange fluid	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Paints and coatings – Diluent in solvent-based paints and coatings	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**

Condition of Use	Unreasonable Risk Determination
Industrial/Commercial Use – Cleaning and furniture care products – Carpet cleaner; wipe cleaner	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Laundry and dishwashing products – Spot remover	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Arts, crafts and hobby materials – Fixatives and finishing spray coatings	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Corrosion inhibitors and anti-scaling agents – Corrosion inhibitors and anti-scaling agents	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Processing aids – Process solvent used in battery manufacture; process solvent used in polymer fiber spinning, fluoroelastomer manufacture, and Alcantara manufacture; extraction solvent used in caprolactam manufacture; precipitant used in beta-cyclodextrin manufacture	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Ink, toner and colorant products – Toner aid	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Automotive care products – Brake and parts cleaners	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Apparel and footwear care products – Shoe polish	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Industrial/Commercial Use – Other commercial uses – Hoof polishes; gun scrubber; pepper spray; other miscellaneous industrial and commercial uses	**Presents an unreasonable risk of injury to health (workers and occupational non-users)**
Disposal	**Presents an unreasonable risk of injury to health (workers)** Does not present an unreasonable risk of injury to health (occupational non-users)
Consumer Use – Solvents (for cleaning or degreasing) – Brake and parts cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Aerosol electronic degreaser/cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Liquid electronic degreaser/cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Liquid degreaser/cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**

Condition of Use	Unreasonable Risk Determination
Consumer Use – Solvents (for cleaning or degreasing) – Aerosol gun scrubber	**Presents an unreasonable risk of injury to health (consumers)** Does not present an unreasonable risk of injury to health (bystanders)
Consumer Use – Solvents (for cleaning or degreasing) – Liquid gun scrubber	**Presents an unreasonable risk of injury to health (consumers)** Does not present an unreasonable risk of injury to health (bystanders)
Consumer Use – Solvents (for cleaning or degreasing) – Mold release	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Aerosol tire cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Solvents (for cleaning or degreasing) – Liquid tire cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Lubricants and greases – Tap and die fluid	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Lubricants and greases – Penetrating lubricant	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Adhesives and sealants – Solvent-based adhesive and sealant	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Adhesives and sealants – Mirror edge sealant	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Adhesives and sealants – Tire repair cement/sealer	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Cleaning and furniture care products – Carpet cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Cleaning and furniture care products – Aerosol spot remover	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Cleaning and furniture care products – Liquid spot remover	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Arts, crafts, and hobby materials – Fixatives and finishing spray coatings	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Apparel and footwear care products – Shoe polish	**Presents an unreasonable risk of injury to health (consumers)** Does not present an unreasonable risk of injury to health (bystanders)
Consumer Use – Other consumer uses – Fabric spray	**Presents an unreasonable risk of injury to health (consumers and bystanders)**

Condition of Use	Unreasonable Risk Determination
Consumer Use – Other consumer uses – Film cleaner	**Presents an unreasonable risk of injury to health (consumers and bystanders)**
Consumer Use – Other consumer uses – Hoof polish	**Presents an unreasonable risk of injury to health (consumers)** Does not present an unreasonable risk of injury to health (bystanders)
Consumer Use – Other consumer uses – Pepper spray	Does not present an unreasonable risk of injury to health (consumers)
Consumer Use – Other consumer uses – Toner aid	**Presents an unreasonable risk of injury to health (consumers and bystanders)**

5.3 Detailed Risk Determinations by Condition of Use

5.3.1 Manufacture – Domestic manufacture

Section 6(b)(4)(A) unreasonable risk determination for domestic manufacture of TCE:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users (ONUs)).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-6)
- Autoimmunity:
  - Chronic inhalation MOEs 19.3 and 2.8 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-6)
- Cancer:
  - Inhalation: 1.3E-04 (high-end) with PPE (respirator APF 50).

- Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-6)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 13.9 (central tendency). (Table 4-6)
- Autoimmunity:
  - Chronic inhalation MOE 0.39 (central tendency). (Table 4-6)
- Cancer:
  - Inhalation: 7.5E-04 (central tendency). (Table 4-6)

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, while non-cancer risk estimates for acute inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA assessed inhalation exposures during manufacturing using monitoring data submitted by the Halogenated Solvents Industry Alliance (HSIA). EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

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 import of TCE:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-19)
- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-19)
- Cancer:
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-19)

Risk Considerations: For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. Acute and chronic inhalation exposures at the central tendency for cancer and non-cancer effects do not indicate risks in the absence of PPE. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA assessed inhalation exposures during import using the repackaging exposure scenario. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Manufacture	Import	Import

5.3.3 Processing – Processing as a reactant/intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants, foam blowing agents and solvents)

Section 6(b)(4)(A) unreasonable risk determination for processing of TCE as a reactant/intermediate:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-7)
- Autoimmunity:
  - Chronic inhalation MOEs 19.3 and 2.8 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-7)
- Cancer:
  - Inhalation: 1.3E-04 (high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-7)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 13.9 (central tendency). (Table 4-7)
- Autoimmunity:
  - Chronic inhalation MOE 0.39 (central tendency). (Table 4-7)
- Cancer:
  - Inhalation: 7.5E-04 (central tendency). (Table 4-7)

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, while non-cancer risk estimates for acute inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection (PF = 20). 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to processing TCE as a reactant. Therefore, EPA used monitoring data from the manufacture of TCE as surrogate data for the processing condition of use. EPA believes the handling and TCE concentrations for both conditions of use to be similar. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Processing	Processing as a Reactant/ Intermediate	Intermediate in industrial gas manufacturing (e.g., manufacture of fluorinated gases used as refrigerants, foam blowing agents and solvents)

5.3.4 Processing – Incorporation into formulation, mixture or reaction product – Solvents (for cleaning or degreasing); adhesives and sealant chemicals; solvents (which become part of product formulation or mixture) (e.g., lubricants and greases, paints and coatings, other uses)

Section 6(b)(4)(A) unreasonable risk determination for incorporation of TCE into formulation, mixture, reaction product, or articles:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-18)
- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-18)
- Cancer:
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-18)

Risk Considerations: For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. Acute and chronic inhalation exposures at the central tendency for cancer and non-cancer effects do not indicate risks in the absence of PPE. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to using TCE when formulating aerosol and non-aerosol products. Therefore, EPA used monitoring data from repackaging as a surrogate. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Processing	Processing - Incorporation into formulation, mixture or reaction product	• Solvents (for cleaning or degreasing) • Adhesives and sealant chemicals • Solvents (which become part of product formulation or mixture) (e.g., lubricants and greases, paints and coatings, other uses)

5.3.5 Processing – Incorporation into articles – Solvents (becomes an integral components of articles)

Section 6(b)(4)(A) unreasonable risk determination for incorporation of TCE into articles as solvents that become integral components of articles:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-18)
- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-18)
- Cancer:

- Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-18)

Risk Considerations: For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. Acute and chronic inhalation exposures at the central tendency for cancer and non-cancer effects do not indicate risks in the absence of PPE. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related using TCE when formulating aerosol and non-aerosol products. Therefore, EPA used monitoring data from repackaging as a surrogate. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Processing	Processing – incorporated into articles	Solvents (becomes an integral components of articles)

5.3.6 Processing – Repackaging – Solvents (for cleaning or degreasing)

Section 6(b)(4)(A) unreasonable risk determination for processing and repackaging of TCE as a solvent for cleaning or degreasing:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-19)

- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-19)
- Cancer:
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-19)

Risk Considerations: For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. Acute and chronic inhalation exposures at the central tendency for cancer and non-cancer effects do not indicate risks in the absence of PPE. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA assessed inhalation exposures during import using the repackaging exposure scenario. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Processing	Processing - repackaging	Solvents (for cleaning or degreasing)

5.3.7 Processing – Recycling

Section 6(b)(4)(A) unreasonable risk determination for recycling of TCE:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-27)

- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-27)
- Cancer:
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-27)

Risk Considerations: For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. Acute and chronic inhalation exposures at the central tendency for cancer and non-cancer effects do not indicate risks in the absence of PPE. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to using TCE when formulating aerosol and non-aerosol products. Therefore, EPA used monitoring data from repackaging as a surrogate for recycling. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Processing	Recycling	Recycling

5.3.8 Distribution in Commerce

Section 6(b)(4)(A) unreasonable risk determination for distribution of TCE:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Risk Considerations: A quantitative evaluation of the distribution of TCE 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 in commerce

5.3.9 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (open-top)

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for batch vapor degreasing (open-top):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 18.9 and 3.4 (central tendency and high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-8)
- Autoimmunity:
  - Chronic inhalation MOEs 0.52 and 9.3E-02 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-8)
- Cancer:
  - Inhalation: 5.5E-04 and 4.0E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-8)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 4.7 (central tendency). (Table 4-8)
- Autoimmunity:
  - Chronic inhalation MOE 0.13 (central tendency). (Table 4-8)
- Cancer:
  - Inhalation: 2.2E-03 (central tendency). (Table 4-8)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from NIOSH investigations at twelve sites using TCE as a degreasing solvent in OTVDs. Due to the large variety in shop types that may use TCE as a vapor degreasing solvent, it is unclear how representative these data are of a “typical” shop. Therefore, EPA supplemented the

identified monitoring data using the Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	Batch vapor degreaser (open-top)

5.3.10 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Batch vapor degreaser (closed-loop)

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for batch vapor degreasing (closed-loop):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-10)
- Autoimmunity:
  - Chronic inhalation MOEs 15.8 and 5.0 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-10)
- Cancer:

- Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-10)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 11.4 (central tendency). (Table 4-10)
- Autoimmunity:
  - Chronic inhalation MOE 0.32 (central tendency). (Table 4-10)
- Cancer (liver, kidney, NHL):
  - Inhalation: 9.1E-04 (central tendency). (Table 4-10)

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, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a European Chemical Safety report using TCE in closed degreasing operations. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	Batch vapor degreaser (closed-loop)

5.3.11 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (conveyorized)

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for in-line vapor degreasing (conveyorized):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 8.1 and 5.4 (central tendency and high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-11)
- Autoimmunity:
  - Chronic inhalation MOEs 0.22 and 0.15 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-11)
- Cancer (liver, kidney, NHL):
  - Inhalation: 1.3E-03 and 2.5E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-11)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 0.16 (central tendency). (Table 4-11)
- Autoimmunity:
  - Chronic inhalation MOE 4.5E-03 (central tendency). (Table 4-11)
- Cancer (liver, kidney, NHL):
  - Inhalation: 6.5E-02 (central tendency). (Table 4-11)

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, all risk estimates indicate risk even with expected respiratory and dermal protection (APF 50 and PF = 20). The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using TCE in conveyorized degreasing. Due to the large variety in shop types that may use TCE as a vapor degreasing solvent, it is unclear how representative these data are of a "typical" shop. Therefore, EPA supplemented the identified monitoring data using the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for this condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	In-line vapor degreaser (conveyorized)

5.3.12 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – In-line vapor degreaser (web cleaner)

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for in-line vapor degreaser (web cleaner):

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 18.5 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-13)
- Autoimmunity:
  - Chronic inhalation MOEs 1.2 and 0.51 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-13)
- Cancer:
  - Inhalation: 2.3E-04 and 5.8E-04 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-13)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 1.7 (central tendency). (Table 4-13)
- Autoimmunity:
  - Chronic inhalation MOE 4.6E-02 (central tendency). (Table 4-13)
- Cancer:
  - Inhalation: 5.9E-03 (central tendency). (Table 4-13)

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, all risk estimates indicate risk even with

expected respiratory and dermal protection (APF 50 and PF = 20). The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify any inhalation exposure monitoring data related to the use of TCE in web degreasing. Therefore, EPA assessed inhalation exposures during web degreasing using the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	In-line vapor degreaser (web cleaner)

5.3.13 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Cold cleaner

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for cold cleaning:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 4.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-14)
- Autoimmunity:
  - Chronic inhalation MOEs 2.2 and 0.13 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-14)

- Cancer:
  - Inhalation: 1.2E-04 and 2.3E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-14)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 2.8 (central tendency). (Table 4-14)
- Autoimmunity:
  - Chronic inhalation MOE 7.9E-02 (central tendency). (Table 4-14)
- Cancer:
  - Inhalation: 3.3E-03 (central tendency). (Table 4-14)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data for the Cold Cleaning condition of use. Therefore, EPA used the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model to estimate exposures to workers and ONUs. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	Cold cleaner

5.3.14 Industrial/Commercial Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner; mold release

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a solvent for aerosol spray degreaser/cleaner and for mold release:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.

- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 15.1 (high-end) with PPE (gloves PF 20). (Table 4-15)
- Autoimmunity:
  - Chronic inhalation MOEs 0.95 and 0.30 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.2 and 0.39 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-15)
- Cancer:
  - Inhalation: 2.9E-04 and 9.7E-04 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 7.6E-04 and 2.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-15)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.0 (high-end). (Table 4-15)
- Autoimmunity:
  - Chronic inhalation MOE 1.0 (central tendency). (Table 4-15)
- Cancer:
  - Inhalation: 2.6E-04 (central tendency). (Table 4-15)

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, all risk estimates indicate risk even with expected respiratory and dermal protection (APF=50 and PF=20). EPA estimated ONU exposures could be as high as worker exposures as a high-end estimate. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to the use of TCE in aerosol degreasers. Therefore, EPA estimated inhalation exposures using the Brake Servicing Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Solvents (for cleaning or degreasing)	• Aerosol spray degreaser/cleaner • Mold release

5.3.15 Industrial/Commercial Use – Lubricants and greases/lubricants and lubricant additives – Tap and die fluid

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a lubricant, grease/lubricant, and lubricant additive in tap and die fluid:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Unreasonable risk driver – ONUs:

- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 29.7 (high-end) with PPE (gloves PF 20). (Table 4-21)
- Autoimmunity:
  - Chronic inhalation MOE 27.5 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 2.3 and 0.76 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-21)
- Cancer:
  - Dermal: 3.9E-04 and 1.5E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-21)

Risk estimate – ONUs:

- Autoimmunity:
  - Chronic inhalation MOE 2.1 (central tendency). (Table 4-21)
- Cancer:
  - Inhalation: 1.3E-04 (central tendency). (Table 4-21)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE, with the exception of acute inhalation exposures at the central tendency. For workers, while non-cancer risk estimates for acute inhalation exposures and cancer risk

estimates from inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from OSHA facility inspections at two sites using TCE in metalworking fluids. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Lubricants and greases/lubricants and lubricant additives	Tap and die fluid

5.3.16 Industrial/Commercial Use – Lubricants and greases/lubricants and lubricant additives – Penetrating lubricant

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as penetrating lubricant:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 15.1 (high-end) with PPE (gloves PF 20). (Table 4-15)
- Autoimmunity:

- Chronic inhalation MOEs 0.95 and 0.30 (central tendency and high-end) with PPE (respirator APF 50).
- Chronic dermal MOEs 1.2 and 0.39 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-15)

- Cancer:
  - Inhalation: 2.9E-04 and 9.7E-04 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 7.6E-04 and 2.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-15)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.0 (high-end). (Table 4-15)
- Autoimmunity:
  - Chronic inhalation MOE 1.0 (central tendency). (Table 4-15)
- Cancer:
  - Inhalation: 2.6E-04 (central tendency). (Table 4-15)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. EPA estimated ONU exposures could be as high as worker exposures as a high-end estimate. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to the use of TCE in aerosol degreasers. Therefore, EPA estimated inhalation exposures using the Brake Servicing Near-field/Far-field Exposure Model. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Lubricants and greases/lubricants and lubricant additives	Penetrating lubricant

5.3.17 Industrial/Commercial Use – Adhesives and sealants – Solvent-based adhesives and sealants; tire repair cement/sealer; mirror edge sealant

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as an adhesive and sealant in solvent-based adhesives and sealants, tire repair cement/sealer, and mirror edge sealant:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.

- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 6.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOEs 25.2 and 8.4 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOEs 1.6 and 0.18 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 0.65 and 0.22 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-04 and 2.0E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 1.4E-03 and 5.3E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-23)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.5 (central tendency). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOE 0.15 (central tendency). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-03 (central tendency). (Table 4-23)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a NIOSH Health Hazard Evaluation report (Chrostek, 1981) using TCE in coating applications and from OSHA facility inspections (OSHA, 2017) at three sites using TCE in adhesives and coatings. The OSHA data also provided two data points where the worker job description was "foreman." EPA assumed this data is applicable to ONU exposure. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Adhesives and sealants	• Solvent-based adhesives and sealants • Tire repair cement/sealer • Mirror edge sealant

5.3.18 Industrial/Commercial Use – Functional fluids (closed systems) – Heat exchange fluid

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as a functional fluid (closed systems) for heat exchange fluid:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-26)
- Autoimmunity:
  - Chronic inhalation MOEs 19.3 and 2.8 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-26)
- Cancer:
  - Inhalation: 1.3E-04 (high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-26)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 13.9 (central tendency). (Table 4-26)

- Autoimmunity:
  - Chronic inhalation MOE 0.39 (central tendency). (Table 4-26)
- Cancer:
  - Inhalation: 7.5E-04 (central tendency). (Table 4-26)

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, while non-cancer risk estimates for acute inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to using TCE for other industrial uses. Therefore, EPA used monitoring data from loading/unloading TCE during manufacturing as a surrogate for this condition of use. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Functional fluids (closed systems)	Heat exchange fluid

5.3.19 Industrial/Commercial Use – Paints and coatings – Diluent in solvent-based paints and coatings

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in paints and coatings as a diluent in solvent-based paint and coatings:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**
- Does not present an unreasonable risk of injury to the environment (aquatic, sediment dwelling and terrestrial organisms).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 6.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOEs 25.2 and 8.4 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOEs 1.6 and 0.18 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 0.65 and 0.22 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-04 and 2.0E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 1.4E-03 and 5.3E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-23)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.5 (central tendency). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOE 0.15 (central tendency). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-03 (central tendency). (Table 4-23)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a NIOSH Health Hazard Evaluation report (Chrostek, 1981) using TCE in coating applications and from OSHA facility inspections (OSHA, 2017) at three sites using TCE in adhesives and coatings. The OSHA data also provided two data points where the worker job description was "foreman." EPA assumed this data is applicable to ONU exposure. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Paints and coatings	Diluent in solvent-based paints and coatings

5.3.20 Industrial/Commercial Use – Cleaning and furniture care products – Carpet cleaner; wipe cleaning

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in cleaning and furniture care products for carpet cleaning and wipe cleaning, and in laundry and dishwashing products as a spot remover:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 5.4 and 1.9 (central tendency and high-end) without respiratory PPE.
  - Acute dermal MOEs 22.7 and 7.6 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOEs 0.15 and 5.1E-02 (central tendency and high-end) without respiratory PPE.
  - Chronic dermal MOEs 0.56 and 0.17 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Cancer:
  - Inhalation: 1.8E-03 and 5.8E-03 (central tendency and high-end) without respiratory PPE.
  - Dermal: 1.6E-03 and 6.9E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-17)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (central tendency). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOE 0.29 (central tendency). (Table 4-17)
- Cancer:
  - Inhalation: 9.2E-04 (central tendency). (Table 4-17)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While workers are unlikely to wear respiratory protection for this condition of use, all other risk estimates indicate risk even with respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified minimal inhalation exposure monitoring data related to the spot cleaning using TCE. Therefore, EPA supplemented the identified monitoring data using the Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Cleaning and furniture care products	• Carpet cleaner • Wipe cleaning

5.3.21 Industrial/Commercial Use – Laundry and dishwashing products – Spot remover

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in laundry and dishwashing products as a spot remover:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 5.4 and 1.9 (central tendency and high-end) without respiratory PPE.

- Acute dermal MOEs 22.7 and 7.6 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)

- Autoimmunity:
  - Chronic inhalation MOEs 0.15 and 5.1E-02 (central tendency and high-end) without respiratory PPE.
  - Chronic dermal MOEs 0.56 and 0.17 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Cancer:
  - Inhalation: 1.8E-03 and 5.8E-03 (central tendency and high-end) without respiratory PPE.
  - Dermal: 1.6E-03 and 6.9E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-17)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (central tendency). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOE 0.29 (central tendency). (Table 4-17)
- Cancer:
  - Inhalation: 9.2E-04 (central tendency). (Table 4-17)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While workers are unlikely to wear respiratory protection for this condition of use, all other risk estimates indicate risk even with respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified minimal inhalation exposure monitoring data related to the spot cleaning using TCE. Therefore, EPA supplemented the identified monitoring data using the Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Laundry and dishwashing products	Spot remover

5.3.22 Industrial/Commercial Use – Arts, crafts and hobby materials – Fixatives and finishing spray coatings

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in arts, crafts and hobby materials as a fixative and finishing spray coating:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 6.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOEs 25.2 and 8.4 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOEs 1.6 and 0.18 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 0.65 and 0.22 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-04 and 2.0E-03 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 1.4E-03 and 5.3E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-23)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.5 (central tendency). (Table 4-23)
- Autoimmunity:
  - Chronic inhalation MOE 0.15 (central tendency). (Table 4-23)
- Cancer:
  - Inhalation: 1.9E-03 (central tendency). (Table 4-23)

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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a NIOSH Health Hazard Evaluation report (Chrostek, 1981) using TCE in coating applications and from OSHA facility inspections (OSHA, 2017) at three sites using TCE in adhesives

and coatings. The OSHA data also provided two data points where the worker job description was "foreman." EPA assumed this data is applicable to ONU exposure. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Arts, crafts and hobby materials	Spot remover

5.3.23 Industrial/Commercial Use – Corrosion inhibitors and anti-scaling agents – Corrosion inhibitors and anti-scaling agents

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as corrosion inhibitor, and anti-scaling agent:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 13.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-24)
- Autoimmunity:
  - Chronic inhalation MOEs 1.1 and 0.38 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-24)
- Cancer:
  - Inhalation: 2.5E-04 and 9.9E-04 (central tendency and high-end) with PPE (respirator APF 50).

- Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-24)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 2.7 (central tendency). (Table 4-24)
- Autoimmunity:
  - Chronic inhalation MOE 7.3E-02 (central tendency). (Table 4-24)
- Cancer:
  - Inhalation: 3.9E-03 (central tendency). (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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a European Commission (EC) Technical Report (European Commission, 2014, 3970806). The data was supplied to the EC as supporting documentation in an application for continued use of TCE under the REACH Regulation. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Corrosion inhibitors and anti-scaling agents	Corrosion inhibitors and anti-scaling agents

5.3.24 Industrial/Commercial Use – Processing aids – Process solvent used in battery manufacture; process solvent used in polymer fiber spinning, fluoroelastomer manufacture, and Alcantara manufacture; extraction solvent used in caprolactam manufacture; precipitant used in beta-cyclodextrin manufacture

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in processing aids as a process solvent used in battery manufacture, polymer fiber spinning, fluoroelastomer manufacture, and Alcantara manufacture, as an extraction solvent used in caprolactam manufacture, and as a precipitant used in beta-cyclodextrin manufacture:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.

- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 13.6 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-24)
- Autoimmunity:
  - Chronic inhalation MOEs 1.1 and 0.38 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-24)
- Cancer:
  - Inhalation: 2.5E-04 and 9.9E-04 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-24)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 2.7 (central tendency). (Table 4-24)
- Autoimmunity:
  - Chronic inhalation MOE 7.3E-02 (central tendency). (Table 4-24)
- Cancer:
  - Inhalation: 3.9E-03 (central tendency). (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, all risk estimates indicate risk even with expected respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a European Commission (EC) Technical Report (European Commission, 2014, 3970806). The data was supplied to the EC as supporting documentation in an application for continued use of TCE under the REACH Regulation. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Processing aids	• Process solvent used in battery manufacture

Life Cycle Stage	Category	Subcategory
		• Process solvent used in polymer fiber spinning, fluoroelastomer manufacture, and Alcantara manufacture • Extraction solvent used in caprolactam manufacture • Precipitant used in beta-cyclodextrin manufacture

5.3.25 Industrial/Commercial Use – Ink, toner, and colorant products – Toner aid

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE as an ink, toner, and colorant product as a toner aid:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 2.5 (high-end) without respiratory PPE.
  - Acute dermal MOE 21.6 (high-end) with PPE (gloves PF 20). (Table 4-25)
- Autoimmunity:
  - Chronic inhalation MOEs 1.7 and 6.9E-02 (central tendency and high-end) without respiratory PPE.
  - Chronic dermal MOEs 1.7 and 0.55 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-25)
- Cancer:
  - Inhalation: 1.7E-04 and 5.4E-03 (central tendency and high-end) without respiratory PPE.
  - Dermal: 5.3E-04 and 2.1E-03 (central tendency and high-end) with PPE (gloves PF=10). (Table 4-25)

Risk estimate – ONUs:

- Autoimmunity:
  - Chronic inhalation MOE 1.7 (central tendency). (Table 4-25)
- Cancer:
  - Inhalation: 1.7E-04 (central tendency). (Table 4-25)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While workers are unlikely to wear respiratory protection for this condition of use, all other risk estimates indicate risk even with respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified inhalation exposure monitoring data from a European Commission (EC) Technical Report (European Commission, 2014, 3970806). The data was supplied to the EC as supporting documentation in an application for continued use of TCE under the REACH Regulation. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Ink, toner and colorant products	Toner aid

5.3.26 Industrial/Commercial Use – Automotive care products – Brake and parts cleaners

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE for automotive care products as a brake and part cleaner:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (high-end) with PPE (respirator APF 50).
  - Acute dermal MOE 15.1 (high-end) with PPE (gloves PF 20). (Table 4-15)
- Autoimmunity:
  - Chronic inhalation MOEs 0.95 and 0.30 (central tendency and high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.2 and 0.39 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-15)
- Cancer:
  - Inhalation: 2.9E-04 and 9.7E-04 (central tendency and high-end) with PPE (respirator APF 50).
  - Dermal: 7.6E-04 and 2.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-15)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 5.0 (high-end). (Table 4-15)
- Autoimmunity:
  - Chronic inhalation MOE 1.0 (central tendency). (Table 4-15)
- Cancer:
  - Inhalation: 2.6E-04 (central tendency). (Table 4-15)

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, all risk estimates indicate risk even with expected respiratory and dermal protection (APF=50 and PF=20). EPA estimated ONU exposures could be as high as worker exposures as a high-end estimate. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to the use of TCE in aerosol degreasers. Therefore, EPA estimated inhalation exposures using the Brake Servicing Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Automotive care products	Brake and parts cleaners

5.3.27 Industrial/Commercial Use – Apparel and footwear care products – Shoe polish

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in apparel and footwear care products as a shoe polish:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 5.4 and 1.9 (central tendency and high-end) without respiratory PPE.
  - Acute dermal MOEs 22.7 and 7.6 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOEs 0.15 and 5.1E-02 (central tendency and high-end) without respiratory PPE.
  - Chronic dermal MOEs 0.56 and 0.17 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Cancer:
  - Inhalation: 1.8E-03 and 5.8E-03 (central tendency and high-end) without respiratory PPE.
  - Dermal: 1.6E-03 and 6.9E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-17)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (central tendency). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOE 0.29 (central tendency). (Table 4-17)
- Cancer:
  - Inhalation: 9.2E-04 (central tendency). (Table 4-17)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While workers are unlikely to wear respiratory protection for this condition of use, all other risk estimates indicate risk even with respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified minimal inhalation exposure monitoring data related to the spot cleaning using TCE. Therefore, EPA supplemented the identified monitoring data using the Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field

approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Apparel and footwear care products	Shoe polish

5.3.28 Industrial/Commercial Use – Hoof polishes; gun scrubber; pepper spray; other miscellaneous industrial and commercial uses

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE in other commercial uses for hoof polishes, gun scrubber, pepper spray, and other miscellaneous industrial and commercial uses:

- **Presents an unreasonable risk of injury to health (workers and occupational non-users).**

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute inhalation and dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic inhalation and dermal exposures.

Unreasonable risk driver – ONUs:

- Immunosuppression resulting from acute inhalation exposures.
- Autoimmunity resulting from chronic inhalation exposures.
- Cancer resulting from chronic inhalation exposures.

Driver benchmarks – workers and ONUs:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute inhalation MOEs 5.4 and 1.9 (central tendency and high-end) without respiratory PPE.
  - Acute dermal MOEs 22.7 and 7.6 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOEs 0.15 and 5.1E-02 (central tendency and high-end) without respiratory PPE.

- Chronic dermal MOEs 0.56 and 0.17 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-17)
- Cancer:
  - Inhalation: 1.8E-03 and 5.8E-03 (central tendency and high-end) without respiratory PPE.
  - Dermal: 1.6E-03 and 6.9E-03 (central tendency and high-end) with PPE (gloves PF = 10). (Table 4-17)

Risk estimate – ONUs:

- Immunosuppression:
  - Acute inhalation MOE 10.9 (central tendency). (Table 4-17)
- Autoimmunity:
  - Chronic inhalation MOE 0.29 (central tendency). (Table 4-17)
- Cancer:
  - Inhalation: 9.2E-04 (central tendency). (Table 4-17)

Risk Considerations: For workers and ONUs, all pathways of occupational exposure for this condition of use indicate risk in the absence of PPE. While workers are unlikely to wear respiratory protection for this condition of use, all other risk estimates indicate risk even with respiratory and dermal protection. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA identified minimal inhalation exposure monitoring data related to the spot cleaning using TCE. Therefore, EPA supplemented the identified monitoring data using the Near-field/Far-field Exposure Model. EPA's inhalation exposure modeling is based on a near-field/far-field approach, where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field. These estimates were used for determining worker and ONU risks. For workers, EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Industrial/commercial use	Other commercial uses	• Hoof polishes • Gun scrubber • Pepper spray • Other miscellaneous industrial and commercial uses

5.3.29 Disposal

Section 6(b)(4)(A) unreasonable risk determination for industrial/commercial use of TCE for disposal:

- **Presents an unreasonable risk of injury to health (workers).**
- Does not present an unreasonable risk of injury to health (occupational non-users).

Unreasonable risk driver – workers:

- Immunosuppression resulting from acute dermal exposures.
- Autoimmunity resulting from chronic inhalation and dermal exposures.
- Cancer resulting from chronic dermal exposures.

Driver benchmarks – workers:

- Immunosuppression: Benchmark MOE = 30.
- Autoimmunity: Benchmark MOE = 30.
- Cancer (liver, kidney, NHL): Benchmark = $1x10^{-4}$.

Risk estimate - workers:

- Immunosuppression:
  - Acute dermal MOE 23.8 (high-end) with PPE (gloves PF 20). (Table 4-27)
- Autoimmunity:
  - Chronic inhalation MOE 6.3 (high-end) with PPE (respirator APF 50).
  - Chronic dermal MOEs 1.8 and 0.61 (central tendency and high-end) with PPE (gloves PF 20). (Table 4-27)
- Cancer:
  - Dermal: 4.9E-04 and 1.9E-03 (central tendency and high-end) with PPE (gloves PF = 20). (Table 4-27)

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, while non-cancer risk estimates for acute inhalation exposures and cancer risk estimates for inhalation exposures do not indicate risks with expected respiratory protection (APF 50), all other risk estimates indicate risk even with expected respiratory and dermal protection. 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. The high volatility of TCE and potentially severe effects from short term exposure are factors when weighing uncertainties. EPA did not identify inhalation exposure monitoring data related to using TCE for other industrial uses. Therefore, EPA used monitoring data from loading/unloading TCE during manufacturing as a surrogate for this condition of use. EPA estimated dermal exposures using the Dermal Exposure to Volatile Liquids Model because dermal exposure data was not reasonably available for the condition of use.

Life Cycle Stage	Category	Subcategory
Disposal	Disposal	• Industrial pre-treatment • Industrial wastewater treatment • Publicly owned treatment works (POTW)

5.3.30 Consumer Use – Solvents (for cleaning or degreasing) – Brake and parts cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for brake and parts cleaners:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.21 (moderate intensity user).
  - Acute dermal MOE 0.48 (moderate intensity user). (Table 4-28)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 0.94 (moderate intensity user). (Table 4-28)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Brake and Parts cleaner

5.3.31 Consumer Use – Solvents (for cleaning or degreasing) – Aerosol electronic degreaser/cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for aerosol electronic degreaser/cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.2 (moderate intensity user). (Table 4-29)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 7.1 (moderate intensity user). (Table 4-29)

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 severity of 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 TCE. 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	Solvents (for cleaning or degreasing)	Aerosol electronic degreaser/cleaner

5.3.32 Consumer Use – Solvents (for cleaning or degreasing) – Liquid electronic degreaser/cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for liquid electronic degreaser/cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:

- Acute inhalation MOE 0.79 (moderate intensity user).
- Acute dermal MOE 9.5E-01 (moderate intensity user). (Table 4-30)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 4.8 (moderate intensity user). (Table 4-30)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Liquid electronic degreaser/cleaner

5.3.33 Consumer Use – Solvents (for cleaning or degreasing) – Aerosol spray degreaser/cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for aerosol spray degreaser/cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 4.6E-02 (moderate intensity user).
  - Acute dermal MOE 0.31 (moderate intensity user). (Table 4-31)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 0.21 (moderate intensity user). (Table 4-31)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Aerosol spray degreaser/cleaner

5.3.34 Consumer Use – Solvents (for cleaning or degreasing) – Liquid degreaser/cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for liquid degreaser/cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.12 (moderate intensity user).
  - Acute dermal MOE 0.13 (moderate intensity user). (Table 4-32)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 0.70 (moderate intensity user). (Table 4-32)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Liquid degreaser/cleaner

5.3.35 Consumer Use – Solvents (for cleaning or degreasing) – Aerosol gun scrubber

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for aerosol gun scrubber:

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Driver benchmarks – consumers:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 24 (moderate intensity user).
  - Acute dermal MOE 0.32 (moderate intensity user). (Table 4-33)

Risk Considerations: All pathways of consumer exposure for this condition of use indicate risk. Consumer risk determinations reflect the severity of 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 do not indicate risk. Because bystanders are not expected to be dermally exposed to TCE, 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	Solvents (for cleaning or degreasing)	Aerosol gun scrubber

5.3.36 Consumer Use – Solvents (for cleaning or degreasing) – Liquid gun scrubber

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for liquid gun scrubber:

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Driver benchmarks – consumers:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 28 (moderate intensity user).
  - Acute dermal MOE 0.14 (moderate intensity user). (Table 4-34)

Risk Considerations: All pathways of consumer exposure for this condition of use indicate risk. Consumer risk determinations reflect the severity of 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 do not indicate risk. Because bystanders are not expected to be dermally exposed to TCE, 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	Solvents (for cleaning or degreasing)	Liquid gun scrubber

5.3.37 Consumer Use – Solvents (for cleaning or degreasing) – Mold release

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for mold release:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.1 (moderate intensity user). (Table 4-35)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 6.4 (moderate intensity user). (Table 4-35)

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 severity of 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 TCE. 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	Solvents (for cleaning or degreasing)	Mold release

5.3.38 Consumer Use – Solvents (for cleaning or degreasing) – Aerosol tire cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for aerosol tire cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.46 (moderate intensity user).
  - Acute dermal MOE 0.70 (moderate intensity user). (Table 4-36)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 2.0 (moderate intensity user). (Table 4-36)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Aerosol tire cleaner

5.3.39 Consumer Use – Solvents (for cleaning or degreasing) – Liquid tire cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a solvent for liquid tire cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.21 (moderate intensity user).
  - Acute dermal MOE 0.12 (moderate intensity user). (Table 4-37)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 0.92 (moderate intensity user). (Table 4-37)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Solvents (for cleaning or degreasing)	Liquid tire cleaner

5.3.40 Consumer Use – Lubricants and greases – Tap and die fluid

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a lubricant and grease in tap and die fluid:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.2 (moderate intensity user). (Table 4-38)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 7.1 (moderate intensity user). (Table 4-38)

Risk Considerations: Consumer and bystander risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE. For the consumer exposure for scenario 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	Lubricants and greases	Tap and die fluid

5.3.41 Consumer Use – Lubricants and greases – Penetrating lubricant

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE as a penetrating lubricant:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 2.7 (moderate intensity user). (Table 4-39)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 16 (moderate intensity user). (Table 4-39)

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 severity of 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 TCE. 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	Lubricants and greases	Penetrating lubricant

5.3.42 Consumer Use – Adhesives and sealants – Solvent-based adhesive and sealant

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in adhesives and sealants as solvent-based adhesive and sealant:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.8 (moderate intensity user). (Table 4-40)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 20 (moderate intensity user). (Table 4-40)

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 severity of 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 TCE. 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	Adhesives and sealants	Solvent-based adhesive and sealant

5.3.43 Consumer Use – Adhesives and sealants – Mirror edge sealant

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in adhesives and sealants as mirror edge sealant:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.7 (moderate intensity user). (Table 4-41)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 10 (moderate intensity user). (Table 4-41)

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 severity of 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 TCE. 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	Adhesives and sealants	Mirror edge sealant

5.3.44 Consumer Use – Adhesives and sealants – Tire repair cement/sealer

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in adhesives and sealants as tire repair cement/sealer:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 2.9 (moderate intensity user). (Table 4-42)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 13 (moderate intensity user). (Table 4-42)

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 severity of 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 TCE. 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	Adhesives and sealants	Tire repair cement/sealer

5.3.45 Consumer Use – Cleaning and furniture care products – Carpet cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in cleaning and furniture care products as carpet cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.29 (moderate intensity user).
  - Acute dermal MOE 0.35 (moderate intensity user). (Table 4-43)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 1.7 (moderate intensity user). (Table 4-43)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Carpet cleaner

5.3.46 Consumer Use – Cleaning and furniture care products – Aerosol spot remover

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in cleaning and furniture care products as aerosol spot remover:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.47 (moderate intensity user).
  - Acute dermal MOE 3.0 (moderate intensity user). (Table 4-44)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 5.6 (moderate intensity user). (Table 4-44)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Aerosol spot remover

5.3.47 Consumer Use – Cleaning and furniture care products – Liquid spot remover

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in cleaning and furniture care products as liquid spot remover:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.39 (moderate intensity user).
  - Acute dermal MOE 0.51 (moderate intensity user). (Table 4-45)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 2.4 (moderate intensity user). (Table 4-45)

Risk Considerations: All pathways of consumer and bystander exposure for this condition of use indicate risk. Consumer and bystander risk determinations reflect the severity of 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 TCE, 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	Liquid spot remover

5.3.48 Consumer Use – Arts, crafts, and hobby materials – Fixatives and finishing spray coatings

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in arts, crafts, and hobby materials as fixative and finishing spray coating:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.2 (moderate intensity user). (Table 4-46)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 7.6 (moderate intensity user). (Table 4-46)

Risk Considerations: Consumer and bystander risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE. 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	Arts, crafts, and hobby materials	Fixatives and finishing spray coatings

5.3.49 Consumer Use – Apparel and footwear care products – Shoe polish

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in apparel and footwear care products in shoe polish:

- **Presents an unreasonable risk of injury to health (consumers).**

- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation and dermal exposures.

Driver benchmarks – consumers:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 5.4 (moderate intensity user).
  - Acute dermal MOE 5.5 (moderate intensity user). (Table 4-47)

Risk Considerations: All pathways of consumer exposure for this condition of use indicate risk. Consumer risk determinations reflect the severity of 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 do not indicate risk. Because bystanders are not expected to be dermally exposed to TCE, 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	Apparel and footwear care products	Shoe polish

5.3.50 Consumer Use – Other consumer uses – Fabric spray

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in fabric spray:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.43 (moderate intensity user). (Table 4-48)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 5.1 (moderate intensity user). (Table 4-48)

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Risk Considerations: Consumer and bystander risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE. 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 consumer uses	Fabric spray

5.3.51 Consumer Use – Other consumer uses – Film cleaner

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in film cleaner:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 0.18 (moderate intensity user). (Table 4-49)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 1.1 (moderate intensity user). (Table 4-49)

Risk Considerations: Consumer and bystander risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use

scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE. 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 consumer uses	Film cleaner

5.3.52 Consumer Use – Other consumer uses – Hoof polish

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in hoof polish:

- **Presents an unreasonable risk of injury to health (consumers).**
- Does not present an unreasonable risk of injury to health (bystanders).

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 8.0 (moderate intensity user). (Table 4-50)

Risk Considerations: Consumer risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE. 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 consumer uses	Hoof polish

5.3.53 Consumer Use – Other consumer uses – Pepper spray

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in pepper spray:

- Does not present an unreasonable risk of injury to health (consumers and bystanders).

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 98 (moderate intensity user). (Table 4-51)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 98 (moderate intensity user). (Table 4-51)

Risk Considerations: Consumer risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation do not indicate risk. For bystanders, MOEs are expected to be equivalent to consumers. 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 TCE.

Life Cycle Stage	Category	Subcategory
Consumer use	Other consumer uses	Pepper spray

5.3.54 Consumer Use – Other consumer uses – Toner aid

Section 6(b)(4)(A) unreasonable risk determination for consumer use of TCE in toner aid:

- **Presents an unreasonable risk of injury to health (consumers and bystanders).**

Unreasonable risk driver – consumers:

- Immunosuppression resulting from acute inhalation exposures.

Unreasonable risk driver – bystanders:

- Immunosuppression resulting from acute inhalation exposures.

Driver benchmarks – consumers and bystanders:

- Immunosuppression: Benchmark MOE = 10.

Risk estimate – consumers:

- Immunosuppression:
  - Acute inhalation MOE 1.3 (moderate intensity user). (Table 4-52)

Risk estimate – bystanders:

- Immunosuppression:
  - Acute inhalation MOE 8.0 (moderate intensity user). (Table 4-52)

Risk Considerations: Consumer and bystander risk determinations reflect the severity of the effects associated with acute exposures. Risk estimates for consumer users at the medium intensity use scenarios of acute inhalation indicate risk. For bystanders the risk estimates for the medium intensity use scenario of acute inhalation 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 TCE.

Life Cycle Stage	Category	Subcategory
Consumer use	Other consumer uses	Toner Aid

REFERENCES

Abernethy, S; Bobra, AM; Shiu, WY; Wells, PG; Mackay, D. (1986). Acute lethal toxicity of hydrocarbons and chlorinated hydrocarbons to two planktonic crustaceans the key role of organism-water partitioning. Aquat Toxicol AMST: 163-174.

Adgate, JL; Church, TR; Ryan, AD; Ramachandran, G; Fredrickson, AL; Stock, TH; Morandi, MT; Sexton, K. (2004). Outdoor, indoor, and personal exposure to VOCs in children. Environ Health Perspect 112: 1386-1392. http://dx.doi.org/10.1289/ehp.7107

AIHA. (2009). Mathematical models for estimating occupational exposure to chemicals. In CB Keil; CE Simmons; TR Anthony (Eds.), (2nd ed.). Fairfax, VA: AIHA Press.

Alanee, S; Clemons, J; Zahnd, W; Sadowski, D; Dynda, D. (2015). Trichloroethylene Is Associated with Kidney Cancer Mortality: A Population-based Analysis. Anticancer Res 35: 4009-4013.

Alexander, HC; McCarty, WM; Bartlett, EA. (1978). Toxicity of perchloroethylene, trichloroethylene, 1,1,1-trichloroethane, and methylene chloride to fathead minnows. Bull Environ Contam Toxicol 20: 344-352. http://dx.doi.org/10.1007/BF01683531

Ando, T; Otsuka, S; Nishiyama, M; Senoo, K; Watanabe, MM; Matsumoto, S. (2003). Toxic Effects of Dichloromethane and Trichloroethylene on the Growth of Planktonic Green Algae, Chlorella vulgaris NIES227, Selenastrum capricornutum NIES35, and Volvulina steinii NIES545. 18: 43-46.

Anttila, A; Pukkala, E; Sallmen, M; Hernberg, S; Hemminki, K. (1995). Cancer incidence among Finnish workers exposed to halogenated hydrocarbons. J Occup Environ Med 37: 797-806.

Arito, H; Takahashi, M; Ishikawa, T. (1994). Effect of subchronic inhalation exposure to low-level trichloroethylene on heart rate and wakefulness-sleep in freely moving rats. Sangyo Igaku 36: 1-8.

ATSDR. (2019). Toxicological Profile for Trichloroethylene: CAS # 79-01-6. Atlanta, GA. https://www.atsdr.cdc.gov/ToxProfiles/tp19.pdf

Axelson, O; Seldén, A; Andersson, K; Hogstedt, C. (1994). Updated and expanded Swedish cohort study on trichloroethylene and cancer risk. J Occup Med 36: 556-562.

Bahr, DE; Aldrich, TE; Seidu, D; Brion, GM; Tollerud, DJ; Plant, PGD. (2011). OCCUPATIONAL EXPOSURE TO TRICHLOROETHYLENE AND CANCER RISK FOR WORKERS AT THE PADUCAH GASEOUS DIFFUSION PLANT. Int J Occup Med Environ Health 24: 67-77. http://dx.doi.org/10.2478/s13382-011-0007-1

Baldwin, PE; Maynard, AD. (1998a). A survey of wind speed in indoor workplaces. Ann Occup Hyg 42: 303-313. http://dx.doi.org/10.1016/S0003-4878(98)00031-3

Banerjee, S; Yalkowsky, SH; Valvani, SC. (1980). Water solubility and octanol-water partition-coefficients of organics - limitations of the solubility-partition coefficient correlation. Environ Sci Technol 14: 1227-1229. http://dx.doi.org/10.1021/es60170a013

Barrows, ME; Petrocelli, SR; Macek, KJ; Carroll, JJ. (1980). Bioconcentration and elimination of selected water pollutants by bluegill sunfish (Lepomis macrochirus). In R Haque (Ed.), Dynamics, exposure and hazard assessment of toxic chemicals (pp. 379-392). Ann Arbor, MI: Ann Arbor Science.

Bassig, BA; Zhang, L; Vermeulen, R; Tang, X; Li, G; Hu, W, ei; Guo, W; Purdue, MP; Yin, S; Rappaport, SM; Shen, M, in; Ji, Z; Qiu, C; Ge, Y; Hosgood, HD; Reiss, B; Wu, B; Xie, Y; Li, L; Yue, F, ei; Freeman, LEB; Blair, A; Hayes, RB; Huang, H; Smith, MT; Rothman, N; Lan, Q. (2016). Comparison of hematological alterations and markers of B-cell activation in workers exposed to benzene, formaldehyde and trichloroethylene. Carcinogenesis 37: 692-700. http://dx.doi.org/10.1093/carcin/bgw053

Bertilsson, J; Petersson, U; Fredriksson, PJ; Magnusson, M; Fransson, PA. (2017). Use of pepper spray in policing: retrospective study of situational characteristics and implications for violent situations. 18: 391-406. http://dx.doi.org/10.1080/15614263.2017.1288119

Blossom, SJ; Cooney, CA; Melnyk, SB; Rau, JL; Swearingen, CJ; Wessinger, WD. (2013). Metabolic changes and DNA hypomethylation in cerebellum are associated with behavioral alterations in mice exposed to trichloroethylene postnatally. Toxicol Appl Pharmacol 269: 263-269. http://dx.doi.org/10.1016/j.taap.2013.03.025

Boice, JD, Jr; Marano, D; Fryzek, J; Sadler, C; Mclaughlin, JK. (1999). Mortality among aircraft manufacturing workers. Occup Environ Med 56: 581-597. http://dx.doi.org/10.1136/oem.56.9.581

Boice, JD; Marano, DE; Cohen, SS; Mumma, MT; Blot, WJ; Brill, AB; Fryzek, JP; Henderson, BE; Mclaughlin, JK. (2006). Mortality among Rocketdyne workers who tested rocket engines, 1948-1999. J Occup Environ Med 48: 1070-1092. http://dx.doi.org/10.1097/01.jom.0000240661.33413.b5

Bouwer, EJ; McCarty, PL. (1983). Transformations of 1- and 2-carbon halogenated aliphatic organic compounds under methanogenic conditions. Appl Environ Microbiol 45: 1286-1294.

Bove, FJ. (1996). Public drinking water contamination and birthweight, prematurity, fetal deaths, and birth defects. Toxicol Ind Health 12: 255-266.

Bove, FJ; Fulcomer, MC; Klotz, JB; Esmart, J; Dufficy, EM; Savrin, JE. (1995). Public drinking water contamination and birth outcomes. Am J Epidemiol 141: 850-862.

Bove, FJ; Ruckart, PZ; Maslia, M; Larson, TC. (2014a). Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study. Environ Health 13: 10. http://dx.doi.org/10.1186/1476-069X-13-10

Bove, FJ; Ruckart, PZ; Maslia, M; Larson, TC. (2014b). Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. Environ Health 13: 68. http://dx.doi.org/10.1186/1476-069X-13-68

Boverhof, DR; Krieger, SM; Hotchkiss, J; Stebbins, KE; Thomas, J; Woolhiser, MR. (2013). Assessment of the immunotoxic potential of trichloroethylene and perchloroethylene in rats following inhalation exposure. J Immunotoxicol 10: 311-320. http://dx.doi.org/10.3109/1547691X.2012.735275

Boyer, A; Finch, W; Runyan, R. (2000). Trichloroethylene inhibits development of embryonic heart valve precursors in vitro. Toxicol Sci 53: 109-117. http://dx.doi.org/10.1093/toxsci/53.1.109

Brack, W; Rottler, H. (1994). Toxicity testing of highly volatile chemicals with green algae: A new assay. Environ Sci Pollut Res Int 1: 223-228.

Brender, JD; Shinde, MU; Zhan, FB; Gong, X; Langlois, PH. (2014). Maternal residential proximity to chlorinated solvent emissions and birth defects in offspring: a case-control study. Environ Health 13: 96. http://dx.doi.org/10.1186/1476-069X-13-96

Bridges, J; Sauer, UG; Buesen, R; Deferme, L; Tollefsen, KE; Tralau, T; van Ravenzwaay, B; Poole, A; Pemberton, M. (2017). Framework for the quantitative weight-of-evidence analysis of 'omics data for regulatory purposes. Regul Toxicol Pharmacol 91: S46-S60. http://dx.doi.org/10.1016/j.yrtph.2017.10.010

Bridges, J; Solomon, KR. (2016). Quantitative weight-of-evidence analysis of the persistence, bioaccumulation, toxicity, and potential for long-range transport of the cyclic volatile methyl siloxanes [Review]. J Toxicol Environ Health B Crit Rev 19: 345-379. http://dx.doi.org/10.1080/10937404.2016.1200505

Broderius, SJ; Kahl, MD; Elonen, GE; Hammermeister, DE; Hoglund, MD. (2005). A Comparison of the Lethal and Sublethal Toxicity of Organic Chemical Mixtures to the Fathead Minnow (Pimphales promelas). 24: 3117-3127.

Brüning, T; Pesch, B; Wiesenhütter, B; Rabstein, S; Lammert, M; Baumüller, A; Bolt, H. (2003). Renal cell cancer risk and occupational exposure to trichloroethylene: Results of a consecutive case-control study in Arnsberg, Germany. Am J Ind Med 43: 274-285. http://dx.doi.org/10.1002/ajim.10185

Buben, JA; O'Flaherty, EJ. (1985). Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. Toxicol Appl Pharmacol 78: 105-122.

Buccafusco, RJ; Ells, SJ; LeBlanc, GA. (1981). Acute toxicity of priority pollutants to bluegill (Lepomis macrochirus). Bull Environ Contam Toxicol 26: 446-452. http://dx.doi.org/10.1007/BF01622118

Buhagen, M; Grønskag, A; Ragde, SF; Hilt, B. (2016). Association between kidney cancer and occupational exposure to trichloroethylene. J Occup Environ Med 58: 957-959. http://dx.doi.org/10.1097/JOM.0000000000000838

Burnham, KP; Anderson, DR. (2002). Model selection and multimodel inference: a practical information-theoretic approach (2nd ed.). New York: Springer. http://www.springer.com/statistics/statistical+theory+and+methods/book/978-0-387-95364-9

Caldwell, PT; Thorne, PA; Johnson, PD; Boitano, S; Runyan, RB; Selmin, O. (2008). Trichloroethylene disrupts cardiac gene expression and calcium homeostasis in rat myocytes. Toxicol Sci 104: 135-143. http://dx.doi.org/10.1093/toxsci/kfn078

CalEPA. (2009). Public health goals for chemicals in drinking water: Trichloroethylene. Sacramento, CA. https://oehha.ca.gov/media/downloads/water/chemicals/phg/tcephg070909_0.pdf

CARB. (2000). Initial statement of reasons for the proposed airborne toxic control measure for emissions of chlorinated toxic air contaminants from automotive maintenance and repair activities. California Air Resources Board.

CARB. (2006). California Dry Cleaning Industry Technical Assessment Report. Stationary Source Division, Emissions Assessment Branch. https://www.arb.ca.gov/toxics/dryclean/finaldrycleantechreport.pdf

Carney, EW; Thorsrud, BA; Dugard, PH; Zablotny, CL. (2006). Developmental toxicity studies in Crl:CD (SD) rats following inhalation exposure to trichloroethylene and perchloroethylene. Birth Defects Res B Dev Reprod Toxicol 77: 405-412. http://dx.doi.org/10.1002/bdrb.20091

Chan, CC; Vainer, L; Martin, JW; Williams, DT. (1990). Determination of organic contaminants in residential indoor air using an adsorption-thermal desorption technique. J Air Waste Manag Assoc 40: 62-67.

Charbotel, B; Fevotte, J; Hours, M; Martin, JL; Bergeret, A. (2006). Case-control study on renal cell cancer and occupational exposure to trichloroethylene. Part II: Epidemiological aspects. Ann Occup Hyg 50: 777-787. http://dx.doi.org/10.1093/annhyg/mel039

Charles River Laboratories. (2019). An oral (drinking water) study of the effects of trichloroethylene (TCE) on fetal heart development in Sprague Dawley rats: Laboratory Project ID 00459506. (EPA-HQ-OPPT-2016-0737-0120).

Chia, SE; Ong, CN; Tsakok, MF; Ho, A. (1996). Semen parameters in workers exposed to trichloroethylene. Reprod Toxicol 10: 295-299. http://dx.doi.org/10.1016/0890-6238(96)00058-5

Chin, JY; Godwin, C; Parker, E; Robins, T; Lewis, T; Harbin, P; Batterman, S. (2014). Levels and sources of volatile organic compounds in homes of children with asthma. Indoor Air 24: 403-415. http://dx.doi.org/10.1111/ina.12086

Chinn, KSK. (1981). A simple model for predicting chemical agent evaporation. Alexandria, VA: U.S. Department of Defense, Defense Technical Information Center, Cameron Station. http://www.epa.gov/opptintr/exposure/presentations/efast/chinn_1981_a_simple_method_for_predicting.pdf

Chiu, WA; Micallef, S; Monster, AC; Bois, FY. (2007). Toxicokinetics of inhaled trichloroethylene and tetrachloroethylene in humans at 1 ppm: Empirical results and comparisons with previous studies. Toxicol Sci 95: 23-36. http://dx.doi.org/10.1093/toxsci/kfl129

Christensen, KY; Vizcaya, D; Richardson, H; Lavoué, J; Aronson, K; Siemiatycki, J. (2013). Risk of selected cancers due to occupational exposure to chlorinated solvents in a case-control study in Montreal. J Occup Environ Med 55: 198-208. http://dx.doi.org/10.1097/JOM.0b013e3182728eab

Clayton, CA; Pellizzari, ED; Whitmore, RW; Perritt, RL; Quackenboss, JJ. (1999). National Human Exposure Assessment Survey (NHEXAS): Distributions and associations of lead, arsenic, and volatile organic compounds in EPA Region 5. J Expo Anal Environ Epidemiol 9: 381-392. http://dx.doi.org/10.1038/sj.jea.7500055

Cocco, P; T'Mannetje, A; Fadda, D; Melis, M; Becker, N; de Sanjosé, S; Foretova, L; Mareckova, J; Staines, A; Kleefeld, S; Maynadié, M; Nieters, A; Brennan, P; Boffetta, P. (2010). Occupational exposure to solvents and risk of lymphoma subtypes: results from the Epilymph case-control study. Occup Environ Med 67: 341-347. http://dx.doi.org/10.1136/oem.2009.046839

Cocco, P; Vermeulen, R; Flore, V; Nonne, T; Campagna, M; Purdue, M; Blair, A; Monnereau, A; Orsi, L; Clavel, J; Becker, N; de Sanjosé, S; Foretova, L; Staines, A; Maynadié, M; Nieters, A; Miligi, L; 'T Mannetje, A; Kricker, A; Brennan, P; Boffetta, P; Lan, Q; Rothman, N. (2013). Occupational exposure to trichloroethylene and risk of non-Hodgkin lymphoma and its major subtypes: a pooled IinterLlymph analysis. Occup Environ Med 70: 795-802. http://dx.doi.org/10.1136/oemed-2013-101551

Collier, JM; Selmin, O; Johnson, PD; Runyan, RB. (2003). Trichloroethylene effects on gene expression during cardiac development. Birth Defects Res A Clin Mol Teratol 67: 488-495. http://dx.doi.org/10.1002/bdra.10073

Cordier, S; Garlantézec, R; Labat, L; Rouget, F; Monfort, C; Bonvallot, N; Roig, B; Pulkkinen, J; Chevrier, C; Multigner, L, uc. (2012). Exposure during pregnancy to glycol ethers and chlorinated solvents and the risk of congenital malformations. Epidemiology 23: 806-812. http://dx.doi.org/10.1097/EDE.0b013e31826c2bd8

Daubert, TE; Danner, RP. (1989). Physical and thermodynamic properties of pure chemicals: Data compilation. Washington, DC: Taylor & Francis.

Daubert, TE; Danner, RP. (1995). Physical and thermodynamic properties of pure chemicals: Data compilation. Washington DC: Taylor and Francis.

Davis, A; Gift, JS; Woodall, GM; Narotsky, MG; Fourman, GL. (2009). The role of developmental toxicity studies in acute exposure assessments: analysis of single-day vs. multiple-day exposure regimens. Regul Toxicol Pharmacol 54: 134-142. http://dx.doi.org/10.1016/j.yrtph.2009.03.006

Dawson, B; Johnson, P; Goldberg, S; Ulreich, J. (1990). Cardiac teratogenesis of trichloroethylene and dichloroethylene in a mammalian model. J Am Coll Cardiol 16: 1304-1309.

Dawson, B; Johnson, P; Goldberg, S; Ulreich, J. (1993). Cardiac teratogenesis of halogenated hydrocarbon-contaminated drinking water. J Am Coll Cardiol 21: 1466-1472. http://dx.doi.org/10.1016/0735-1097(93)90325-U

Dekant, W; Bridges, J. (2016). Assessment of reproductive and developmental effects of DINP, DnHP and DCHP using quantitative weight of evidence. Regul Toxicol Pharmacol 81: 397-406. http://dx.doi.org/10.1016/j.yrtph.2016.09.032

Demou, E; Hellweg, S; Wilson, MP; Hammond, SK; McKone, TE. (2009). Evaluating indoor exposure modeling alternatives for LCA: A case study in the vehicle repair industry. Environ Sci Technol 43: 5804-5810. http://dx.doi.org/10.1021/es803551y

Di Toro, DM. (1984). Probability Model of Stream Quality Due to Runoff. ASCE. J Environ Eng 110: 607-628.

Dierickx, PJ. (1993). Comparison between fish lethality data and the in vitro cytotoxicity of lipophilic solvents to cultured fish cells in a two-compartment model. Chemosphere 27: 1511-1518.

Dilling, WL; Tefertiller, NB; Kallos, GJ. (1975). Evaporation rates and reactivities of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. Environ Sci Technol 9: 833-838. http://dx.doi.org/10.1021/es60107a008

Dobaradaran, S; Mahvi, AH; Nabizadeh, R; Ramavandi, B; Nazmara, S; Zarei, S. (2012). BIOASSAY COMPARISON OF TRICHLOROETHYLENE (TCE) TOXICITY ON DAPHNIA MAGNA (D. MAGNA) BEFORE AND AFTER ULTRASOUND AND PHOTOLYSIS PROCESSES. Fresen Environ Bull 21: 1533-1538.

Dodson, RE; Levy, JI; Spengler, JD; Shine, JP; Bennett, DH. (2008). Influence of basements, garages, and common hallways on indoor residential volatile organic compound concentrations. Atmos Environ 42: 1569-1581. http://dx.doi.org/10.1016/j.atmosenv.2007.10.088

Dorfmueller, MA; Henne, SP; York, RG; Bornschein, RL; Manson, JM. (1979). Evaluation of teratogenicity and behavioral toxicity with inhalation exposure of maternal rats to trichloroethylene. Toxicology 14: 153-166. http://dx.doi.org/10.1016/0300-483X(79)90061-1

Dosemeci, M; Cocco, P; Chow, WH. (1999). Gender differences in risk of renal cell carcinoma and occupational exposures to chlorinated aliphatic hydrocarbons. Am J Ind Med 36: 54-59. http://dx.doi.org/10.1002/(SICI)1097-0274(199907)36:1<54::AID-AJIM8>3.0.CO;2-0

Dow, J; Green, T. (2000). Trichloroethylene induced vitamin B(12) and folate deficiency leads to increased formic acid excretion in the rat. Toxicology 146: 123-136. http://dx.doi.org/10.1016/S0300-483X(00)00156-6

Drake, V; Koprowski, S; Lough, J; Hu, N; Smith, S. (2006a). Trichloroethylene exposure during cardiac valvuloseptal morphogenesis alters cushion formation and cardiac hemodynamics in the avian embryo. Environ Health Perspect 114: 842-847. http://dx.doi.org/10.1289/ehp.8781

Drake, VJ; Koprowski, SL; Hu, N; Smith, SM; Lough, J. (2006b). Cardiogenic effects of trichloroethylene and trichloroacetic acid following exposure during heart specification of avian development. Toxicol Sci 94: 153-162. http://dx.doi.org/10.1093/toxsci/kfl083

Duteaux, SB; Berger, T; Hess, RA; Sartini, BL; Miller, MG. (2004). Male reproductive toxicity of trichloroethylene: Sperm protein oxidation and decreased fertilizing ability. Biol Reprod 70: 1518-1526. http://dx.doi.org/10.1095/biolreprod.103.022210

EC. (2018). Memorandum on weight of evidence and uncertainties. Revision 2018. Scientific Committee on Health, Environmental and Emerging Risks (SCHEER). https://ec.europa.eu/health/sites/health/files/scientific_committees/scheer/docs/scheer_o_014.pdf

ECB. (2000). IUCLID dataset: CAS No. 79-01-6: Trichloroethylene. Ispra, Italy: European Chemicals Bureau, European Commission. Retrieved from https://echa.europa.eu/substance-information/-/substanceinfo/100.001.062

ECB. (2004). European Union risk assessment report: Trichloroethylene (pp. 1-348). (EUR 21057 EN). European Commission. https://echa.europa.eu/documents/10162/83f0c99f-f687-4cdf-a64b-514f1e26fdc0

ECHA. (2004). Summary risk assessment report: Trichloroethylene. (I.04.29). Ispra, Italy: European Commission Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau. https://echa.europa.eu/documents/10162/d30e53cc-89e7-4d1c-89c0-7ec216f84d48

ECHA. (2017). Registration dossier: Trichloroethylene. Reasonably available online at https://echa.europa.eu/el/registration-dossier/-/registered-dossier/14485 (accessed October 1, 2018).

EFSA. (2017). Guidance on the use of the weight of evidence approach in scientific assessments. EFSA J 15: 1-69. http://dx.doi.org/10.2903/j.efsa.2017.4971

Engineers, USACo. (2018). Weight-of-Evidence Concepts: Introduction and Application to Sediment Management. https://apps.dtic.mil/dtic/tr/fulltext/u2/1048843.pdf

Environment Canada and Health Canada. (1993). Canadian Environmental protection act priority substances list assessment report trichloroethylene. Ottawa Canada.

Epstein, DL; Nolen, GA; Randall, JL; Christ, SA; Read, EJ; Stober, JA; Smith, MK. (1992). Cardiopathic effects of dichloroacetate in the fetal Long-Evans rat. Teratology 46: 225-235. http://dx.doi.org/10.1002/tera.1420460306

Esmen, N; Corn, M; Hammad, Y; Whittier, D; Kotsko, N. (1979). Summary of measurements of employee exposure to airborne dust and fiber in sixteen facilities producing man-made mineral fibers. Am Ind Hyg Assoc J 40: 108-117.

Etterson, M. (2019). Species Sensitivity Distribution (SSD) Toolbox. Duluth, MN: US Environmental Protection Agency.

European Solvents Industry Group (ESIG). (2012). SPERC fact sheet: Manufacture of substance - industrial (solvent-borne). Brussels, Belgium. https://www.esig.org/reach-ges/environment/

Fisher, J; Channel, S; Eggers, J; Johnson, P; Macmahon, K; Goodyear, C; Sudberry, G; Warren, D; Latendresse, J; Graeter, L. (2001). Trichloroethylene, trichloroacetic acid, and dichloroacetic acid: Do they affect fetal rat heart development. Int J Toxicol 20: 257-267. http://dx.doi.org/10.1080/109158101753252992

Fleeman, TL; Cappon, GD; Hurtt, ME. (2004). Postnatal closure of membranous ventricular septal defects in Sprague-Dawley rat pups after maternal exposure with trimethadione. Birth Defects Res B Dev Reprod Toxicol 71: 185-190. http://dx.doi.org/10.1002/bdrb.20011

Forand, SP; Lewis-Michl, EL; Gomez, MI. (2012). Adverse birth outcomes and maternal exposure to trichloroethylene and tetrachloroethylene through soil vapor intrusion in New York State. Environ Health Perspect 120: 616-621. http://dx.doi.org/10.1289/ehp.1103884

Forkert, P; Lash, L; Nadeau, V; Tardif, R; Simmonds, A. (2002). Metabolism and toxicity of trichloroethylene in epididymis and testis. Toxicol Appl Pharmacol 182: 244-254.

Fort, D; Rayburn, J; Deyoung, D; Bantle, J. (1991). Assessing the efficacy of an Aroclor 1254-induced exogenous metabolic activation system for FETAX. Drug Chem Toxicol 14: 143-160. http://dx.doi.org/10.3109/01480549109017873

Fort, D; Rogers, R; Stover, E; Finch, R. (2001). Optimization of an exogenous metabolic activation system for FETAX. I. Post-isolation rat liver microsome mixtures. Drug Chem Toxicol 24: 103-115. http://dx.doi.org/10.1081/DCT-100102604

Fort, DJ; Stover, EL; Rayburn, JR; Hull, M; Bantle, JA. (1993). Evaluation of the developmental toxicity of trichloroethylene and detoxification metabolites using Xenopus. Birth Defects Res B Dev Reprod Toxicol 13: 35-45.

Fredriksson, A; Danielsson, BRG; Eriksson, P. (1993). Altered behaviour in adult mice orally exposed to tri- and tetrachloroethylene as neonates. Toxicol Lett 66: 13-19. http://dx.doi.org/10.1016/0378-4274(93)90074-8

Gangwal, S; Reif, DM; Mosher, S; Egeghy, PP; Wambaugh, JF; Judson, RS; Hubal, EA. (2012). Incorporating exposure information into the toxicological prioritization index decision support framework. Sci Total Environ 435-436: 316-325. http://dx.doi.org/10.1016/j.scitotenv.2012.06.086

Gash, D; Rutland, K; Hudson, N; Sullivan, P; Bing, G; Cass, W; Pandya, J; Liu, M; Choi, D; Hunter, R; Gerhardt, G; Smith, C; Slevin, J; Prince, T. (2008). Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. Ann Neurol 63: 184-192. http://dx.doi.org/10.1002/ana.21288

Geiger, DL; Northcott, CE; Call, DJ; Brooke, LT. (1985). Acute toxicities of organic chemicals to fathead minnows (Pimephales promelas): Volume II. Superior, WI: University of Wisconsin-Superior, Center for Lake Superior Environmental Studies.

George, JD; Reel, J. R.; Myers, CB; Lawton, AD; Lamb, JC. (1986). Trichloroethylene: Reproduction and fertility assessment in F344 rats when administered in the feed (pp. 312 PP). (NTP-86-085). Research Triangle Park, NC: National Institute of Environmental Health Sciences, National Toxicology Program.

Gilboa, SM; Desrosiers, TA; Lawson, C; Lupo, PJ; Riehle-Colarusso, TJ; Stewart, PA; van Wijngaarden, E; Waters, MA; Correa, A; Stud, NBDP. (2012). Association between maternal occupational exposure to organic solvents and congenital heart defects, National Birth Defects Prevention Study, 1997-2002. Occup Environ Med 69: 628-635. http://dx.doi.org/10.1136/oemed-2011-100536

Goldberg, SJ; Lebowitz, MD; Graver, EJ; Hicks, S. (1990). An association of human congenital cardiac malformations and drinking water contaminants. J Am Coll Cardiol 16: 155-164.

Goldman, SM; Quinlan, PJ; Ross, GW; Marras, C; Meng, C; Bhudhikanok, GS; Comyns, K; Korell, M; Chade, AR; Kasten, M; Priestley, B; Chou, KL; Fernandez, HH; Cambi, F; Langston, JW; Tanner, CM. (2012). Solvent exposures and parkinson disease risk in twins. Ann Neurol 71: 776-784. http://dx.doi.org/10.1002/ana.22629

Golsteijn, L; Huizer, D; Hauck, M; van Zelm, R; Huijbregts, MA. (2014). Including exposure variability in the life cycle impact assessment of indoor chemical emissions: the case of metal degreasing. Environ Int 71: 36-45. http://dx.doi.org/10.1016/j.envint.2014.06.003

Gough, D. (2007). Weight of Evidence: a framework for the appraisal of the quality and relevance of evidence. 22: 213-228. http://dx.doi.org/10.1080/02671520701296189

Green, T; Dow, J; Ong, C; Ng, V; Ong, H; Zhuang, Z; Yang, X; Bloemen, L. (2004). Biological monitoring of kidney function among workers occupationally exposed to trichloroethylene. Occup Environ Med 61: 312-317. http://dx.doi.org/10.1136/oem.2003.007153

Greenland, S; Salvan, A; Wegman, DH; Hallock, MF; Smith, TJ. (1994). A case-control study of cancer mortality at a transformer-assembly facility. Int Arch Occup Environ Health 66: 49-54. http://dx.doi.org/10.1007/BF00386579

Hansen, J; Raaschou-Nielsen, O; Christensen, JM; Johansen, I; Mclaughlin, JK; Lipworth, L; Blot, WJ; Olsen, JH. (2001). Cancer incidence among Danish workers exposed to trichloroethylene. J Occup Environ Med 43: 133-139.

Hansen, J; Sallmen, M; Selden, AI; Anttila, A; Pukkala, E; Andersson, K; Bryngelsson, I; Raaschou-Nielsen, O, le; Olsen, JH; Mclaughlin, JK. (2013). Risk of Cancer Among Workers Exposed to Trichloroethylene: Analysis of Three Nordic Cohort Studies. J Natl Cancer Inst 105: 869-877. http://dx.doi.org/10.1093/jnci/djt107

Hardell, L; Eriksson, M; Degerman, A. (1994). Exposure to phenoxyacetic acids, chlorophenols, or organic solvents in relation to histopathology, stage, and anatomical localization of non-Hodgkin's lymphoma. Cancer Res 54: 2386-2389.

Hardin, BD; Bond, GP; Sikov, MR; Andrew, FD; Beliles, RP; Niemeier, RW. (1981). Testing of selected workplace chemicals for teratogenic potential. Scand J Work Environ Health 7: 66-75.

Harris, AP; Ismail, KA; Nunez, M; Martopullo, I; Lencinas, A; Selmin, OI; Runyan, RB. (2018). Trichloroethylene perturbs HNF4a expression and activity in the developing chick heart. Toxicol Lett 285: 113-120. http://dx.doi.org/10.1016/j.toxlet.2017.12.027

Hassoun, E; Kariya, C; Williams, F. (2005). Dichloroacetate-induced developmental toxicity and production of reactive oxygen species in zebrafish embryos. J Biochem Mol Toxicol 19: 52-58. http://dx.doi.org/10.1002/jbt.20051

Hayashi, M; Ueda, T; Uyeno, K; Wada, K; Kinae, N; Saotome, K; Tanaka, N; Takai, A; Sasaki, YF; Asano, N; Sofuni, T; Ojima, Y. (1998). Development of genotoxicity assay systems that use aquatic organisms. Mutat Res 399: 125-133.

Healy, TEJ; Poole, TR; Hopper, A. (1982). Rat fetal development and maternal exposure to trichloroethylene 100 ppm. Br J Anaesth 54: 337-341.

Heavner, DL; Morgan, WT; Ogden, MW. (1995). Determination of volatile organic compounds and ETS apportionment in 49 homes. Environ Int 21: 3-21. http://dx.doi.org/10.1016/0160-4120(94)00018-3

Hellweg, S; Demou, E; Bruzzi, R; Meijer, A; Rosenbaum, RK; Huijbregts, MA; Mckone, TE. (2009). Integrating human indoor air pollutant exposure within Life Cycle Impact Assessment [Review]. Environ Sci Technol 43: 1670-1679. http://dx.doi.org/10.1021/es8018176

Higgins, JP; Thompson, SG; Deeks, JJ; Altman, DG. (2003). Measuring inconsistency in meta-analyses [Review]. BMJ 327: 557-560. http://dx.doi.org/10.1136/bmj.327.7414.557

Hill, AB. (1965). The environment and disease: Association or causation? Proc R Soc Med 58: 295-300.

Horvath, AL; Getzen, FW; Maczynska, Z. (1999). IUPAC-NIST Solubility data series 67: Halogenated ethanes and ethenes with water. J Phys Chem Ref Data 28: 395-627. http://dx.doi.org/10.1063/1.556039

Houde, M; Douville, M; Gagnon, P; Sproull, J; Cloutier, F. (2015). Exposure of Daphnia magna to trichloroethylene (TCE) and vinyl chloride (VC): evaluation of gene transcription, cellular activity, and life-history parameters. Ecotoxicol Environ Saf 116: 10-18. http://dx.doi.org/10.1016/j.ecoenv.2015.02.031

Hudson, NL; Dotson, GS. (2017). NIOSH Skin Notation (SK) Profile: Trichloroethylene (TCE) (CAS No. 79-01-6).

Hunter, E; Rogers, E; Schmid, J; Richard, A. (1996). Comparative effects of haloacetic acids in whole embryo culture. Teratology 54: 57-64. http://dx.doi.org/10.1002/(SICI)1096-9926(199606)54:2<57::AID-TERA1>3.0.CO;2-1

IARC. (2014). IARC Monographs on the evaluation of carcinogenic risks to humans: Trichloroethylene, tetrachloroethylene, and some other chlorinated agents. Geneva, Switzerland: World Health Organization, International Agency for Research on Cancer. http://monographs.iarc.fr/ENG/Monographs/PDFs/index.php

Ikeda, M; Imamura, T. (1973). Biological half-life of trichloroethylene and tetrachloroethylene in human subjects [Review]. Int Arch Occup Environ Health 31: 209-224. http://dx.doi.org/10.1007/BF00539241

IRTA. (2007). Spotting chemicals: Alternatives to perchloroethylene and trichloroethylene in the textile cleaning industry. Prepared for: Cal/EPA's Department of Toxic Substances Control and U.S. Environmental Protection Agency Region IX. http://www.irta.us/reports/DTSC%20Spotting%20Chemical%20for%20Web.pdf

Isaacs, K. (2014). The consolidated human activity database - master version (CHAD-Master) technical memorandum. Washington, DC: U.S. Environmental Protection Agency, National Exposure Research Laboratory. https://www.epa.gov/sites/production/files/2015-02/documents/chadmaster_091814_1.pdf

Isaacson, LG; Spohler, SA; Taylor, DH. (1990). Trichloroethylene affects learning and decreases myelin in the rat hippocampus. Neurotoxicol Teratol 12: 375-381. http://dx.doi.org/10.1016/0892-0362(90)90057-J

Jayjock, MA. (2012). Engineering case report: Estimating overspray exposure potential from aerosol sprayed products onto surfaces. J Occup Environ Hyg 9: D155-D160. http://dx.doi.org/10.1080/15459624.2012.700191

Jenkins, KJ; Correa, A; Feinstein, JA; Botto, L; Britt, AE; Daniels, SR; Elixson, M; Warnes, CA; Webb, CL. (2007). Noninherited risk factors and congenital cardiovascular defects: Current knowledge: A scientific statement from the American Heart Association Council on cardiovascular disease in the young. Circulation 115: 2995-3014. http://dx.doi.org/10.1161/CIRCULATIONAHA.106.183216

Jia, C; Batterman, S; Godwin, C. (2008a). VOCs in industrial, urban and suburban neighborhoods, Part 1: Indoor and outdoor concentrations, variation, and risk drivers. Atmos Environ 42: 2083-2100. http://dx.doi.org/10.1016/j.atmosenv.2007.11.055

Jia, CR; D'Souza, J; Batterman, S. (2008b). Distributions of personal VOC exposures: A population-based analysis. Environ Int 34: 922-931. http://dx.doi.org/10.1016/j.envint.2008.02.002

Jiang, Y; Wang, D; Zhang, G; Wang, G; Tong, J; Chen, T. (2015). Disruption of cardiogenesis in human embryonic stem cells exposed to trichloroethylene. Environ Toxicol 31: 1372-1380. http://dx.doi.org/10.1002/tox.22142

Johnson, P. (2008). Personal communication from Paula Johnson, University of Arizona, to Susan Makris, U.S. EPA, 26 August 2008 [Personal Communication].

Johnson, P. (2014). [Personal communication from Paula Johnson, University of Arizona, to Susan Makris, U.S. EPA, 21 February 2014] [Personal Communication].

Johnson, PD; Dawson, BV; Goldberg, SJ. (1998). Cardiac teratogenicity of trichloroethylene metabolites. J Am Coll Cardiol 32: 540-545. http://dx.doi.org/10.1016/S0735-1097(98)00232-0

Johnson, PD; Goldberg, SJ; Mays, MZ; Dawson, BV. (2003). Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat. Environ Health Perspect 111: 289-292. http://dx.doi.org/10.1289/ehp.5125

Johnson, PD; Goldberg, SJ; Mays, MZ; Dawson, BV. (2005). Erratum: Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat ” (Johnson et al. 2003) [Erratum]. Environ Health Perspect 113: A18.

Johnson, PD; Goldberg, SJ; Mays, MZ; Dawson, BV. (2014). Erratum: Erratum for Johnson et al. [Environ Health Perspect 113:A18 (2005)] [Erratum]. Environ Health Perspect 122: A94. http://dx.doi.org/10.1289/ehp.122-A94

Jung, H; Kim, H; Song, B; Kim, E. (2012). Trichloroethylene Hypersensitivity Syndrome: A Disease of Fatal Outcome. Yonsei Med J 53: 231-235. http://dx.doi.org/10.3349/ymj.2012.53.1.231

Kan, FW; Forkert, PG; Wade, MG. (2007). Trichloroethylene exposure elicits damage in epididymal epithelium and spermatozoa in mice. Histol Histopathol 22: 977-988. http://dx.doi.org/10.14670/HH-22.977

Kaneko, T; Saegusa, M; Tasaka, K; Sato, A. (2000). Immunotoxicity of trichloroethylene: A study with MRL-lpr/lpr mice. J Appl Toxicol 20: 471-475. http://dx.doi.org/10.1002/1099-1263(200011/12)20:6<471::AID-JAT716>3.0.CO;2-E

Kasting, BG; Miller, MA. (2006). Kinetics of finite dose absorption through skin 2: Volatile compounds. J Pharm Sci 95: 268-280. http://dx.doi.org/10.1002/jps.20497

Keil, DE; Peden-Adams, MM; Wallace, S; Ruiz, P; Gilkeson, GS. (2009). Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease. J Environ Sci Health A Tox Hazard Subst Environ Eng 44: 443-453. http://dx.doi.org/10.1080/10934520902719738

Kjellstrand, P; Holmquist, B; Alm, P; Kanje, M; Romare, S; Jonsson, I; Månsson, L; Bjerkemo, M. (1983). Trichloroethylene: Further studies of the effects on body and organ weights and plasma butyrylcholinesterase activity in mice. Acta Pharmacol Toxicol 53: 375-384. http://dx.doi.org/10.1111/j.1600-0773.1983.tb03438.x

Kjellstrand, P; Kanje, M; Bjerkemo, M. (1987). Regeneration of the sciatic nerve in mice and rats exposed to trichloroethylene. Toxicol Lett 38: 187-191. http://dx.doi.org/10.1016/0378-4274(87)90127-5

Klein, P; Kurz, J. (1994a). [Reduction of Solvent Concentrations in Surroundings of Dry-Cleaning Shops]. Bonningheim, Germany: Hohenstein Physiological lnstitute on Clothing.

Klimisch, HJ; Andreae, M; Tillmann, U. (1997). A systematic approach for evaluating the quality of experimental toxicological and ecotoxicological data. Regul Toxicol Pharmacol 25: 1-5. http://dx.doi.org/10.1006/rtph.1996.1076

Kumar, P; Prasad, A; Mani, U; Maji, B; Dutta, K. (2001). Trichloroethylene induced testicular toxicity in rats exposed by inhalation. Hum Exp Toxicol 20: 585-589. http://dx.doi.org/10.1191/096032701718620882

Kumar, P; Prasad, AK; Saxena, DK; Manu, U; Maji, BK; Dutta, KK. (2000). Fertility and general reproduction studies in trichloroethylene exposed rats. Indian Journal of Occupational Health 43: 117-126.

Labra, M; Mattia, F; Bernasconi, M; Bertacchi, D; Grassi, F; Bruni, I; Citterio, S. (2010). The Combined Toxic and Genotoxic Effects of Chromium and Volatile Organic Contaminants to Pseudokirchneriella subcapitata. Water Air Soil Pollut 213: 57-70. http://dx.doi.org/10.1007/s11270-010-0367-3

Lagakos, SW; Wessen, BJ; Zelen, M. (1986). An analysis of contaminated well water and health effects in Woburn, Massachusetts. J Am Stat Assoc 81: 583-596. http://dx.doi.org/10.2307/2288982

LeBlanc, GA. (1980). Acute toxicity of priority pollutants to water flea (Daphnia magna). Bull Environ Contam Toxicol 24: 684-691. http://dx.doi.org/10.1007/BF01608174

Leblanc, M; Allen, JG; Herrick, RF; Stewart, JH. (2018). Comparison of the near field/far field model and the advanced reach tool (ART) model V1.5: exposure estimates to benzene during parts washing with mineral spirits. Int J Hyg Environ Health 221: 231-238. http://dx.doi.org/10.1016/j.ijheh.2017.10.016

Leighton, DT, Jr; Calo, JM. (1981). Distribution coefficients of chlorinated hydrocarbons in dilute air-water systems for groundwater contamination applications. Journal of Chemical and Engineering Data 26: 382-585. http://dx.doi.org/10.1021/je00026a010

Lide, DR. (2007). CRC handbook of chemistry and physics: A ready-reference book of chemical and physical data. In DR Lide (Ed.), (88th ed.). Boca Raton, FL: CRC Press.

Lindstrom, AB; Proffitt, D; Fortune, CR. (1995). Effects of modified residential construction on indoor air quality. Indoor Air 5: 258-269. http://dx.doi.org/10.1111/j.1600-0668.1995.00005.x

Linkov, I; Massey, O; Keisler, J; Rusyn, I; Hartung, T. (2015). From "Weight of Evidence" to Quantitative Data Integration using Multicriteria Decision Analysis and Bayesian Methods. ALTEX 32: 3-8. http://dx.doi.org/10.14573/altex.1412231

Lipworth, L; Sonderman, JS; Mumma, MT; Tarone, RE; Marano, DE; Boice, JD; Mclaughlin, JK. (2011). Cancer mortality among aircraft manufacturing workers: an extended follow-up. J Occup Environ Med 53: 992-1007. http://dx.doi.org/10.1097/JOM.0b013e31822e0940

Liu, M, ei; Choi, DY; Hunter, RL; Pandya, JD; Cass, WA; Sullivan, PG; Kim, HC; Gash, D; Bing, G. (2010). Trichloroethylene induces dopaminergic neurodegeneration in Fisher 344 rats. J Neurochem 112: 773-783. http://dx.doi.org/10.1111/j.1471-4159.2009.06497.x

Loeber, C; Hendrix, M; Diez De Pinos, S; Goldberg, S. (1988). Trichloroethylene: A cardiac teratogen in developing chick embryos. Pediatr Res 24: 740-744. http://dx.doi.org/10.1203/00006450-198812000-00018

Long, JL; Stensel, HD; Ferguson, JF; Strand, SE; Ongerth, JE. (1993). Anaerobic and aerobic treatment of chlorinated aliphatic compounds. J Environ Eng 119: 300-320.

Lukavsky, J; Furnadzhieva, S; Dittrt, F. (2011). Toxicity of Trichloroethylene (TCE) on Some Algae and Cyanobacteria. Bull Environ Contam Toxicol 86: 226-231. http://dx.doi.org/10.1007/s00128-011-0195-1

Lutz, WK; Gaylor, DW; Conolly, RB; Lutz, RW. (2005). Nonlinearity and thresholds in dose-response relationships for carcinogenicity due to sampling variation, logarithmic dose scaling, or small differences in individual susceptibility [Review]. Toxicol Appl Pharmacol 207: S565-S569. http://dx.doi.org/10.1016/j.taap.2005.01.038

Makris, SL; Scott, CS; Fox, J; Knudsen, TB; Hotchkiss, AK; Arzuaga, X; Euling, SY; Powers, CM; Jinot, J; Hogan, KA; Abbott, BD; Hunter, ES; Narotsky, MG. (2016). A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development [Review]. Reprod Toxicol 65: 321-358. http://dx.doi.org/10.1016/j.reprotox.2016.08.014

Makwana, O; Ahles, L; Lencinas, A; Selmin, OI; Runyan, RB. (2013). Low-dose trichloroethylene alters cytochrome P450-2C subfamily expression in the developing chick heart. Cardiovasc Toxicol 13: 77-84. http://dx.doi.org/10.1007/s12012-012-9180-0

Makwana, O; King, NM; Ahles, L; Selmin, O; Granzier, HL; Runyan, RB. (2010). Exposure to low-dose trichloroethylene alters shear stress gene expression and function in the developing chick heart. Cardiovasc Toxicol 10: 100-107. http://dx.doi.org/10.1007/s12012-010-9066-y

Malarkey, DE; Bucher, JR. (2011). Summary report of the National Toxicology Program and Environmental Protection Agency-sponsored review of pathology materials from selected Ramazzini Institute rodent cancer bioassays [NTP]. Research Triangle Park: National Toxicology Program. http://ntp.niehs.nih.gov/ntp/about_ntp/partnerships/international/summarypwg_report_ri_bioassays.pdf

Maltoni, C; Lefemine, G; Cotti, G. (1986). Experimental research on trichloroethylene carcinogenesis. In Experimental research on trichloroethylene carcinogenesis. Princeton, NJ: Princeton Scientific Publishing.

Mao, B; Qiu, J; Zhao, N; Shao, Y; Dai, W; He, W; Cui, H; Lin, X; Lv, L; Tang, Z; Xu, S; Huang, H; Zhou, M; Xu, X; Qiu, W; Liu, Q; Zhang, Y. (2017). Maternal folic acid supplementation and dietary folate intake and congenital heart defects. PLoS ONE 1: 1-14. http://dx.doi.org/10.1371/journal.pone.0187996

Marquart, H; Franken, R; Goede, H; Fransman, W; Schinkel, J. (2017). Validation of the dermal exposure model in ECETOC TRA. Annals of Work Exposures and Health 61: 854-871. http://dx.doi.org/10.1093/annweh/wxx059

McDaniel, T; Martin, P; Ross, N; Brown, S; Lesage, S; Pauli, B. (2004). Effects of chlorinated solvents on four species of North American amphibians. Arch Environ Contam Toxicol 47: 101-109.

Miligi, L; Costantini, AS; Benvenuti, A; Kriebel, D; Bolejack, V; Tumino, R; Ramazzotti, V; Rodella, S; Stagnaro, E; Crosignani, P; Amadori, D; Mirabelli, D; Sommani, L; Belletti, I; Troschel, L; Romeo, L; Miceli, G; Tozzi, GA; Mendico, I; Vineis, P. (2006). Occupational exposure to solvents and the risk of lymphomas. Epidemiology 17: 552-561. http://dx.doi.org/10.1097/01.ede.0000231279.30988.4d

Mishima, N; Hoffman, S; Hill, EG; Krug, EL. (2006). Chick embryos exposed to trichloroethylene in an ex ovo culture model show selective defects in early endocardial cushion tissue formation. Birth Defects Res A Clin Mol Teratol 76: 517-527. http://dx.doi.org/10.1002/bdra.20283

Moore, LE; Boffetta, P; Karami, S; Brennan, P; Stewart, PS; Hung, R; Zaridze, D; Matveev, V; Janout, V; Kollarova, H; Bencko, V; Navratilova, M; Szeszenia-Dabrowska, N; Mates, D; Gromiec, J; Holcatova, I; Merino, M; Chanock, S; Chow, WH; Rothman, N. (2010). Occupational trichloroethylene exposure and renal carcinoma risk: Evidence of genetic susceptibility by

reductive metabolism gene variants. Cancer Res 70: 6527-6536. http://dx.doi.org/10.1158/0008-5472.CAN-09-4167

Morgan, RW; Kelsh, MA; Zhao, K; Heringer, S. (1998). Mortality of aerospace workers exposed to trichloroethylene. Epidemiology 9: 424-431.

Morris, M; Wolf, K. (2005). Evaluation of New and Emerging Technologies for Textile Cleaning. Institute for Research and Technical Assistance (IRTA). http://wsppn.org/pdf/irta/Emerging_Technologies_Textile_%20Clea.pdf

NAC/AEGL. (2009). Trichloroethylene (CAS reg. no. 79-01-6): Interim acute exposure guidelines levels (AEGLs) [AEGL]. Washington, DC: National Advisory Committee for Acute Exposure Guideline Levels. https://www.epa.gov/sites/production/files/2014-08/documents/trichloroethylene_interim_dec_2008_v1.pdf

Narotsky, MG; Weller, EA; Chinchilli, VM; Kavlock, RJ. (1995). Nonadditive developmental toxicity in mixtures of trichloroethylene, di(2-ethylhexyl) phthalate, and heptachlor in a 5 x 5 x 5 design. Fundam Appl Toxicol 27: 203-216. http://dx.doi.org/10.1093/toxsci/27.2.203

NCI. (1976). Carcinogenesis bioassay of trichloroethylene. (NCI-CG-TR-2). Bethesda, MD: U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health. http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr002.pdf

Nicas, M. (2009). The near field/far field (two-box) model with a constant contamination emission rate. In CB Keil; CE Simmons; TR Anthony (Eds.), (2nd ed., pp. 47-52). Fairfax, VA: AIHA Press.

NICNAS. (2000). Trichloroethylene: Priority existing chemical assessment report no. 8. (8). Sydney, Australia. http://www.nicnas.gov.au/Publications/CAR/PEC/PEC8.asp

Niederlehner, B; Cairns, J; Smith, E. (1998). Modeling acute and chronic toxicity of nonpolar narcotic chemicals and mixtures to Ceriodaphnia dubia. Ecotoxicol Environ Saf 39: 136-146. http://dx.doi.org/10.1006/eesa.1997.1621

NIOSH. (2001). Respirator Usage in Private Sector Firms. Washington D.C.: United States Department of Labor, Bureau of Labor Statistics and National Institute for Occupational Safety and Health. https://www.cdc.gov/niosh/docs/respsurv/

Nordström, M; Hardell, L; Magnuson, A; Hagberg, H; Rask-Andersen, A. (1998). Occupational exposures, animal exposure and smoking as risk factors for hairy cell leukaemia evaluated in a case-control study. Br J Cancer 77: 2048-2052.

NRC. (2001). Standing Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Chemicals. http://www.nap.edu/catalog.php?record_id=10122

NRC. (2006). Assessing the human health risks of trichloroethylene: Key scientific issues. Washington, DC: The National Academies Press. http://www.nap.edu/catalog.php?record_id=11707

NTP. (1988). Toxicology and carcinogenesis studies of trichloroethylene (CAS No. 79-01-6) in four strains of rats (ACI, August, Marshall, Osborne-Mendel) (gavage studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr273.pdf

NTP. (2015). Handbook for conducting a literature-based health assessment using OHAT approach for systematic review and evidence integration. U.S. Dept. of Health and Human Services, National Toxicology Program. https://ntp.niehs.nih.gov/ntp/ohat/pubs/handbookjan2015_508.pdf

O'Neil, MJ; Heckelman, PE; Koch, CB. (2006). The Merck index: An encyclopedia of chemicals, drugs, and biologicals (14th ed.). Whitehouse Station, NJ: Merck & Co.

O'Neil, MJ; Smith, A; Heckelman, PE. (2001). Trichloroethylene. In Merck Index. Whitehouse Station, NJ: Merck & Co., Inc.

Opdam, JJ. (1989). Intra and interindividual variability in the kinetics of a poorly and highly metabolising solvent. Br J Ind Med 46: 831-845. http://dx.doi.org/10.1136/oem.46.12.831

Ou, J; Ou, Z; Mccarver, D; Hines, R; Oldham, K; Ackerman, A; Pritchard, K. (2003). Trichloroethylene decreases heat shock protein 90 interactions with endothelial nitric oxide synthase: implications for endothelial cell proliferation. Toxicol Sci 73: 90-97. http://dx.doi.org/10.1093/toxsci/kfg062

Palbykin, B; Borg, J; Caldwell, PT; Rowles, J; Papoutsis, AJ; Romagnolo, DF; Selmin, OI. (2011). Trichloroethylene Induces Methylation of the Serca2 Promoter in H9c2 Cells and Embryonic Heart. Cardiovasc Toxicol 11: 204-214. http://dx.doi.org/10.1007/s12012-011-9113-3

Peden-Adams, MM; Eudaly, JG; Heesemann, LM; Smythe, J; Miller, J; Gilkeson, GS; Keil, DE. (2006). Developmental immunotoxicity of trichloroethylene (TCE): Studies in B6C3F1 mice. J Environ Sci Health A Tox Hazard Subst Environ Eng 41: 249-271. http://dx.doi.org/10.1080/10934520500455289

Pennsylvania State University. (2016). Horse Stable Ventilation. https://extension.psu.edu/horse-stable-ventilation

Persson, B; Fredrikson, M. (1999). Some risk factors for non-Hodgkin's lymphoma. Int J Occup Med Environ Health 12: 135-142.

Pesch, B; Haerting, J; Ranft, U; Klimpel, A; Oelschlägel, B; Schill, W. (2000). Occupational risk factors for renal cell carcinoma: Agent-specific results from a case-control study in Germany. Int J Epidemiol 29: 1014-1024. http://dx.doi.org/10.1093/ije/29.6.1014

Poet, TS; Corley, RA; Thrall, KD; Edwards, JA; Tanojo, H; Weitz, KK; Hui, X; Maibach, HI; Wester, RC. (2000). Assessment of the percutaneous absorption of trichloroethylene in rats and humans using MS/MS real-time breath analysis and physiologically based pharmacokinetic modeling. Toxicol Sci 56: 61-72. http://dx.doi.org/10.1093/toxsci/56.1.61

Poole, C; Greenland, S. (1999). Random-effects meta-analyses are not always conservative. Am J Epidemiol 150: 469-475. http://dx.doi.org/10.1093/oxfordjournals.aje.a010035

Purdue, MP; Bakke, B; Stewart, P; De Roos, AJ; Schenk, M; Lynch, CF; Bernstein, L; Morton, LM; Cerhan, JR; Severson, RK; Cozen, W; Davis, S; Rothman, N; Hartge, P; Colt, JS. (2011). A case-control study of occupational exposure to trichloroethylene and non-Hodgkin lymphoma. Environ Health Perspect 119: 232-238. http://dx.doi.org/10.1289/ehp.1002106

Purdue, MP; Stewart, PA; Friesen, MC; Colt, JS; Locke, SJ; Hein, MJ; Waters, MA; Graubard, BI; Davis, F; Ruterbusch, J; Schwartz, K; Chow, WH; Rothman, N; Hofmann, JN. (2016). Occupational exposure to chlorinated solvents and kidney cancer: a case-control study. Occup Environ Med 74: 268-274. http://dx.doi.org/10.1136/oemed-2016-103849

Raaschou-Nielsen, O; Hansen, J; Mclaughlin, JK; Kolstad, H; Christensen, JM; Tarone, RE; Olsen, JH. (2003). Cancer risk among workers at Danish companies using trichloroethylene: A cohort study. Am J Epidemiol 158: 1182-1192. http://dx.doi.org/10.1093/aje/kwg282

Radican, L; Blair, A; Stewart, P; Wartenberg, D. (2008). Mortality of aircraft maintenance workers exposed to trichloroethylene and other hydrocarbons and chemicals: Extended follow-up. J Occup Environ Med 50: 1306-1319. http://dx.doi.org/10.1097/JOM.0b013e3181845f7f

Reif, DM; Sypa, M; Lock, EF; Wright, FA; Wilson, A; Cathey, T; Judson, RR; Rusyn, I. (2013). ToxPi GUI: an interactive visualization tool for transparent integration of data from diverse sources of evidence. Bioinformatics 29: 402-403. http://dx.doi.org/10.1093/bioinformatics/bts686

Rhomberg, LR; Goodman, JE; Bailey, LA; Prueitt, RL; Beck, NB; Bevan, C; Honeycutt, M; Kaminski, NE; Paoli, G; Pottenger, LH; Scherer, RW; Wise, KC; Becker, RA. (2013). A survey of frameworks for best practices in weight-of-evidence analyses [Review]. Crit Rev Toxicol 43: 753-784. http://dx.doi.org/10.3109/10408444.2013.832727

Robinson, KW; Flanagan, SM; Ayotte, JD; Campo, KW; Chalmers, A. (2004). Water Quality in the New England Coastal Basins, Maine, New Hampshire, Massachusetts, and Rhode Island, 1999-2001. (NTIS/02928874). Robinson, KW; Flanagan, SM; Ayotte, JD; Campo, KW; Chalmers, A.

Rooney, AA; Boyles, AL; Wolfe, MS; Bucher, JR; Thayer, KA. (2014). Systematic review and evidence integration for literature-based environmental health science assessments. Environ Health Perspect 122: 711-718. http://dx.doi.org/10.1289/ehp.1307972

Rott, B; Viswanathan, R; Freitag, D; Korte, F. (1982). Vergleichende Untersuchung der Anwendbarkeit verschiedener Tests zur Überprüfung der Abbaubarkeit von Umweltchemikalien. Chemosphere 11: 531-538. http://dx.doi.org/10.1016/0045-6535(82)90186-2

Ruckart, PZ; Bove, FJ; Maslia, M. (2013). Evaluation of exposure to contaminated drinking water and specific birth defects and childhood cancers at Marine Corps Base Camp Lejeune, North Carolina: a case--control study. Environ Health 12: 104. http://dx.doi.org/10.1186/1476-069X-12-104

Ruckart, PZ; Bove, FJ; Maslia, M. (2014). Evaluation of contaminated drinking water and preterm birth, small for gestational age, and birth weight at Marine Corps Base Camp Lejeune, North Carolina: a cross-sectional study. Environ Health 13: 99. http://dx.doi.org/10.1186/1476-069X-13-99

Rufer, ES; Hacker, TA; Flentke, GR; Drake, VJ; Brody, MJ; Lough, J; Smith, SM. (2010). Altered cardiac function and ventricular septal defect in avian embryos exposed to low-dose trichloroethylene. Toxicol Sci 113: 444-452. http://dx.doi.org/10.1093/toxsci/kfp269

Ruijten, MW; Verberk, MM; Sallé, HJ. (1991). Nerve function in workers with long term exposure to trichloroethene. Br J Ind Med 48: 87-92.

Saillenfait, AM; Langonne, I; Sabate, JP. (1995). Developmental toxicity of trichloroethylene, tetrachloroethylene and four of their metabolites in rat whole embryo culture. Arch Toxicol 70: 71-82. http://dx.doi.org/10.1007/BF02733666

Sanchez-Fortun, S; Sanz, F; Santa-Maria, A; Ros, JM; De Vicente, ML; Encinas, MT; Vinagre, E; Barahona, MV. (1997). Acute sensitivity of three age classes of Artemia salina larvae to seven chlorinated solvents. Bull Environ Contam Toxicol 59: 445-451. http://dx.doi.org/10.1007/s001289900498

Sanders, VM; Tucker, AN; White, KL, Jr; Kauffmann, BM; Hallett, P; Carchman, RA; Borzelleca, JF; Munson, AE. (1982). Humoral and cell-mediated immune status in mice exposed to trichloroethylene in the drinking water. Toxicol Appl Pharmacol 62: 358-368. http://dx.doi.org/10.1016/0041-008X(82)90138-7

Sato, A; Nakajima, T; Fujiwara, Y; Murayama, N. (1977). A pharmacokinetic model to study the excretion of trichloroethylene and its metabolites after an inhalation exposure. Br J Ind Med 34: 56-63.

Sauer, TC. (1981). Volatile organic compounds in open ocean and coastal surface waters. Organic Geochemistry 3: 91-101.

Sax, SN; Bennett, DH; Chillrud, SN; Kinney, PL; Spengler, JD. (2004). Differences in source emission rates of volatile organic compounds in inner-city residences of New York City and Los Angeles. J Expo Anal Environ Epidemiol 14: S95-109. http://dx.doi.org/10.1038/sj.jea.7500364

Schell, JD, Jr. (1987) Interactions of halogenated hydrocarbon mixtures in the embryo of the Japanese medaka (Oryzias latipes). (Doctoral Dissertation). Rutgers University, New Brunswick, NJ.

Schmidt, KR; Tiehm, A. (2008). Natural attenuation of chloroethenes: identification of sequential reductive/oxidative biodegradation by microcosm studies. Water Sci Technol 58: 1137-1145. http://dx.doi.org/10.2166/wst.2008.729

Schwetz, BA; Leong, BKJ; Gehring, PJ. (1975). The effect of maternally inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol Appl Pharmacol 32: 84-96. http://dx.doi.org/10.1016/0041-008X(75)90197-0

Selgrade, MK; Gilmour, MI. (2010). Suppression of pulmonary host defenses and enhanced susceptibility to respiratory bacterial infection in mice following inhalation exposure to

trichloroethylene and chloroform. J Immunotoxicol 7: 350-356. http://dx.doi.org/10.3109/1547691X.2010.520139

Selmin, OI; Thorne, PA; Caldwell, PT; Taylor, MR. (2008). Trichloroethylene and trichloroacetic acid regulate calcium signaling pathways in murine embryonal carcinoma cells p19. Cardiovasc Toxicol 8: 47-56. http://dx.doi.org/10.1007/s12012-008-9014-2

Sexton, K; Mongin, SJ; Adgate, JL; Pratt, GC; Ramachandran, G; Stock, TH; Morandi, MT. (2007). Estimating volatile organic compound concentrations in selected microenvironments using time-activity and personal exposure data. J Toxicol Environ Health A 70: 465-476. http://dx.doi.org/10.1080/15287390600870858

Shelton, KL; Nicholson, KL. (2014). Pharmacological classification of the abuse-related discriminative stimulus effects of trichloroethylene vapor. 3: 235839. http://dx.doi.org/10.4303/jdar/235839

Siemiatycki, J, . (1991). Risk factors for cancer in the workplace. In J Siemiatycki (Ed.). Boca Raton, FL: CRC Press.

Silver, SR; Pinkerton, LE; Fleming, DA; Jones, JH; Allee, S; Luo, L; Bertke, SJ. (2014). Retrospective Cohort Study of a Microelectronics and Business Machine Facility. Am J Ind Med 57: 412-424. http://dx.doi.org/10.1002/ajim.22288

Singh, HB; Salas, LJ; Stiles, RE. (1983). Selected man-made halogenated chemicals in the air and oceanic environment. J Geophys Res 88: 3675-3683.

Smith, AD; Bharath, A; Mallard, C; Orr, D; Smith, K; Sutton, JA; Vukmanich, J; McCarty, LS; Ozburn, GW. (1991). The acute and chronic toxicity of ten chlorinated organic compounds to the American flagfish (Jordanella floridae). Arch Environ Contam Toxicol 20: 94-102. http://dx.doi.org/10.1007/BF01065334

Smith, EP; Lipkovich, I; Ye, K. (2002). Weight-of-Evidence (WOE): Quantitative Estimation of Probability of Impairment for Individual and Multiple Lines of Evidence. Hum Ecol Risk Assess 8: 1585-1596.

Smith, MK; Randall, JL; Read, EJ; Stober, JA. (1989). Teratogenic activity of trichloroacetic acid in the rat. Teratology 40: 445-451. http://dx.doi.org/10.1002/tera.1420400506

Smith, MK; Randall, JL; Read, EJ; Stober, JA. (1992). Developmental toxicity of dichloroacetate in the rat. Teratology 46: 217-223. http://dx.doi.org/10.1002/tera.1420460305

Stuckhardt, JL; Poppe, SM. (1984). Fresh visceral examination of rat and rabbit fetuses used in teratogenicity testing. Teratog Carcinog Mutagen 4: 181-188. http://dx.doi.org/10.1002/tcm.1770040203

Su, FC; Mukherjee, B; Batterman, S. (2013). Determinants of personal, indoor and outdoor VOC concentrations: An analysis of the RIOPA data. Environ Res 126: 192-203. http://dx.doi.org/10.1016/j.envres.2013.08.005

Swartz, MD; Cai, Y; Chan, W; Symanski, E; Mitchell, LE; Danysh, HE; Langlois, PH; Lupo, PJ. (2015). Air toxics and birth defects: a Bayesian hierarchical approach to evaluate multiple pollutants and spina bifida. Environ Health 14: 16. http://dx.doi.org/10.1186/1476-069X-14-16

Taylor, DH; Lagory, KE; Zaccaro, DJ; Pfohl, RJ; Laurie, RD. (1985). Effect of trichloroethylene on the exploratory and locomotor activity of rats exposed during development. Sci Total Environ 47: 415-420. http://dx.doi.org/10.1016/0048-9697(85)90345-6

Tielemans, E; Schneider, T; Goede, H; Tischer, M; Warren, N; Kromhout, H; Van Tongeren, M; Van Hemmen, J; Cherrie, JW. (2008). Conceptual model for assessment of inhalation exposure: Defining modifying factors. Ann Occup Hyg 52: 577-586. http://dx.doi.org/10.1093/annhyg/men059

Tobajas, M; Verdugo, V; Polo, AM; Rodriguez, JJ; Mohedano, AF. (2016). Assessment of toxicity and biodegradability on activated sludge of priority and emerging pollutants. Environ Technol 37: 713-721. http://dx.doi.org/10.1080/09593330.2015.1079264

Tsai, KP; Chen, CY. (2007). An algal toxicity database of organic toxicants derived by a closed-system technique. Environ Toxicol Chem 26: 1931-1939. http://dx.doi.org/10.1897/06-612R.1

U.S. BLS. (2014). Employee Tenure News Release. http://www.bls.gov/news.release/archives/tenure_09182014.htm

U.S. BLS. (2016). May 2016 Occupational Employment and Wage Estimates: National Industry-Specific Estimates [Website]. http://www.bls.gov/oes/tables.htm

U.S. Census Bureau. (2013). Census 2012 Detailed Industry Code List [Database]. https://www.census.gov/topics/employment/industry-occupation/guidance/code-lists.html

U.S. Census Bureau. (2015). Statistics of U.S. Businesses (SUSB). https://www.census.gov/data/tables/2015/econ/susb/2015-susb-annual.html

U.S. Census Bureau. (2019). Survey of Income and Program Participation data [Website]. https://www.census.gov/programs-surveys/sipp/data/datasets/2008-panel/wave-1.html

U.S. EPA. (1977). Environmental monitoring near industrial sites methylchloroform [EPA Report]. (EPA-560/5-77-025). Washington, DC.

U.S. EPA. (1987). Household solvent products: A national usage survey. (EPA-OTS 560/5-87-005). Washington, DC: Office of Toxic Substances, Office of Pesticides and Toxic Substances. https://ntrl.ntis.gov/NTRL/dashboard/searchResults.xhtml?searchQuery=PB88132881

U.S. EPA. (1988). Recommendations for and documentation of biological values for use in risk assessment [EPA Report] (pp. 1-395). (EPA/600/6-87/008). Cincinnati, OH: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=34855

U.S. EPA. (1991). Guidelines for developmental toxicity risk assessment (pp. 1-71). (EPA/600/FR-91/001). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=23162

U.S. EPA. (1992). Guidelines for exposure assessment. Federal Register 57(104):22888-22938 [EPA Report]. (EPA/600/Z-92/001). Washington, DC. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263

U.S. EPA. (1994a). Guidelines for Statistical Analysis of Occupational Exposure Data: Final. United States Environmental Protection Agency :: U.S. EPA.

U.S. EPA. (1994b). Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry [EPA Report]. (EPA/600/8-90/066F). Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=71993&CFID=51174829&CFTOKEN=25006317

U.S. EPA. (1996). Guidelines for reproductive toxicity risk assessment (pp. 1-143). (EPA/630/R-96/009). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www.epa.gov/sites/production/files/2014-11/documents/guidelines_repro_toxicity.pdf

U.S. EPA. (1998). Guidelines for ecological risk assessment [EPA Report]. (EPA/630/R-95/002F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www.epa.gov/risk/guidelines-ecological-risk-assessment

U.S. EPA. (2001). Sources, emission and exposure for trichloroethylene (TCE) and related chemicals [EPA Report]. In Govt Reports Announcements & Index (pp. 138). (EPA/600/R-00/099). Washington, DC. https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=21006

U.S. EPA. (2002). A review of the reference dose and reference concentration processes (pp. 1-192). (EPA/630/P-02/002F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://www.epa.gov/osa/review-reference-dose-and-reference-concentration-processes

U.S. EPA. (2005). Guidelines for carcinogen risk assessment [EPA Report] (pp. 1-166). (EPA/630/P-03/001F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://www2.epa.gov/osa/guidelines-carcinogen-risk-assessment

U.S. EPA. (2006). Approaches for the application of physiologically based pharmacokinetic (PBPK) models and supporting data in risk assessment (Final Report) [EPA Report] (pp. 1-123). (EPA/600/R-05/043F). Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=157668

U.S. EPA. (2007). Exposure and fate assessment screening tool (E-FAST): Version 2.0, documentation manual [EPA Report]. (EPA Contract No. EP-W-04-035). Springfield, VA. http://www.epa.gov/opptintr/exposure/pubs/efast2man.pdf

U.S. EPA. (2011b). Appendices for the Toxicological review of trichloroethylene (CAS No. 79-01-6) in support of summary information on the Integrated Risk Information System (IRIS) [EPA Report]. (EPA/635/R-09/011F). Washington, DC. https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100CB6V.txt

U.S. EPA. (2011c). Exposure factors handbook: 2011 edition (final) [EPA Report]. (EPA/600/R-090/052F). Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=236252

U.S. EPA. (2011d). Recommended use of body weight 3/4 as the default method in derivation of the oral reference dose (pp. 1-50). (EPA/100/R11/0001). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum, Office of the Science Advisor. https://www.epa.gov/risk/recommended-use-body-weight-34-default-method-derivation-oral-reference-dose

U.S. EPA. (2011e). Toxicological review of trichloroethylene (CASRN 79-01-6) in support of summary information on the Integrated Risk Information System (IRIS) [EPA Report]. (EPA/635/R-09/011F). Washington, DC. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0106tr.pdf

U.S. EPA. (2012a). Benchmark dose technical guidance. (EPA/100/R-12/001). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www.epa.gov/risk/benchmark-dose-technical-guidance

U.S. EPA. (2012b). Estimation Programs Interface Suite™ for Microsoft® Windows, v 4.11 [Computer Program]. Washington, DC. Retrieved from https://www.epa.gov/tsca-screening-tools/epi-suitetm-estimation-program-interface

U.S. EPA. (2012c). Sustainable futures P2 framework manual [EPA Report]. (EPA-748-B12-001). Washington DC. http://www.epa.gov/sustainable-futures/sustainable-futures-p2-framework-manual

U.S. EPA. (2013a). Final peer review comments for the OPPT trichloroethylene (TCE) draft risk assessment [Website]. https://www.epa.gov/sites/production/files/2017-06/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf

U.S. EPA. (2013b). Interpretive assistance document for assessment of discrete organic chemicals. Sustainable futures summary assessment [EPA Report]. Washington, DC. http://www.epa.gov/sites/production/files/2015-05/documents/05-iad_discretes_june2013.pdf

U.S. EPA. (2014a). Framework for human health risk assessment to inform decision making. Final [EPA Report]. (EPA/100/R-14/001). Washington, DC: U.S. Environmental Protection, Risk Assessment Forum. https://www.epa.gov/risk/framework-human-health-risk-assessment-inform-decision-making

U.S. EPA. (2014b). TSCA work plan chemical risk assessment. Trichloroethylene: Degreasing, spot cleaning and arts & crafts uses. (740-R1-4002). Washington, DC: Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. http://www2.epa.gov/sites/production/files/2015-09/documents/tce_opptworkplanchemra_final_062414.pdf

U.S. EPA. (2014c). Exposure and Fate Assessment Screening Tool Version 2014 (E-FAST 2014). Washington, DC: Office of Pollution Prevention and Toxics. https://www.epa.gov/tsca-screening-tools/e-fast-exposure-and-fate-assessment-screening-tool-version-2014

U.S. EPA. (2015a). EDSP: Weight of Evidence Analysis of Potential Interaction with the Estrogen, Androgen or Thyroid Pathways. Chemical: Glyphosate. Office of Pesticide Programs. https://www.epa.gov/endocrine-disruption/endocrine-disruptor-screening-program-tier-1-screening-determinations-and

U.S. EPA. (2015b). Update of human health ambient water quality criteria: Trichloroethylene (TCE) 79-01-6. (EPA 820-R-15-066). Washington D.C.: Office of Water, Office of Science and Technology. https://www.regulations.gov/document?D=EPA-HQ-OW-2014-0135-0173

U.S. EPA. (2016b). Instructions for reporting 2016 TSCA chemical data reporting. Washington, DC: Office of Pollution Prevention and Toxics. https://www.epa.gov/chemical-data-reporting/instructions-reporting-2016-tsca-chemical-data-reporting

U.S. EPA. (2016c). Non-confidential 2016 Chemical Data Reporting (CDR) Database [Website]. http://www.epa.gov/cdr/

U.S. EPA. (2016d). Public database 2016 chemical data reporting (May 2017 release). Washington, DC: US Environmental Protection Agency, Office of Pollution Prevention and Toxics. Retrieved from https://www.epa.gov/chemical-data-reporting

U.S. EPA. (2016e). Supplemental exposure and risk reduction technical report in support of risk management options for trichloroethylene (TCE) use in consumer aerosol degreasing. Washington D.C.: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0023

U.S. EPA. (2016f). Supplemental occupational exposure and risk reduction technical report in support of risk management options for trichloroethylene (TCE) use in aerosol degreasing. (RIN 2070-AK03). Washington D.C.: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0021

U.S. EPA. (2016g). Supplemental occupational exposure and risk reduction technical report in support of risk management options for trichloroethylene (TCE) use in spot cleaning. (RIN 2070-AK03). Washington D.C.: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0024

U.S. EPA. (2016h). Supplemental occupational exposure and risk reduction technical report in support of risk management options for trichloroethylene (TCE) use in vapor degreasing. (RIN 2070-AK11). Washington D.C.: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0387-0126

U.S. EPA. (2016i). Weight of evidence in ecological assessment [EPA Report]. (EPA100R16001). Washington, DC: Office of the Science Advisor. https://cfpub.epa.gov/si/si_public_record_report.cfm?dirEntryId=335523

U.S. EPA. (2017a). Chemical test rule data: Trichloroethylene. Washington, DC. Retrieved from http://java.epa.gov/chemview

U.S. EPA. (2017b). Consumer Exposure Model (CEM) version 2.0: User guide. U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. https://www.epa.gov/sites/production/files/2017-06/documents/cem_2.0_user_guide.pdf

U.S. EPA. (2017c). Preliminary information on manufacturing, processing, distribution, use, and disposal: Trichloroethylene [Comment]. (EPA-HQ-OPPT-2016-0737-003). Washington, DC: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0003

U.S. EPA. (2017d). Scope of the risk evaluation for trichloroethylene. CASRN: 79 - 01 - 6 [EPA Report]. (EPA-740-R1-7004). https://www.epa.gov/sites/production/files/2017-06/documents/tce_scope_06-22-17.pdf

U.S. EPA. (2017e). Strategy for conducting literature searches for trichloroethylene (TCE): Supplemental document to the TSCA Scope Document. CASRN: 79-01-6 [EPA Report]. https://www.epa.gov/sites/production/files/2017-06/documents/tce_lit_search_strategy_053017_0.pdf

U.S. EPA. (2017f). Toxics Release Inventory (TRI), reporting year 2015. Retrieved from https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools

U.S. EPA. (2017h). Trichloroethylene market and use report. Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention, Chemistry, Economics, and Sustainable Strategies Division. https://www.epa.gov/sites/production/files/2016-05/documents/instructions_for_reporting_2016_tsca_cdr_13may2016.pdf

U.S. EPA. (2017i). Tricholoroethylene (79 - 01 - 6) bibliography: Supplemental file for the TSCA Scope Document [EPA Report]. https://www.epa.gov/sites/production/files/2017-06/documents/tce_comp_bib.pdf

U.S. EPA. (2018a). 2014 National Emissions Inventory (NEI) data (2 ed.). Washington, DC. https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data

U.S. EPA. (2018b). Application of systematic review in TSCA risk evaluations. (740-P1-8001). Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. https://www.epa.gov/sites/production/files/2018-06/documents/final_application_of_sr_in_tsca_05-31-18.pdf

U.S. EPA. (2018c). ECOTOX user guide: ECOTOXicology database system. Version 5.0. https://cfpub.epa.gov/ecotox/

U.S. EPA. (2018d). Problem Formulation of the Risk Evaluation for Trichloroethylene. (EPA-740-R1-7014). Washington, DC: Office of Chemical Safety and Pollution Prevention, United States Environmental Protection Agency. https://www.epa.gov/sites/production/files/2018-06/documents/tce_problem_formulation_05-31-31.pdf

U.S. EPA. (2018e). Strategy for assessing data quality in TSCA risk evaluations. Washington DC: U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics.

U.S. EPA. (2019a). Consumer Exposure Model (CEM) 2.1 User Guide. (EPA Contract # EP-W-12-010). Washington, DC.

U.S. EPA. (2019b). Consumer Exposure Model (CEM) 2.1 User Guide - Appendices. (EPA Contract # EP-W-12-010). Washington, DC.

U.S. EPA. (2019c). Organic chemicals, plastics, and synthetic fibers. (40 CFR Part 414). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=5c5a19d4dd729db1e53fb9ca47e16706&mc=true&node=pt40.31.414&rgn=div5

U.S. EPA. (2019d). Biennial Review of 40 CFR Part 503 As Required Under the Clean Water Act Section 405(d)(2)(C). https://www.epa.gov/sites/production/files/2019-06/documents/2016-2017-biosolids-biennial-review.pdf

USGS. (2003). A national survey of methyl tert-butyl ether and other volatile organic compounds in drinking-water sources: Results of the random survey. Reston, VA: U.S. Department of the Interior, U.S. Geological Survey. https://pubs.er.usgs.gov/publication/wri024079

USGS. (2006). Water-quality conditions of Chester Creek, Anchorage, Alaska, 1998-2001. Reston, VA: U.S. Department of the Interior, U.S. Geological Survey. https://pubs.er.usgs.gov/publication/sir20065229

Van Raaij, MTM; Janssen, PAH; Piersma, AH. (2003). The relevance of developmental toxicity endpoints for acute limits settings (pp. 1-88). (RIVM Report 601900004). Nederlands: Nederlands National Institute for Public Health and the Environment. http://www2.epa.gov/sites/production/files/2014-04/documents/mtg35b.pdf

Vidal, M; Bassères, A; Narbonne, J. (2001). Potential biomarkers of trichloroethylene and toluene exposure in Corbicula fluminea. Environ Toxicol Pharmacol 9: 87-97. http://dx.doi.org/10.1016/S1382-6689(00)00068-5

Vlaanderen, J; Straif, K; Pukkala, E; Kauppinen, T; Kyyronen, P; Martinsen, J; Kjaerheim, K; Tryggvadottir, L; Hansen, J; Sparen, P, ar; Weiderpass, E. (2013). Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries. Occup Environ Med 70: 393-401. http://dx.doi.org/10.1136/oemed-2012-101188

Vogel, TM; McCarty, PL. (1985). Biotransformation of tetrachloroethylene to trichloroethylene, dichloroethylene, vinyl chloride, and carbon dioxide under methanogenic conditions. Appl Environ Microbiol 49: 1080-1083.

von Grote, J; Hürlimann, C; Scheringer, M; Hungerbühler, K. (2006). Assessing occupational exposure to perchloroethylene in dry cleaning. J Occup Environ Hyg 3: 606-619. http://dx.doi.org/10.1080/15459620600912173

Von Grote, J; Hurlimann, JC; Scheringer, M; Hungerbuhler, K. (2003). Reduction of Occupational Exposure to Perchloroethylene and Trichloroethylene in Metal Degreasing over the Last 30 years: Influence of Technology Innovation and Legislation. J Expo Anal Environ Epidemiol 13: 325-340. http://dx.doi.org/10.1038/sj.jea.7500288

Wallace, LA. (1987). The total exposure assessment methodology (TEAM) study: Summary and analysis: Volume I [EPA Report]. (EPA/600/6-87/002a). Washington, DC: U.S. Environmental Protection Agency; Office of Acid Deposition, Environmental Monitoring, and Quality Assurance.

Wang, R; Zhang, Y; Lan, Q; Holford, TR; Leaderer, B; Zahm, SH; Boyle, P; Dosemeci, M; Rothman, N; Zhu, Y; Qin, Q; Zheng, T. (2009). Occupational exposure to solvents and risk of non-Hodgkin lymphoma in Connecticut women. Am J Epidemiol 169: 176-185. http://dx.doi.org/10.1093/aje/kwn300

Ward, GS; Tolmsoff, AJ; Petrocelli, SR. (1986). ACUTE TOXICITY OF TRICHLOROETHYLENE TO SALTWATER ORGANISMS. Bull Environ Contam Toxicol 37: 830-836.

Weast, RC; Selby, SM. (1966). Ethene, trichloro. In CRC handbook of chemistry and physics. Cleaveland, OH: The Chemical Rubber Co.

Weed, DL. (2005). Weight of evidence: A review of concept and methods [Review]. Risk Anal 25: 1545-1557. http://dx.doi.org/10.1111/j.1539-6924.2005.00699.x

Whittaker, C; Rice, F; McKernan, L; Dankovic, D; Lentz, T; Macmahon, K; Kuempel, E; Zumwalde, R; Schulte, P. (2016). Current Intelligence Bulletin 68: NIOSH Chemical Carcinogen Policy. Whittaker, C; Rice, F; Mckernan, L; Dankovic, D; Lentz, T; Macmahon, K; Kuempel, E; Zumwalde, R; Schulte, P.

Whittaker, SG; Johanson, CA. (2011). A profile of the dry cleaning industry in King County, Washington: Final report. (LHWMP 0048). Seattle, WA: Local Hazardous Waste Management Program in King County. http://www.hazwastehelp.org/publications/publications_detail.aspx?DocID=Oh73%2fQilg9Q%3d

WHO. (1985). Environmental health criteria: Trichloroethylene. Geneva, Switzerland.

Wikoff, D; Urban, JD; Harvey, S; Haws, LC. (2018). Role of Risk of Bias in Systematic Review for Chemical Risk Assessment: A Case Study in Understanding the Relationship Between Congenital Heart Defects and Exposures to Trichloroethylene. Int J Toxicol 37: 125-143. http://dx.doi.org/10.1177/1091581818754330

Williams, FE; Sickelbaugh, TJ; Hassoun, E. (2006). Modulation by ellagic acid of DCA-induced developmental toxicity in the zebrafish (Danio rerio). J Biochem Mol Toxicol 20: 183-190. http://dx.doi.org/10.1002/jbt.20135

Wilmer, JW; Spencer, PJ; Ball, N; Bus, JS. (2014). Assessment of the genotoxicity of trichloroethylene in the in vivo micronucleus assay by inhalation exposure. Mutagenesis 29: 209-214. http://dx.doi.org/10.1093/mutage/geu006

Wirbisky, SE; Damayanti, N; Mahapatra, CT; Sepulveda, MS; Irudayaraj, J; Freeman, JL. (2016). Mitochondrial Dysfunction, Disruption of F-Actin Polymerization, and Transcriptomic Alterations in Zebrafish Larvae Exposed to Trichloroethylene. Chem Res Toxicol 29: 169-179. http://dx.doi.org/10.1021/acs.chemrestox.5b00402

Woolhiser, MR; Krieger, SM; Thomas, J; Hotchkiss, JA. (2006). Trichloroethylene (TCE): Immunotoxicity potential in CD rats following a 4-week vapor inhalation exposure. (031020). Midland, MI: Dow Chemical Company.

Wright, JM; Evans, A; Kaufman, JA; Rivera-Núñez, Z; Narotsky, MG. (2017). Disinfection by-product exposures and the risk of specific cardiac birth defects. Environ Health Perspect 125: 269-277. http://dx.doi.org/10.1289/EHP103

Xu, H; Tanphaichitr, N; Forkert, PG; Anupriwan, A; Weerachatyanukul, W; Vincent, R; Leader, A; Wade, MG. (2004). Exposure to trichloroethylene and its metabolites causes impairment of sperm fertilizing ability in mice. Toxicol Sci 82: 590-597. http://dx.doi.org/10.1093/toxsci/kfh277

Yauck, JS; Malloy, ME; Blair, K; Simpson, PM; Mccarver, DG. (2004). Proximity of residence to trichloroethylene-emitting sites and increased risk of offspring congenital heart defects among older women. Birth Defects Res A Clin Mol Teratol 70: 808-814. http://dx.doi.org/10.1002/bdra.20060

Yoshioka, Y; Ose, Y; Sato, T. (1986). Correlation of the five test methods to assess chemical toxicity and relation to physical properties. Ecotoxicol Environ Saf 12: 15-21.

Zeise, L; Wilson, R; Crouch, EA. (1987). Dose-response relationships for carcinogens: A review [Review]. Environ Health Perspect 73: 259-306.

Zhang, J. (2015). A review of spontaneous closure of ventricular septal defects. 28: 516-520.

Zhang, L; Bassig, BA; Mora, JL; Vermeulen, R; Ge, Y; Curry, JD; Hu, W; Shen, M; Qiu, C; Ji, Z; Reiss, B; Mchale, CM; Liu, S; Guo, W; Purdue, MP; Yue, F; Li, L; Smith, MT; Huang, H; Tang, X; Rothman, N; Lan, Q. (2013). Alterations in serum immunoglobulin levels in workers occupationally exposed to trichloroethylene. Carcinogenesis. http://dx.doi.org/10.1093/carcin/bgs403

Zhao, Y; Krishnadasan, A; Kennedy, N; Morgenstern, H; Ritz, B. (2005). Estimated effects of solvents and mineral oils on cancer incidence and mortality in a cohort of aerospace workers. Am J Ind Med 48: 249-258. http://dx.doi.org/10.1002/ajim.20216

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(a)	Provides EPA with the authority to prohibit or limit the manufacture (including import), processing, distribution in commerce, use or disposal of a chemical if EPA evaluates the risk and concludes that the chemical presents an unreasonable risk to human health or the environment.	Proposed rule under section 6 of TSCA to address the unreasonable risks presented by TCE use in vapor degreasing (82 FR 7432; January 19, 2017).
TSCA - Section 6(a)	Provides EPA with the authority to prohibit or limit the manufacture (including import), processing, distribution in commerce, use or disposal of a chemical if EPA evaluates the risk and concludes that the chemical presents an unreasonable risk to human health or the environment	Proposed rule under section 6 of TSCA to address the unreasonable risks presented by TCE use in commercial and consumer aerosol degreasing and for spot cleaning at dry cleaning facilities (81 FR 91592; December 16, 2016).
TSCA - Section 6(b)	Directs EPA to promulgate regulations to establish processes for prioritizing chemicals and conducting risk evaluations on priority chemicals. In the meantime, 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.	TCE is on the initial list of chemicals to be evaluated for unreasonable risks under TSCA (81 FR 91927, December 19, 2016).
TSCA - Section 5(a)	Once EPA determines that a use of a chemical substance is a significant new use under TSCA section 5(a), persons are required to submit a significant new use notice (SNUN) to EPA at least 90 days before they manufacture (including import) or process the chemical substance for that use.	Significant New Use Rule (SNUR) (81 FR 20535; April 8, 2016). TCE is subject to reporting under the SNUR for manufacture (including import) or processing of TCE for use in a consumer product except for use in cleaners and solvent degreasers, film cleaners, hoof polishes, lubricants, mirror

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
		edge sealants and pepper spray. This SNUR ensures that EPA will have the opportunity to review any new consumer uses of TCE and, if appropriate, take action to prohibit or limit those uses.
TSCA - Section 8(a)	The TSCA section 8(a) CDR rule requires manufacturers (including importers) to give EPA basic exposure-related information on the types, quantities and uses of chemical substances produced domestically and imported into the United States.	TCE manufacturing (including importing), processing and use information is reported under the CDR rule (76 FR 50816, August 16, 2011).
TSCA - Section 8(b)	EPA must compile, keep current and publish a list (the TSCA Inventory) of each chemical substance manufactured, processed or imported in the United States.	TCE was on the initial TSCA Inventory and was therefore not subject to EPA's new chemicals review process (60 FR 16309, March 29, 1995).
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.	28 substantial risk notifications received for TCE (U.S. EPA, ChemView. Accessed April 13, 2017).
TSCA - Section 4	Provides EPA with authority to issue rules and orders requiring manufacturers (including importers) and processors to test chemical substances and mixtures.	Seven studies received for TCE (U.S. EPA, ChemView. Accessed April 13, 2017).
Emergency Planning and Community Right-to-Know Act (EPCRA) - Section 313	Requires annual reporting from facilities in specific industry sectors that employ 10 or more full time equivalent employees and that manufacture, process, or otherwise use a TRI-listed chemical in quantities above threshold levels. A facility that meets reporting requirements must submit a reporting form for each chemical for which it triggered reporting, providing data across a variety of categories, including activities and uses of the chemical, releases and other waste management (e.g., quantities recycled, treated, combusted) and pollution prevention activities (under section 6607	TCE 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
	of the Pollution Prevention Act). These data include on- and off-site data as well as multimedia data (i.e., air, land and water).	
Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) - Section 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.	TCE is no longer used as an inert ingredient in pesticide products.
Clean Air Act (CAA) - Section 112(b)	Defines the original list of CAA hazardous air pollutants (HAPs). Under 112(c) of the CAA, EPA must identify and list source categories that emit HAPs 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 HAPs by adding or deleting a substance.	Lists TCE as a HAP (42 U.S.C. 7412(b)(1)).
CAA - Section 112(d)	Section 112(d) states that the EPA must establish a National Emission Standards for Hazardous Air Pollutants (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 EPA determines to be achievable by each particular source category. Different criteria for maximum achievable control technology (MACT) apply for new and existing sources. Less stringent standards, known as generally available	EPA has promulgated a number of NESHAP regulating industrial source categories that emit trichloroethylene and other HAP. These include, for example, the NESHAP for Halogenated Solvent Cleaning (59 FR 61801; December 2, 1994), among others.

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	control technology (GACT) standards, are allowed at the Administrator's discretion for area sources.	
CAA - Sections 112(d) and 112 (f)	Risk and technology review (RTR) of section 112(d) MACT standards. Section 112(f)(2) requires EPA to conduct risk assessments for each source category subject to section 112(d) MACT standards, and to determine if additional standards are needed to reduce remaining risks. Section 112(d)(6) requires EPA to review and revise the MACT standards, 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 Halogenated Solvent Cleaning (72 FR 25138; May 3, 2007) and will do so, as required, for the remaining source categories with NESHAP.
CWA – Sections 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. Regulations apply to existing and new sources.	TCE is designated as a toxic pollutant under section 307(a)(1) of the CWA and as such, is subject to effluent limitations.
CWA - Section 307(a)	Establishes a list of toxic pollutants or combination of pollutants under the to the CWA. The statute specifies a list of families of toxic pollutants also listed in 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 technology effluent limitations must be established on either a national basis through rules, or on a case-by-case best professional judgement basis in National Pollutant Discharge Elimination System (NPDES) permits.	
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	EPA issued drinking water standards for TCE pursuant to section 1412 of the SDWA. EPA promulgated the NPDWR for TCE

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	health of persons; 2. are known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern; and 3. in the sole judgement of the Administrator, regulation of the contaminant presents a meaningful opportunity for health risk reductions for persons served by public water systems. When EPA publishes an MCLG, EPA must also promulgate a National Primary Drinking Water Regulation (NPDWR) which includes either an enforceable maximum contaminant level (MCL), or a required treatment technique. Public water systems are required to comply with NPDWRs	in 1987 with a MCLG of zero an enforceable MCL of 0.005 mg/L (52 FR 25690, July 8, 1987).
RCRA - Section 3001	Directs EPA to develop and promulgate criteria for identifying the characteristics of hazardous waste, and for listing hazardous waste, taking into account toxicity, persistence, and degradability in nature, potential for accumulation in tissue and other related factors such as flammability, corrosiveness, and other hazardous characteristics.	TCE is included on the list of commercial chemical products, manufacturing chemical intermediates or off-specification commercial chemical products or manufacturing chemical intermediates that, when disposed (or when formulations containing any one of these as a sole active ingredient are disposed) unused, become hazardous wastes pursuant to RCRA 3001. RCRA Hazardous Waste Status: D040 at 0.5 mg/L; F001, F002; U228
Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) - Section 102(a)	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	TCE is a hazardous substance with a reportable quantity pursuant to section 102(a) of CERCLA (40 CFR 302.4) and EPA is actively overseeing cleanup of sites contaminated with TCE pursuant to the National Contingency Plan (NCP) (40 CFR 751).

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
	knowledge of a release of a hazardous substance above the reportable quantity threshold.	
Other Federal Regulations		
OSHA	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.	In 1971, OSHA issued occupational safety and health standards for TCE that included a Permissible Exposure Limit (PEL) of 100 ppm TWA, exposure monitoring, control measures and respiratory protection (29 CFR 1910.1000). While OSHA has established a PEL for TCE, OSHA has recognized that many of its permissible exposure limits (PELs) are outdated and inadequate for ensuring protection of worker health. Most of OSHA's PELs were issued shortly after adoption of the Occupational Safety and Health (OSH) Act in 1970, and have not been updated since that time. Section 6(a) of the OSH Act granted the Agency the authority to adopt existing Federal standards or national consensus standards as enforceable OSHA standards. For TCE, OSHA recommends the use of the NIOSH REL of 2 ppm (as a 60-minute ceiling) during the usage of TCE as an anesthetic agent and 25 ppm (as a 10-hour TWA) during all other exposures.
Atomic Energy Act	The Atomic Energy Act authorizes the Department of Energy to regulate the health and safety of its contractor employees	10 CFR 851.23, Worker Safety and Health Program, requires the use of the ACGIH TLVs if they are more protective than the OSHA PEL. The 2012 TLV for TCE is 10 ppm and the short-term limit is 25 ppm (ATSDR, 2019).
Federal Food, Drug, and Cosmetic Act (FFDCA)	Provides the FDA with authority to oversee the safety of food, drugs and cosmetics.	Tolerances are established for residues of TCE resulting from its use as a solvent in the manufacture

Statutes/Regulations	Description of Authority/Regulation	Description of Regulation
		of decaffeinated coffee and spice oleoresins (21 CFR 173.290).

A.2 State Laws and Regulations

Table_Apx A-2. State Laws and Regulations

State Actions	Description of Action
California Code of Regulations (CCR), Title 17, Section 94509(a)	Lists standards for VOCs for consumer products sold, supplied, offered for sale or manufactured for use in California. As part of that regulation, use of consumer general purpose degreaser products that contain TCE are banned in California and safer substitutes are in use (17 CCR, Section 94509(a).
State Permissible Exposure Limits (PELs)	Most states have set PELs identical to the OSHA 100 ppm 8-hour TWA PEL. Nine states have PELs of 50 ppm. California's PEL of 25 ppm is the most stringent (CCR, Title 8, Table AC-1).
VOC regulations for consumer products	Many states regulate TCE as a VOC. These regulations may set VOC limits for consumer products and/or ban the sale of certain consumer products as an ingredient and/or impurity. Regulated products vary from state to state, and could include contact and aerosol adhesives, aerosols, electronic cleaners, footwear or leather care products and general degreasers, among other products. California (Title 17, California Code of Regulations, Division 3, Chapter 1, Subchapter 8.5, Articles 1, 2, 3 and 4), Connecticut (R.C.S.A Sections 22a-174-40, 22a-174-41, and 22a-174-44), Delaware (Adm. Code Title 7, 1141), District of Columbia (Rules 20-720, 20-721, 20-735, 20-736, 20-737), Illinois (35 Adm Code 223), Indiana (326 IAC 8-15), Maine (Chapter 152 of the Maine Department of Environmental Protection Regulations), Maryland (COMAR 26.11.32.00 to 26.11.32.26), Michigan (R 336.1660 and R 336. 1661), New Hampshire (Env-A 4100) New Jersey (Title 7, Chapter 27, Subchapter 24), New York (6 CRR-NY III A 235), Rhode Island (Air Pollution Control Regulation No. 31) and Virginia (9VAC5 Chapter 45) all have VOC regulations or limits for consumer products. Some of these states also require emissions reporting.
Other	TCE is on California Proposition 65 List of chemicals known to cause cancer in 1988 or birth defects or other reproductive harm in 2014 (CCR Title 27, section 27001). TCE is on California's Safer Consumer Products Regulations Candidate List of chemicals that exhibit a hazard trait and are on an authoritative list (CCR Title 22, Chapter 55).

A.3 International Laws and Regulations

Table_Apx A-3. Regulatory Actions by Other Governments and Tribes

Country/ Organization	Requirements and Restrictions
Canada	TCE is on the Canadian List of Toxic Substances (CEPA 1999 Schedule 1). TCE 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 TCE into the environment from solvent degreasing facilities using more than 1000 kilograms of TCE per year. The regulation includes a market intervention by establishing tradable allowances for the use of TCE in solvent degreasing operations that exceed the 1000 kilograms threshold per year.
European Union	In 2011, TCE was added to Annex XIV (Authorisation list) of regulation (EC) No 1907/2006 - REACH (Registration, Evaluation, Authorization and Restriction of Chemicals). Entities that would like to use TCE needed to apply for authorization by October 2014, and those entities without an authorization must stop using TCE by April 2016. The European Chemicals Agency (ECHA) received 19 applications for authorization from entities interested in using TCE beyond April 2016. TCE is classified as a carcinogen category 1B, and was added to the EU REACH restriction of substances classified as carcinogen category 1A or 1B under the EU Classification and Labeling regulation (among other characteristics) in 2009. The restriction bans the placing on the market or use of TCE as substance, as constituent of other substances, or, in mixtures for supply to the general public when the individual concentration in the substance or mixture is equal to or greater than 0.1 % w/w (Regulation (EC) No 1907/2006 - REACH (Registration, Evaluation, Authorization and Restriction of Chemicals)). Previous regulations, such as the Solvent Emissions Directive (Directive 1999/13/EC) introduced stringent emission controls of TCE.
Australia	In 2000, TCE was assessed (National Industrial Chemicals Notification and Assessment Scheme, NICNAS (2000), *Trichloroethylene*. Accessed April, 18 2017).
Japan Chemical Substances Control Law	TCE 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 -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, 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 TCE (GESTIS International limit values for chemical agents (Occupational exposure limits, OELs) database. Accessed April 18, 2017).

Appendix B LIST OF SUPPLEMENTAL DOCUMENTS

List of supplemental documents (see Docket: EPA-HQ-OPPT-2019-0500 for access to all files):

Associated **Systematic Review Data Quality Evaluation and Data Extraction** Documents – Provides additional detail and information on individual study evaluations and data extractions including criteria and scoring results:

Physical/Chemical Properties, Fate and Transport

a. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Physical-Chemical Properties Studies*

b. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Fate and Transport Studies*

c. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Extraction for Environmental Fate and Transport Studies*

Occupational Exposures and Releases

d. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Data*

e. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Releases and Occupational Exposure Common Sources*

f. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: List of Key and Supporting Studies for Environmental Releases and Occupational Exposure*

Consumer and Environmental Exposures

g. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation for Data Sources on Consumer and Environmental Exposure*

h. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Extraction Tables for Environmental Monitoring Data*

i. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Extraction for Biomonitoring Data*

Environmental Hazard

j. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Environmental Hazard Studies*

k. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Extraction for Environmental Hazard Studies*

Human Health Hazard

l. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Animal and Mechanistic Data*

m. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies - Epidemiological Data*

n. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies*

o. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: Data Extraction for Human Health Hazard Studies*

p. *Risk Evaluation for Trichloroethylene, Systematic Review Supplemental File: List of Key and Supporting Studies for Human Health Hazard Assessment*

Associated **Supplemental Information Documents** – Provides additional details and information on exposure, hazard and risk assessments:

Occupational Exposures and Releases

q. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Environmental Releases and Occupational Exposure Assessment*

r. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Risk Calculator for Occupational Exposures*

Consumer and Environmental Exposures

s. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Aquatic Exposure Modeling Outputs from E-FAST*

t. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Consumer Exposure Assessment Model Input Parameters*

u. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Exposure Modeling Results and Risk Estimates for Consumer Inhalation Exposures*

v. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Exposure Modeling Results and Risk Estimates for Consumer Dermal Exposures*

Human Health

w. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Data Table for Congenital Heart Defects Weight of Evidence Analysis*

x. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Personal Communication to OPPT. Raw Data Values from Selgrade and Gilmour, 2010*

y. *Risk Evaluation for Trichloroethylene, Supplemental Information File: PBPK Model and ReadMe (zipped)*

Additional Information

z. *Risk Evaluation for Trichloroethylene, Supplemental Information File: Memorandum_NIOSH_BLS Respirator Usage in Private Sector Firms*

Appendix C ENVIRONMENTAL EXPOSURES

A break-out of facility-specific modeling results organized per OES, with predicted surface water concentrations and associated days of COC exceedance, are included in Table_Apx C-1. These facility-specific modeling results are utilized and discussed in environmental risk characterization presented in Section 4.1.2.

Table_Apx C-1. Facility-Specific Aquatic Exposure Modeling Results

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
OES: Manufacturing									
Axiall Corporation, Westlake, LA NPDES: LA0007129	Surface Water	NPDES LA0007129	Surface water	350	1.266	0.00156	0.0051	3	0
								788	0
								52,000	0
				20	22.150	0.0273	0.0897	3	0
								788	0
								52,000	0
Olin Blue Cube, Freeport, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.069	0.26	2.42	3	**37**
								788	0
								52,000	0
				20	1.200	4.51	42.14	3	11
								788	0
								52,000	0
Solvents & Chemicals, Pearland, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.0564	0.53	3	17
								788	0
								52,000	0
				20	0.265	1.01	9.48	3	5
								788	0
								52,000	0
	Surface Water	Organic Chemicals Manuf.	Surface water	350	0.015	0.30	2.77	3	**40**
								788	0
								52,000	0
				20	0.265	5.34	49.91	3	12
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Occidental Chemical Corp Wichita, Wichita, KS NPDES: KS0096903 and Organic Chem MFG SIC	Surface Water	Surrogate NPDES KS0043036	Surface water	350	0.015	0.02	0.07	3	0
								788	0
								52,000	0
				20	0.265	0.27	1.33	3	0
								788	0
								52,000	0
	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.0564	0.53	3	17
								788	0
								52,000	0
				20	0.265	1.01	9.48	3	5
								788	0
								52,000	0
OES: Processing as a Reactant									
440 unknown sites[8] NPDES: Not applicable	Off-site Waste-water Treatment	Organic Chemicals Manufacture	Surface water	350	0.005	0.0188	0.18	3	5
								788	0
								52,000	0
				20	0.089	0.33	3.13	3	2
								788	0
								52,000	0
	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.005	0.0989	0.92	3	**23**
								788	0
								52,000	0
				20	0.089	1.76	16.45	3	7
								788	0
								52,000	0
Arkema Inc. Calvert City, KY NPDES: KY0003603	Surface Water	NPDES KY0003603	Surface water	350	0.017	0.000197	0.000737	3	0
								788	0
								52,000	0
				20	0.295	0.00342	0.128	3	0
								788	0
								52,000	0
				350	0.0128	0.0000158		3	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Honeywell International - Geismar Complex, Geismar, LA NPDES: LA0006181	Surface Water	NPDES LA0006181	Surface water				0.0000518	788	0
								52,000	0
				20	0.224	0.000276	0.000907	3	0
								788	0
								52,000	0
Praxair Technology Center, Tonawanda, NY NPDES: NY0000281	Surface Water	NPDES NY0000281	Still body	350	0.00169	n/a	169.00	3	**350**
								788	0
								52,000	0
				20	0.030	n/a	3000.00	3	**20**
								788	**20**
								52,000	0
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, and Metalworking Fluids)									
Texas Instruments, Inc., Attleboro, MA NPDES: MA0001791	Surface Water	NPDES MA0001791	Surface water	260	0.005	0.00502	0.0188	3	0
								788	0
								52,000	0
				20	0.067	0.0673	0.25	3	0
								788	0
								52,000	0
Accellent Inc/Collegeville Microcoax, Collegeville, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.002	0.00711	0.0425	3	0
								788	0
								52,000	0
				20	0.029	0.10	0.62	3	0
								788	0
								52,000	0
Ametek Inc. U.S. Gauge Div., Sellersville, PA NPDES: PA0056014	Surface Water	Surrogate NPDES PA0020460	Surface water	260	0.001	0.0113	0.0619	3	0
								788	0
								52,000	0
				20	0.011	0.12	0.68	3	0
								788	0
								52,000	0
Atk-Allegany Ballistics Lab (Nirop),	Surface Water	NPDES WV0020371	Surface water	260	0.0005	0.000669	0.00311	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Keyser, WV NPDES: WV0020371								52,000	0
				20	0.0061	0.00803	0.0373	3	0
								788	0
								52,000	0
Handy & Harman Tube Co/East Norriton, Norristown, PA NPDES: PA0011436	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
US Nasa Michoud Assembly Facility, New Orleans, LA NPDES: LA0052256	Surface Water	Surrogate NPDES LA0003280	Still body	260	1.96	n/a	765.63	3	**260**
								788	0
								52,000	0
				20	25.44	n/a	9937.50	3	**20**
								788	**20**
								52,000	0
GM Components Holdings LLC, Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	260	0.13	3.14	10.97	3	**117**
								788	0
								52,000	0
				20	1.71	41.38	144.47	3	**20**
								788	0
								52,000	0
Akebono Elizabethtown Plant, Elizabethtown, KY NPDES: KY0089672	Surface Water	Surrogate NPDES KY0022039	Surface water	260	0.07	1.15	4.87	3	**27**
								788	0
								52,000	0
				20	0.897	14.77	62.38	3	16
								788	0
								52,000	0
Delphi Harrison Thermal Systems, Dayton, OH NPDES: OH0009431	Surface Water	NPDES OH0009431	Surface water	260	0.04	0.0175	0.0752	3	0
								788	0
								52,000	0
				20	0.465	0.20	0.87	3	0
								788	0
								52,000	0
Chemours Company Fc LLC,				260	0.03	0.000631	0.00301	3	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Washington, WV NPDES: WV0001279	Surface Water	NPDES WV0001279	Surface water					788	0
								52,000	0
				20	0.334	0.00703	0.0335	3	0
								788	0
								52,000	0
Equistar Chemicals Lp, La Porte, TX NPDES: TX0119792	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.02	0.46	2.22	3	**38**
								788	1
								52,000	0
				20	0.218	5.06	24.44	3	12
								788	1
								52,000	0
GE Aviation, Lynn, MA NPDES: MA0003905	Surface Water	NPDES MA0003905	Still water	260	0.01	n/a	0.0425	3	0
								788	0
								52,000	0
				20	0.128	n/a	0.54	3	0
								788	0
								52,000	0
Certa Vandalia LLC, Vandalia, OH NPDES: OH0122751	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.01	0.23	1.11	3	**28**
								788	0
								52,000	0
				20	0.107	2.46	11.89	3	9
								788	1
								52,000	0
GM Components Holdings LLC Kokomo Ops, Kokomo, IN NPDES: IN0001830	Surface Water	NPDES IN0001830	Surface water	260	0.01	0.0387	0.20	3	0
								788	0
								52,000	0
				20	0.086	0.33	1.73	3	0
								788	0
								52,000	0
Amphenol Corp-Aerospace Operations, Sidney, NY	Surface Water	NPDES NY0003824	Surface water	260	0.01	0.00882	0.0486	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
NPDES: NY0003824				20	0.082	0.0723	0.40	3	0
								788	0
								52,000	0
Emerson Power Trans Corp, Maysville, KY NPDES: KY0100196	Surface Water	Surrogate NPDES KY0020257	Surface water	260	0.01	0.000076	0.000400	3	3
								788	3
								52,000	3
				20	0.081	0.000995	0.00522	3	0
								788	0
								52,000	0
Olean Advanced Products, Olean, NY NPDES: NY0073547	Surface Water	Surrogate NPDES NY0027162	Surface water	260	0.01	0.00462	0.0188	3	0
								788	0
								52,000	0
				20	0.068	0.0314	0.13	3	0
								788	0
								52,000	0
Hollingsworth Saco Lowell, Easley, SC NPDES: SC0046396	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00469	0.11	0.52	3	**24**
								788	0
								52,000	0
				20	0.061	1.40	6.78	3	6
								788	1
								52,000	0
Trelleborg YSH Incorporated Sandusky Plant, Sandusky, MI NPDES: MI0028142	Surface Water	NPDES MI0028142	Surface water	260	0.00360	0.21	1.76	3	1
								788	0
								52,000	0
				20	0.047	2.69	23.04	3	4
								788	0
								52,000	0
Timken Us Corp Honea Path, Honea Path, SC NPDES: SC0047520	Surface Water	Surrogate NPDES SC0000698	Surface water	260	0.00355	0.20	1.06	3	2
								788	0
								52,000	0
				20	0.0462	2.63	13.77	3	5
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Johnson Controls Incorporated, Wichita, KS NPDES: KS0000850	Surface Water	NPDES KS0000850	Surface water	260	0.00228	0.0068	0.0548	3	0
								788	0
								52,000	0
				20	0.0296	0.0898	0.72	3	0
								788	0
								52,000	0
National Railroad Passenger Corporation (Amtrak) Wilmington Maintenance Facility, Wilmington, DE NPDES: DE0050962	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00203	0.0467	0.230	3	**21**
								788	0
								52,000	0
				20	0.026	0.60	2.89	3	3
								788	0
								52,000	0
Electrolux Home Products (Formerly Frigidaire), Greenville, MI NPDES: MI0002135	Surface Water	NPDES MI0002135	Surface water	260	0.00201	0.00644	0.0171	3	0
								788	0
								52,000	0
				20	0.026	0.0834	0.22	3	0
								788	0
								52,000	0
Rex Heat Treat Lansdale Inc, Lansdale, PA NPDES: PA0052965	Surface Water	Surrogate NPDES PA0026182	Surface water	260	0.00194	0.00896	0.0523	3	0
								788	0
								52,000	0
				20	0.025	0.12	0.67	3	0
								788	0
								52,000	0
Carrier Corporation, Syracuse, NY NPDES: NY0001163	Surface Water	NPDES NY0001163	Still water	260	0.00177	n/a	0.220	3	0
								788	0
								52,000	0
				20	0.023	n/a	2.84	3	0
								788	0
								52,000	0
Cascade Corp (0812100207),				260	0.00117	0.0269	0.130	3	18

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Springfield, OH NPDES: OH0085715	Surface Water	Primary Metal Forming Manuf.	Surface water					788	0
								52,000	0
				20	0.015	0.35	1.67	3	3
								788	0
								52,000	0
USAF-Wurtsmith Afb, Oscoda, MI NPDES: MI0042285	Surface Water	Surrogate NPDES MI0028282	Surface water	260	0.00115	0.000320	0.000753	3	0
								788	0
								52,000	0
				20	0.015	0.00417	0.00983	3	0
								788	0
								52,000	0
AAR Mobility Systems, Cadillac, MI NPDES: MI0002640	Surface Water	Surrogate NPDES MI0020257	Surface water	260	0.00112	0.00413	0.00916	3	0
								788	0
								52,000	0
				20	0.014	0.0517	0.11	3	0
								788	0
								52,000	0
Eaton Mdh Company Inc, Kearney, NE NPDES: NE0114405	Surface Water	Surrogate NPDES NE0052647	Still water	260	0.00107	n/a	0.130	3	0
								788	0
								52,000	0
				20	0.014	n/a	1.69	3	0
								788	0
								52,000	0
Lake Region Medical, Trappe, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.000500	0.00178	0.0106	3	0
								788	0
								52,000	0
				20	0.007	0.0249	0.15	3	0
								788	0
								52,000	0
Motor Components L L C, Elmira, NY NPDES: NY0004081	Surface Water	NPDES NY0004081	Surface water	260	0.00096	0.0143	0.0618	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
				20	0.0125	0.19	0.83	3	0
								788	0
								52,000	0
Salem Tube Mfg, Greenville, PA NPDES: PA0221244	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000897	0.0206	0.0997	3	17
								788	0
								52,000	0
				20	0.012	0.28	1.33	3	2
								788	0
								52,000	0
GE (Greenville) Gas Turbines LLC, Greenville, SC NPDES: SC0003484	Surface Water	NPDES SC0003484	Surface water	260	0.000806	0.0378	0.0821	3	0
								788	0
								52,000	0
				20	0.010	0.47	1.02	3	0
								788	0
								52,000	0
Parker Hannifin Corporation, Waverly, OH NPDES: OH0104132	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000747	0.0172	0.0830	3	16
								788	0
								52,000	0
				20	0.010	0.23	1.11	3	2
								788	0
								52,000	0
Mahle Engine Components Usa Inc, Muskegon, MI NPDES: MI0004057	Surface Water	NPDES MI0004057	Surface water	260	0.000742	0.00808	0.0336	3	0
								788	0
								52,000	0
				20	0.010	0.11	0.45	3	0
								788	0
								52,000	0
General Electric Company - Waynesboro, Waynesboro, VA NPDES: VA0002402	Surface Water	NPDES VA0002402	Surface water	260	0.000733	0.00241	0.00705	3	0
								788	0
								52,000	0
				20	0.010	0.0329	0.0962	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Globe Engineering Co Inc, Wichita, KS NPDES: KS0086703	Surface Water	Surrogate NPDES KS0043036	Surface water	260	0.00173	0.00175	0.00853	3	0
								788	0
								52,000	0
				20	0.023	0.0232	0.110	3	0
								788	0
								52,000	0
Gayston Corp, Dayton, OH NPDES: OH0127043	Surface Water	Surrogate NPDES OH0024881	Surface water	260	0.000643	0.000281	0.00121	3	0
								788	0
								52,000	0
				20	0.008	0.0035	0.0150	3	0
								788	0
								52,000	0
Styrolution America LLC, Channahon, IL NPDES: IL0001619	Surface Water	NPDES IL0001619	Surface water	260	0.000637	0.0000845	0.000221	3	0
								788	0
								52,000	0
				20	0.008	0.00106	0.00278	3	0
								788	0
								52,000	0
Remington Arms Co Inc, Ilion, NY NPDES: NY0005282	Surface Water	NPDES NY0005282	Surface water	260	0.000612	0.000291	0.000799	3	0
								788	0
								52,000	0
				20	0.008	0.00380	0.0104	3	0
								788	0
								52,000	0
United Technologies Corporation, Pratt And Whitney Division, East Hartford, CT NPDES: CT0001376	Surface Water	NPDES CT0001376	Surface water	260	0.000480	0.0000218	0.0000822	3	0
								788	0
								52,000	0
				20	0.006	0.000273	0.00103	3	0
								788	0
								52,000	0
				260	0.000470	0.000629	0.00292	3	0

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Atk-Allegany Ballistics Lab (Nirop), Keyser, WV NPDES: WV0020371	Surface Water	NPDES WV0020371	Surface water					788	0
								52,000	0
				20	0.006	0.00803	0.0373	3	0
								788	0
								52,000	0
Sperry & Rice Manufacturing Co LLC, Brookville, IN NPDES: IN0001473	Surface Water	NPDES IN0001473	Surface water	260	0.000328	0.00117	0.00569	3	0
								788	0
								52,000	0
				20	0.004	0.0143	0.0694	3	0
								788	0
								52,000	0
Owt Industries, Pickens, SC NPDES: SC0026492	Surface Water	NPDES SC0026492	Surface water	260	0.000314	0.000820	0.00213	3	0
								788	0
								52,000	0
				20	0.004	0.0104	0.0272	3	0
								788	0
								52,000	0
Boler Company, Hillsdale, MI NPDES: MI0053651	Surface Water	Surrogate NPDES MI0022136	Surface water	260	0.000269	0.00461	0.0204	3	0
								788	0
								52,000	0
				20	0.003	0.0514	0.23	3	0
								788	0
								52,000	0
Mccanna Inc., Carpentersville, IL NPDES: IL0071340	Surface Water	Surrogate NPDES IL0027944	Surface water	260	0.000268	0.000260	0.000911	3	0
								788	0
								52,000	0
				20	0.003	0.00291	0.0102	3	0
								788	0
								52,000	0
Cutler Hammer, Horseheads, NY NPDES: NY0246174	Surface Water	Surrogate NPDES NY0004081	Surface water	260	0.000238	0.00352	0.0153	3	0
								788	0
								52,000	0

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				20	0.003	0.0443	0.19	3	0
								788	0
								52,000	0
US Air Force Offutt Afb Ne, Offutt A F B, NE NPDES: NE0121789	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000159	0.00366	0.0177	3	5
								788	0
								52,000	0
				20	0.002	0.0460	0.22	3	2
								788	0
								52,000	0
Troxel Company, Moscow, TN NPDES: TN0000451	Surface Water	NPDES TN0000451	Surface water	260	0.000134	0.000254	0.000741	3	0
								788	0
								52,000	0
				20	0.002	0.00379	0.0111	3	0
								788	0
								52,000	0
Austin Tube Prod, Baldwin, MI NPDES: MI0054224	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000114	0.00262	0.0127	3	3
								788	0
								52,000	0
				20	0.001	0.023	0.11	3	1
								788	0
								52,000	0
LS Starrett Precision Tools, Athol, MA NPDES: MA0001350	Surface Water	NPDES MA0001350	Surface water	260	0.000102	0.000339	0.00153	3	0
								788	0
								52,000	0
				20	0.001	0.00333	0.015	3	0
								788	0
								52,000	0
Avx Corp, Raleigh, NC NPDES: NC0089494	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.0000883	0.00203	0.00981	3	2
								788	0
								52,000	0
				20	0.001	0.023	0.11	3	1
								788	0

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								52,000	0
Indian Head Division, Naval Surface Warfare Center, Indian Head, MD NPDES: MD0003158	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
General Dynamics Ordnance Tactical Systems, Red Lion, PA NPDES: PA0043672	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Trane Residential Solutions - Fort Smith, Fort Smith, AR NPDES: AR0052477	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Lexmark International Inc., Lexington, KY NPDES: KY0097624	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Alliant Techsystems Operations LLC, Elkton, MD NPDES: MD0000078	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Daikin Applied America, Inc. (Formally Mcquay International), Scottsboro, AL NPDES: AL0069701	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Beechcraft Corporation, Wichita, KS NPDES: KS0000183	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Federal-Mogul Corp, Scottsville, KY NPDES: KY0106585	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Cessna Aircraft Co (Pawnee Facility), Wichita, KS NPDES: KS0000647	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
N.G.I, Parkersburg, WV	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
NPDES: WV0003204									
Hyster-Yale Group, Inc, Sulligent, AL NPDES: AL0069787	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Hitachi Electronic Devices (Usa), Inc., Greenville, SC NPDES: SC0048411	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
OES: Spot Cleaning and Carpet Cleaning									
Boise State University, Boise, ID NPDES: IDG911006	Surface Water	Surrogate NPDES ID0023981	Surface water	300	0.00008	0.000205	0.00388	3	0
								788	0
								52,000	0
				20	0.001	0.00256	0.0485	3	0
								788	0
								52,000	0
Venetian Hotel And Casino, Las Vegas, NV NPDES: NV0022888	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
63,746 unknown sites NPDES: All POTW SIC	Surface Water or POTW	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
OES: Repackaging									
Hubbard-Hall Inc, Waterbury, CT NPDES: Unknown	Off-site Waste-water Treatment	Receiving Facility: Recycle Inc.; POTW (Ind.)	Surface water	250	1.108	5.33	27.18	3	**194**
								788	0
								52,000	0
				20	13.85	66.45	339.11	3	**20**
								788	1
								52,000	0
Oiltanking Houston Inc, Houston, TX NPDES: TX0091855	Surface Water	Surrogate NPDES TX0065943	Surface water	250	0.003	0.32	6.52	3	2
								788	0
								52,000	0
				20	0.041	4.36	89.13	3	4
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
St. Gabriel Terminal, Saint Gabriel, LA NPDES: LA0005487	Surface Water	NPDES LA0005487	Surface water	250	0.00550	0.00000677	0.0000223	3	0
								788	0
								52,000	0
				20	0.069	0.0000850	0.000279	3	0
								788	0
								52,000	0
Vopak Terminal Westwego Inc, Westwego, LA NPDES: LA0124583	Surface Water	Surrogate NPDES LA0042064	Surface water	250	0.00468	0.00000576	0.0000189	3	0
								788	0
								52,000	0
				20	0.058	0.0000714	0.000235	3	0
								788	0
								52,000	0
Research Solutions Group Inc, Pelham, AL NPDES: AL0074276	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Carlisle Engineered Products Inc, Middlefield, OH NPDES: OH0052370	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
OES: Process Solvent Recycling and Worker Handling of Wastes									
Clean Water Of New York Inc, Staten Island, NY NPDES: NY0200484	Surface Water	Surrogate NPDES NJ0000019	Still body	250	0.004	n/a	11.76	3	**250**
								788	0
								52,000	0
				20	0.047	n/a	138.24	3	**20**
								788	0
								52,000	0
Reserve Environmental Services, Ashtabula, OH NPDES: OH0098540	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Veolia Es Technical Solutions LLC, Middlesex, NJ NPDES: NJ0020141	Off-site Waste-water Treatment	Receiving Facility: Middlesex Cnty UA;	Still body	250	24.1	n/a	2.85	3	0
								788	0
								52,000	0
				20	301.78	n/a	35.72	3	**20**
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
		NPDES NJ0020141						52,000	0
Clean Harbors Deer Park LLC, La Porte, TX NPDES: TX0005941	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.35	1.68	8.57	3	**110**
								788	0
								52,000	0
				20	4.36	20.92	106.75	3	19
								788	0
								52,000	0
Clean Harbors El Dorado LLC, El Dorado, AR NPDES: AR0037800	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.04	0.19	0.98	3	6
								788	0
								52,000	0
				20	0.455	2.21	11.26	3	11
								788	0
								52,000	0
OES: Adhesives, Sealants, Paints, and Coatings									
Able Electropolishing Co Inc, Chicago, IL NPDES: Not available	POTW	Adhesives and Sealants Manuf.	Surface water	250	0.298	0.86	7.28	3	8
								788	0
								52,000	0
Garlock Sealing Technologies, Palmyra, NY NPDES: NY0000078	Surface Water	NPDES NY0000078	Surface water	250	0.00033	0.00252	0.00716	3	0
								788	0
								52,000	0
				20	0.00407	0.0312	0.0889	3	0
								788	0
								52,000	0
Ls Starrett Co, Athol, MA NPDES: MAR05B615	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Aerojet Rocketdyne[8], Inc., East Camden, AR NPDES: AR0051071, ARR00A521, ARR00A520	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Best One Tire & Service[8], Nashville, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Bridgestone Aircraft Tire (Usa), Inc. [8], Mayodan, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Clayton Homes Inc[8], Oxford, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Cmh Manufacturing, Inc. Dba Schult Homes - Plant 958[8], Richfield, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Delphi Thermal Systems[8], Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	250	0.013	0.31	1.10	3	2
								788	0
								52,000	0
				20	0.160	3.87	13.50	3	11
								788	0
								52,000	0
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Green Bay Packaging Inc - Coon Rapids[8], Coon Rapids, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Mastercraft Boat Company[8], Vonore, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Michelin Aircraft Tire Company[8], Norwood, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
M-Tek, Inc[8], Manchester, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Olin Corp[8], East Alton, IL NPDES: IL0000230	Surface Water	NPDES IL0000230	Surface water	250	0.013	0.08	0.18	3	0
								788	0
								52,000	0
				20	0.160	1.03	2.26	3	7
								788	0
								52,000	0
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Parker Hannifin Corp – Paraflex Division[8], Manitowoc, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Parrish Tire Company[8], Yadkinville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Republic Doors And Frames[8], Mckenzie, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Ro-Lab Rubber Company Inc.[8], Tracy, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Royale Comfort Seating, Inc. [8] - Plant No. 1, Taylorsville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Snider Tire, Inc. [8], Statesville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Snyder Paper Corporation[8], Hickory, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Stellana Us[8], Lake Geneva, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Thomas Built Buses - Courtesy Road[8], High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Unicel Corp[8], Escondido, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Acme Finishing Co Llc[8], Elk Grove Village, IL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
Aerojet Rocketdyne, Inc.[8], Rancho Cordova, CA NPDES: CA0004111	Surface Water	NPDES CA0004111	Surface water	250	0.013	0.000295	0.000818	3	0
								788	0
								52,000	0
				20	0.160	0.00363	0.0101	3	0
								788	0
								52,000	0
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.0374000	0.320000	3	0
								788	0
								52,000	0
Allegheny Cnty Airport Auth/ Pgh Intl Airport[8], Coroapolis Pittsburgh, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Amphenol Corp – Aerospace Operations[8], Sidney, NY NPDES: NY0003824	Surface Water	NPDES NY0003824	Surface water	250	0.013	0.0115	0.0631	3	0
								788	0
								52,000	0
				20	0.160	0.14	0.78	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.03740	0.3200	3	0
								788	0
								52,000	0
Aprotech Powertrain[8], Asheville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Coating & Converting Tech Corp/ Adhesive Coatings[8], Philadelphia, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Corpus Christi Army Depot[8], Corpus Christi, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Electronic Data Systems Camp Pendleton[8], Camp Pendleton, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Florida Production Engineering, Inc. [8], Ormond Beach, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Goodrich Corporation[8], Jacksonville, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Kasai North America Inc[8], Madison Plant, Madison, MS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Kirtland Air Force Base[8], Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Marvin Windows & Doors[8], Warroad, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
Mcneilus Truck & Manufacturing Inc[8], Dodge Center, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Metal Finishing Co. [8] – Wichita (S Mclean Blvd), Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Murakami Manufacturing Usa Inc[8], Campbellsville, KY NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Peterbilt Motors Denton Facility[8], Denton, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Portsmouth Naval Shipyard[8], Kittery, ME NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
R.D. Henry & Co.[8], Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Raytheon Company[8], Portsmouth, RI NPDES: RI0000281	Surface Water	NPDES RI0000281	Still body	250	0.013	n/a	10.83	3	**250**
								788	0
								52,000	0
				20	0.160	n/a	133.33	3	**20**
								788	0
								52,000	0
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.03740	0.32	3	0
								788	0
								52,000	0
Rehau Inc[8], Cullman, AL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Rotochopper Inc[8], Saint Martin, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Rubber Applications[8], Mulberry, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Sapa Precision Tubing Rockledge, Llc[8], Rockledge, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Thomas & Betts[8], Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Thomas Built Buses - Fairfield Road[8], High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Timco, Dba Haeco Americas Airframe Services[8], Greensboro, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Trelleborg Coated Systems Us, Inc[8] – Grace Advanced Materials, Rutherfordton, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
				250	0.013	0.20	1.67	3	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
U.S. Coast Guard Yard - Curtis Bay[8], Curtis Bay, MD NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water					788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
Viracon Inc[8], Owatonna, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	0.20	1.67	3	0
								788	0
								52,000	0
				20	0.160	2.42	20.57	3	3
								788	0
								52,000	0
	POTW			250	0.013	0.0374	0.32	3	0
								788	0
								52,000	0
OES: Industrial Processing Aid									
Occidental Chemical Corp Niagara Plant, Niagara Falls, NY NPDES: NY0003336	Surface Water	NPDES NY0003336	Still body	300	0.019	n/a	0.14	3	0
								788	0
								52,000	0
				20	0.292	n/a	2.200	3	0
								788	0
								52,000	0
Stepan Co Millsdale Road, Elwood, IL NPDES: IL0002453	Surface Water	NPDES IL0002453	Surface water	300	0.001	0.00016	0.000419	3	0
								788	0
								52,000	0
				20	0.008	0.00128	0.00335	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Entek International LLC, Lebanon, OR NPDES: N/A	Off-site Waste-water Treatment	No info on receiving facility; POTW (Ind.)	Surface water	300	0.38	1.82	9.30	3	**140**
								788	0
								52,000	0
				20	5.65	27.11	138.34	3	**20**
								788	0
								52,000	0
National Electrical Carbon Products Dba Morgan Adv Materials, Fostoria, OH NPDES: OH0052744	Off-site Waste-water Treatment	Receiving Facility: City of Fostoria; NPDES OH0052744	Surface water	300	0.008	0.0336	0.15	3	0
								788	0
								52,000	0
				20	0.115	0.50	2.32	3	1
								788	0
								52,000	0
PPG Industries Inc Barberton, Barberton, OH NPDES: OH0024007	Off-site Waste-water Treatment	Receiving Facility: City of Barberton; NPDES OH0024007	Surface water	300	0.005	0.00478	0.0141	3	0
								788	0
								52,000	0
				20	0.070	0.067	0.20	3	0
								788	0
								52,000	0
Daramic LLC, Corydon, IN NPDES: IN0020893	Surface Water	NPDES IN0020893	Surface water	300	0.008	0.00572	0.0206	3	0
								788	0
								52,000	0
				20	0.114	0.0816	0.29	3	0
								788	0
								52,000	0
OES: Commercial Printing and Copying									
Printing And Pub Sys Div, Weatherford, OK NPDES: OK0041785	Surface Water	Printing	Surface water	250	0.00020	0.000662	0.00292	3	0
								788	0
								52,000	0
				20	0.00250	0.00827	0.0365	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
OES: Other Industrial Uses									
Eli Lilly And Company-Lilly Tech Ctr, Indianapolis, IN NPDES: IN0003310	Surface Water	NPDES IN0003310	Surface water	250	1.553	1.63	9.03	3	**35**
								788	0
								52,000	0
				20	19.410	20.47	113.09	3	17
								788	0
								52,000	0
Oxy Vinyls LP - Deer Park Pvc, Deer Park, TX NPDES: TX0007412	Surface Water	NPDES TX0007412	Surface water	250	0.148	0.13	0.49	3	1
								788	0
								52,000	0
				20	1.854	1.58	5.98	3	9
								788	0
								52,000	0
Washington Penn Plastics, Frankfort, KY NPDES: KY0097497	Surface Water	Surrogate NPDES KY0028410	Surface water	250	0.032	1.25	7.53	3	**22**
								788	0
								52,000	0
				20	0.399	15.62	94.12	3	13
								788	0
								52,000	0
Natrium Plant, New Martinsville, WV NPDES: WV0004359	Surface Water	NPDES WV0004359	Surface water	250	0.022	0.000566	0.00262	3	0
								788	0
								52,000	0
				20	0.274	0.00695	0.0322	3	0
								788	0
								52,000	0
Leroy Quarry, Leroy, NY NPDES: NY0247189	Surface Water	Surrogate NPDES NY0030546	Surface water	250	0.019	0.16	0.71	3	0
								788	0
								52,000	0
				20	0.242	2.05	8.91	3	3
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
George C Marshall Space Flight Center, Huntsville, AL NPDES: AL0000221	Surface Water	Surrogate NPDES AL0025585	Surface water	250	0.010	0.0738	0.20	3	0
								788	0
								52,000	0
				20	0.128	0.96	2.63	3	8
								788	0
								52,000	0
Whelan Energy Center Power Plant, Hastings, NE NPDES: NE0113506	Surface Water	NPDES NE0113506	Surface water	250	0.009	0.67	2.92	3	**30**
								788	0
								52,000	0
				20	0.118	8.95	38.96	3	13
								788	0
								52,000	0
Army Cold Regions Research & Engineering Lab, Hanover, NH NPDES: NH0001619	Surface Water	Surrogate NPDES NH0100099	Surface water	250	0.0002	0.0000266	0.000103	3	0
								788	0
								52,000	0
				20	0.0029	0.000398	0.00154	3	0
								788	0
								52,000	0
Corning - Canton Plant, Canton, NY NPDES: NY0085006	Surface Water	Surrogate NPDES NY0034762	Surface water	250	0.0002	0.000101	0.000340	3	0
								788	0
								52,000	0
				20	0.0028	0.00152	0.00510	3	0
								788	0
								52,000	0
Ames Rubber Corp Plant #1, Hamburg Boro, NJ NPDES: NJ0000141	Surface Water	Surrogate NPDES NJ0000141[i]	Surface water	250	0.00011	0.00258	0.0149	3	**53[i]**
								788	**50[i]**
								52,000	**50[i]**
				20	0.00133	0.0304	0.18	3	6
								788	4
								52,000	4
Gorham, Providence, RI	Surface Water	POTW (Ind.)	Surface water	250	0.0001	0.00253	0.0129	3	0
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
NPDES: RIG85E004								52,000	0
				20	0.0012	0.0253	0.13	3	0
								788	0
								52,000	0
Solvay - Houston Plant, Houston, TX NPDES: TX0007072	Surface Water	NPDES TX0007072	Surface water	350	0.024	0.22	4.44	3	3
								788	0
								52,000	0
				20	0.414	3.72	75.93	3	5
								788	0
								52,000	0
Akzo Nobel Surface Chemistry LLC, Morris, IL NPDES: IL0026069	Surface Water	NPDES IL0026069	Surface water	350	0.000329	0.000300	0.000688	3	0
								788	0
								52,000	0
				20	0.006	0.00546	0.0125	3	0
								788	0
								52,000	0
Solutia Nitro Site, Nitro, WV NPDES: WV0116181	Surface Water	Surrogate NPDES WV0023229	Surface water	350	0.000318	0.0000214	0.0000941	3	0
								788	0
								52,000	0
				20	0.006	0.000401	0.00176	3	0
								788	0
								52,000	0
Amphenol Corporation - Columbia, Columbia, SC NPDES: SC0046264	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.000202	0.00395	0.037	3	0
								788	0
								52,000	0
				20	0.004	0.0791	0.74	3	1
								788	0
								52,000	0
Keeshan and Bost Chemical Co., Inc., Manvel, TX NPDES: TX0072168	Surface Water	NPDES TX0072168	Still body	350	0.000095	n/a	9.50	3	**350**
								788	0
								52,000	0
				20	0.002	n/a	200.00	3	**20**

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								788	0
								52,000	0
Chemtura North and South Plants, Morgantown, WV NPDES: WV0004740	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Indorama Ventures Olefins, LLC, Sulphur, LA NPDES: LA0069850	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Emerson Power Transmission, Ithaca, NY NPDES: NY0002933	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
William E. Warne Power Plant, Los Angeles County, CA NPDES: CA0059188	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Raytheon Aircraft Co(Was Beech Aircraft), Boulder, CO NPDES: COG315176	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
OES: Other Commercial Uses									
Corning Hospital, Corning, NY NPDES: NY0246701	Surface Water	Surrogate NPDES NY0025721	Surface water	250	0.013	0.00597	0.0271	3	0
								788	0
								52,000	0
				20	0.159	0.0735	0.33	3	0
								788	0
								52,000	0
Water Street Commercial Bldg, Dayton, OH NPDES: OH0141496	Surface Water	Surrogate NPDES OH0009521	Surface water	250	0.003	0.00131	0.00564	3	0
								788	0
								52,000	0
				20	0.035	0.0153	0.0658	3	0
								788	0
								52,000	0
				250	0.00040	0.0196	0.0881	3	**213**[j]

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
Union Station North Wing Office Building, Denver, CO NPDES: COG315293	Surface Water	Surrogate NPDES CO0020095[j]	Surface water					788	**213**[j]
								52,000	**213**[j]
				20	0.00499	0.24	1.10	3	18
								788	17
								52,000	17
Confluence Park Apartments, Denver, CO NPDES: COG315339	Surface Water	Surrogate NPDES CO0020095[j]	Surface water	250	0.00028	0.0137	0.0617	3	**213**[j]
								788	**213**[j]
								52,000	**213**[j]
				20	0.00354	0.17	0.77	3	17
								788	17
								52,000	17
Park Place Mixed Use Development, Annapolis, MD NPDES: MD0068861	Surface Water	Surrogate NPDES MD0052868	Still body	250	0.00027	n/a	9.00	3	**250**
								788	0
								52,000	0
				20	0.00334	n/a	110.00	3	**20**
								788	0
								52,000	0
Tree Top Inc Wenatchee Plant, Wenatchee, WA NPDES: WA0051527	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Wynkoop Denver LLCP St, Denver, CO NPDES: COG603115	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
Greer Family Llc, South Burlington, VT NPDES: VT0001376	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
John Marshall III Site, Mclean, VA NPDES: VA0090093	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.							
OES: N/A (WWTP)									
New Rochelle STP, New Rochelle, NY NPDES: NY0026697	Surface Water	NPDES NY0026697	Still body	365	0.043	n/a	0.70	3	0
								788	0
								52,000	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
				20	0.786	n/a	12.79	3	**20**
								788	0
								52,000	0
Everett Water Pollution Control Facility, Everett, WA NPDES: WA0024490	Surface Water	NPDES WA0024490	Surface water	365	0.016	0.13	0.17	3	0
								788	0
								52,000	0
				20	0.299	2.37	3.11	3	7
								788	0
								52,000	0
Sullivan WWTP, Sullivan, MO NPDES: MO0104736	Surface Water	NPDES MO0104736	Surface water	365	0.010	0.16	0.61	3	2
								788	0
								52,000	0
				20	0.176	2.81	10.97	3	7
								788	0
								52,000	0
Sunnyside STP, Sunnyside, WA NPDES: WA0020991	Surface Water	NPDES WA0020991	Surface water	365	0.005	0.00146	0.00673	3	0
								788	0
								52,000	0
				20	0.083	0.0242	0.110	3	0
								788	0
								52,000	0
Port Of Sunnyside Industrial WWTF, Sunnyside, WA NPDES: WA0052426	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.0505	0.26	3	0
								788	0
								52,000	0
				20	0.035	0.88	4.51	3	5
								788	0
								52,000	0
U.S. Air Force Shaw AFB SC, Shaw AFB, SC NPDES: SC0024970	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.0505	0.26	3	0
								788	0
								52,000	0
				20	0.032	0.81	4.12	3	4
								788	0

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
								52,000	0
Gnf-A Wilmington-Castle Hayne WWTP, Wilmington, NC NPDES: NC0001228	Surface Water	NPDES NC0001228	Surface water	365	0.0004	0.000304	0.00194	3	0
								788	0
								52,000	0
				20	0.0067	0.00533	0.0340	3	0
								788	0
								52,000	0
Cameron Trading Post WWTP, Cameron, AZ NPDES: NN0021610	Surface Water	POTW (Ind.)	Surface water	365	0.0003	0.00758	0.0387	3	0
								788	0
								52,000	0
				20	0.0047	0.13	0.64	3	0
								788	0
								52,000	0
Coal Grove WWTP, Coal Grove, OH NPDES: OH0104558	Surface Water	NPDES OH0029432	Surface water	365	0.0002	0.00000250	0.00001 27	3	0
								788	0
								52,000	0
				20	0.0031	0.0000375	0.00019	3	0
								788	0
								52,000	0

[1] Release media are either direct (release from 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 81% is applied to all indirect releases.
[2] If a valid NPDES of facility was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location discharging into the same water body) or a representative generic industry sector.
[3] EFAST uses ether the "surface water" model, for rivers and streams, or the "still water" model, for lakes, bays, and oceans.
[4] Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.
[5] The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.
[6] For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.
[7] To determine the PDM days of exceedance for still bodies of water, the release days provided by the EPA Engineers is equal to the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.
[8] Predicted water releases for the indicated sites changed slightly between modeling and publication of the draft risk evaluation. For the 440 unknown sites in the Processing as a Reactant OES changed from 1.75 kg/yr to 2.2 kg/yr. For the sites listed under the Adhesives, Sealants, Paints, and Coatings OES, annual release predictions changed from 3.25 kg/yr to 4.4 kg/yr. These slight differences (i.e., between 0.5 to 1.2 kg/yr) are unlikely to impact risk characterization.

Name, Location, and ID of Active Releaser Facility	Release Media[1]	Modeled Facility or Industry Sector in EFAST[2]	EFAST Waterbody Type[3]	Days of Release[4]	Release[5] (kg/day)	Harmonic Mean SWC (ppb)	7Q10 SWC[6] (ppb)	COC (ppb)	Days of Exceedance[7] (days/yr)
[9] The predicted days of exceedance are presented although the estimated 7Q10 never approaches the lowest COC due to the fact that the EFAST database has minimum stream flow of 0 MLD and a mean stream flow of 2.69 MLD for this site. Therefore, these days of exceedances were not considered in environmental risk characterization. [10] The predicted days of exceedance are presented although the estimated 7Q10 never approaches the lowest COC due to the fact that the EFAST database has minimum stream flow of 0 MLD and a mean stream flow of 0 MLD for this site. Therefore, these days of exceedances were not considered in environmental risk characterization.									

Appendix D CONSUMER EXPOSURES

D.1 Model Sensitivity

The CEM developers conducted a detailed sensitivity analysis for CEM, as described in Appendix C of the CEM User Guide (U.S. EPA, 2019b). The CEM developers included results of model corroboration analysis in Appendix D of the CEM User Guide (U.S. EPA, 2019b).

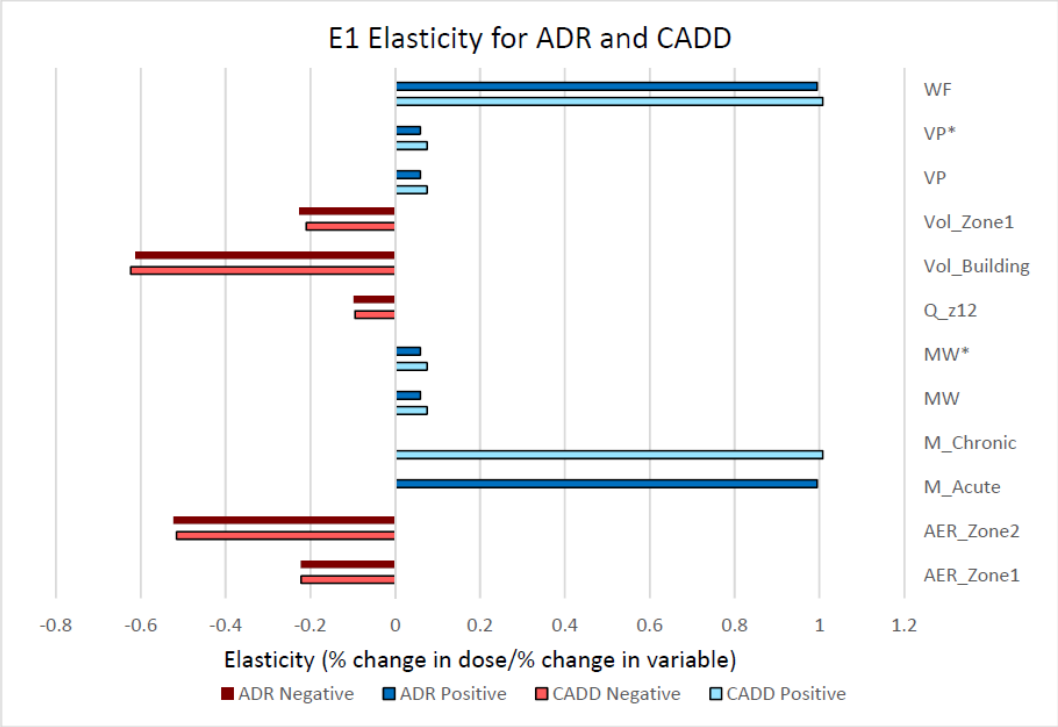
In brief, the analysis was conducted on continuous variables and categorical variables that were used in CEM emission or dermal models. A base run of different CEM models using various product or article categories, along with CEM defaults, was used. Individual variables were modified, one at a time, and the resulting Acute Dose Rate (ADR) and Chronic Average Daily Dose (CADD) were compared to the corresponding results for the base run. Benzyl alcohol, a VOC, was used as an example for product models such as those applied in this evaluation of TCE.

The tested model parameters were increased by 10%. The measure of sensitivity for continuous variables such as mass of product used, weight fraction, and air exchange rate was "elasticity," defined as the ratio of percent change in each result to the corresponding percent change in model input. A positive elasticity indicates that an increase in the model parameter resulted in an increase in the model output, whereas a parameter with negative elasticity is associated with a decrease in the model output. For categorical variables such as receptor activity pattern (i.e., work schedule) and room of use, the percent difference in model outputs for different category pairs was used as the measure of sensitivity.

The results are summarized below for the inhalation and dermal models used to evaluate consumer exposures to TCE (i.e., emission models E1 and E3 and the dermal permeability model P_DER2b. For full results and additional background, refer to Appendix C of the CEM User Guide (U.S. EPA, 2017b).

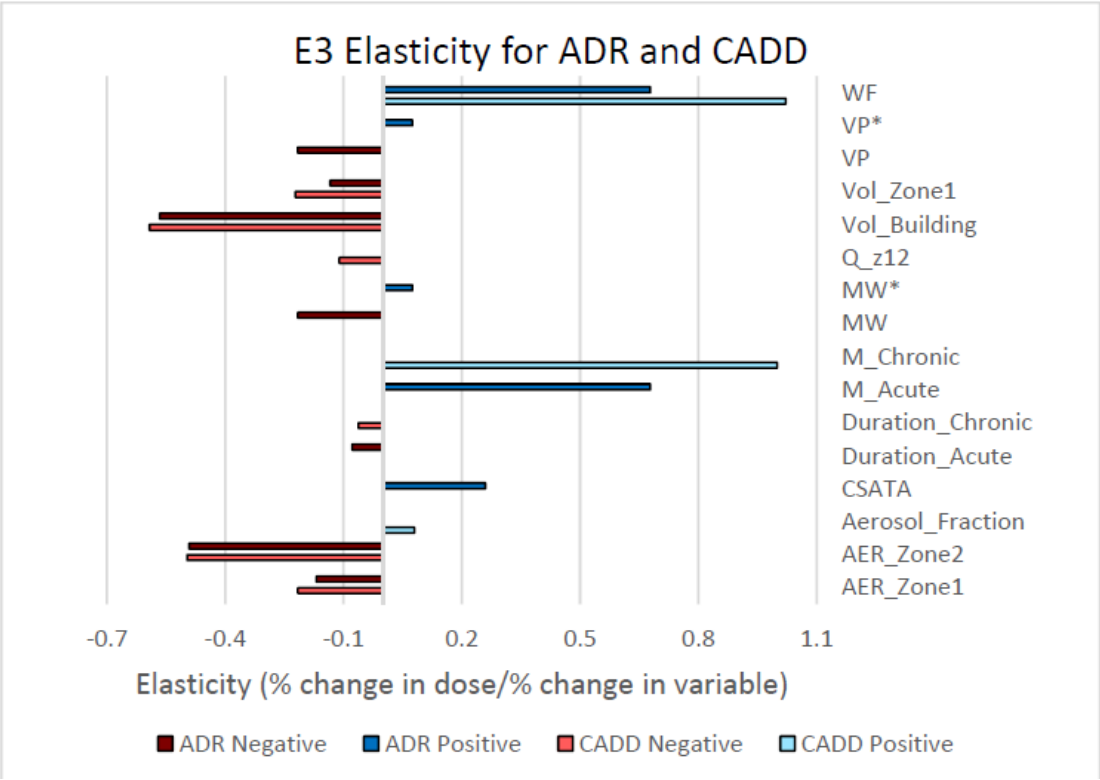
D.1.1 Continuous Variables

For acute exposures generated from emission model E1, WF (weight fraction) and M_acute (mass of product used) have the greatest positive elasticities of the tested parameters (see Figure_Apx D-1). The next most sensitive parameters demonstrate negative elasticity and include: Vol_Building (building volume); AER_Zone2 (air exchange rate in Zone 2); AER_Zone1 (air exchange rate in Zone 1); Vol_Zone1 (room of use, or Zone 1 volume). Inhalation exposures from liquid consumer product formulations were modeled using E1 and the two most sensitive variables identified in this analysis were varied to estimate a range of exposures.



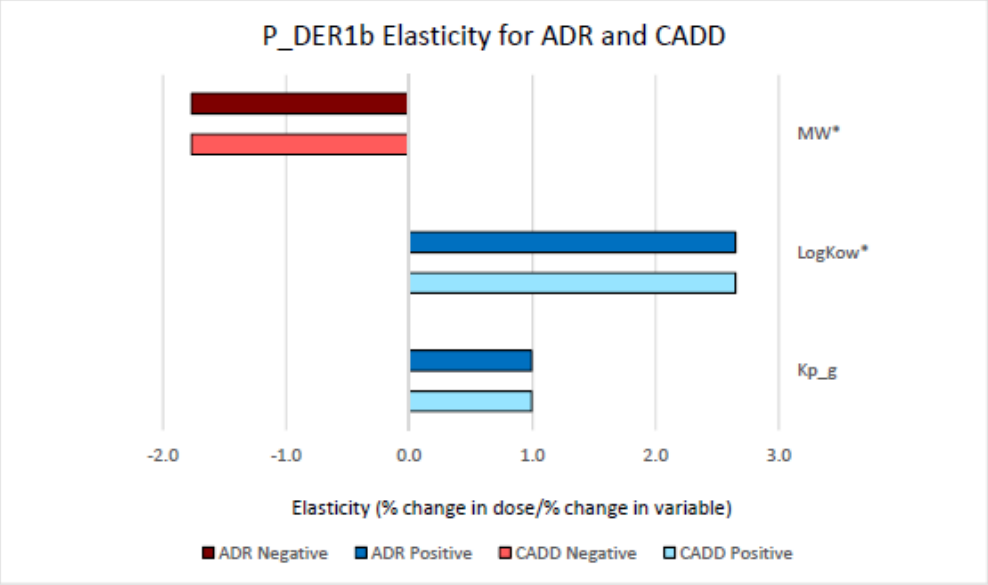
Figure_Apx D-1. Elasticities (≥ 0.05) for Parameters Applied in E1

For acute exposures generated from emission model E3, WF (weight fraction) and M_acute (mass of product used) have the greatest positive elasticities of the tested parameters (see Figure_Apx D-2). The next most sensitive parameters demonstrate negative elasticity and include: Vol_Building (building volume); AER_Zone2 (air exchange rate in Zone 2); MW (molecular weight); VP (vapor pressure); AER_Zone1 (air exchange rate in Zone 1); Vol_Zone1 (room of use, or Zone 1 volume). Inhalation exposures from aerosol or spray consumer product formulations were modeled using E3 and the two most sensitive variables identified in this analysis were varied to estimate a range of exposures.



Figure_Apx D-2. Elasticities (≥ 0.05) for Parameters Applied in E3

For acute exposures generated from emission model P_DER2b, the chemical properties that inform absorption rate, or absorption rate estimates, have the greatest elasticities (see Figure_Apx D-3). Dermal exposures from consumer product formulations were modeled using P_DER2B with a measured Kp (permeability coefficient). Therefore, $LogK_{OW}$ (octanol/water partition coefficient) and MW (molecular weight) were not used to estimate skin penetration.



Figure_Apx D-3. Elasticities (≥ 0.05) for Parameters Applied in P_DER2b

D.1.2 Categorical Variables

For categorical variables there were multiple parameters that affected other model inputs. For example, varying the room type changed the ventilation rates, volume size and the amount of time per day that a person spent in the room. Thus, each modeling result was calculated as the percent difference from the base run. For continuous variables, each modeling result was calculated as elasticity.

Among the categorical variables, the most sensitive parameters included receptor type (adult vs. child), room of use (Zone 1) selection, and application of the near-field bubble within Zone 1. However, these types of variables were held constant within a given product modeling scenario and were applied using consistent assumptions across all modeling scenarios.

D.2 Monitoring Data

D.2.1 Indoor Air Monitoring

Systematic review identified indoor air monitoring studies reporting levels of TCE in residential indoor air samples. The air concentrations reported in these studies are not used to evaluate risk to consumers since measurements are not attributable to consumer conditions of use. The full suite of extracted data (including residential, commercial) and associated data evaluation forms are found in [*Data Extraction Tables for Environmental Monitoring Data*. *Docket: EPA-HQ-OPPT-2019-0500*].

Concentrations of TCE in residential indoor air in the United States and Canada collected from nine studies identified during Systematic Review are summarized in Table_Apx D-1. Overall, more than 1,800 samples were collected between 1986 and 2010 in 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 (detection limits varied) to 42 µg/m^3. The highest concentrations were observed in residential garages and apartment hallways. Measures of central tendency (mean or median) across all studies were generally less than 1 µg/m^3, with a couple central tendency measurements above 3 µg/m^3.

Data extracted for residential indoor air samples from studies conducted outside of North America, as well as studies conducted in schools and commercial establishments in the US and other countries, are provided in [*Data Extraction Tables for Environmental Monitoring Data*. *Docket: EPA-HQ-OPPT-2019-0500*].

Table_Apx D-1. TCE Residential Indoor Air Concentrations (µg/m^3) in the United States and Canada

Study Info	Site Description	LOQ	Min.	Mean	Median	Max.	Variance	Data Eval. Score
(Chin et al., 2014) US, 2009-2010 (n=126; DF = 0.06)	Detroit, MI area; Homes (n=126) with children with asthma	0.09	ND	0.07	0.04	1.48	0.14 (SD)	High
(Dodson et al., 2008)[a] US, 2004-2005 (n=83; DF = 0.93)	Boston, MA; Interior room of residences	0.04	ND	0.6	0.2	2.2 (95th)	1.7 (SD)	High
(Dodson et al., 2008)[a] US, 2004-2005 (n=52; DF = 0.75)	Boston, MA; Basement of residences	0.04	ND	0.4	0.1	1.4 (95th)	1.1 (SD)	High
(Dodson et al., 2008)[a] US, 2004-2005 (n=10; DF = 0.9)	Boston, MA; Apartment hallway of residences	0.04	ND	3.7	0.3	23 (95th)	7.3 (SD)	High
(Dodson et al., 2008)[a] US, 2004-2005 (n=16; DF = 0.63)	Boston, MA; Garage of residences	0.04	ND	3.3	0.1	42 (95th)	10 (SD)	High
(Jia et al., 2008a) US, 2004-2005 (n=252; DF = 0.56)	Ann Arbor, Ypsilanti, and Dearborn MI; Residences (n=159) in industrial, urban, and suburban cities over two seasons	0.008	ND	0.06	0.03	2.01	--	Medium
(Adgate et al., 2004) US, 2000 (n=113; DF = 0.828)	Minneapolis, MN; Inside home, during the winter. Sampling from room where child spent the most time.	--	ND (10^{th} 0.1)	--	0.3	--	--	Medium
(Adgate et al., 2004) US, 2000 (n=113; DF = 0.737)	Minneapolis, MN; Inside home, during the spring. Sampling from room where child spent the most time.	--	ND (10^{th} 0.1)	--	0.2	--	--	Medium
(Sax et al., 2004) US, 2000 (n=32; DF = 0.47)	Los Angeles, CA; Homes (n=35) in inner-city neighborhood, sampled in the fall	0.13	ND	0.2	0.1	0.8	0.2 (SD)	High
(Sax et al., 2004) US, 2000 (n=40; DF = 0.68)	Los Angeles, CA; Homes (n=40) in inner-city neighborhood, sampled in the winter	0.13	ND	0.2	0.2	1.2	0.3 (SD)	High
(Sax et al., 2004) US, 1999 (n=36; DF = 0.92)	New York, NY; Homes (n=38) in inner-city neighborhood, sampled in the winter	0.13	ND	1.1	0.4	19	3.2 (SD)	High
(Sax et al., 2004) US, 1999 (n=30; DF = 0.44)	New York, NY; Homes (n=41) in inner-city	0.13	ND	0.3	0.1	2.6	0.5 (SD)	High

Study Info	Site Description	LOQ	Min.	Mean	Median	Max.	Variance	Data Eval. Score
	neighborhood, sampled in the summer							
(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)	--	--	0.99	0.22	1.74 (95th)	7.29 (SD)	Medium
(Clayton et al., 1999)[c] US, 1995-1997 (n=402; DF = 0.361)	IL, IN, OH, MI, MN, WI (Great Lakes Region); Non-institutionalized persons residing in households in six states	--	ND	3.84	0.56	2.28 (90th)	--	High
(Lindstrom et al., 1995) US, 1994 (n=9; DF = 0.56)	Denver, CO; Homes, occupied (n=9)	0.12	ND	0.64	0.61	--	0.66 (SD)	Medium
(Chan et al., 1990) CA, 1987 (n=6; DF = 0.83)	Homes (n=6), main floor	--	ND	1.6	--	5	--	Medium
(Chan et al., 1990) CA, 1986 (n=12; DF = 0.42)	Homes (n=12), main floor	--	ND	0.5	--	2	--	Medium

Study Info: The information provided includes the 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. GSD = geometric standard deviation. 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 were collected as part of the BEAMS study.
[b] Samples from this study were collected as part of the RIOPA study.
[c] Samples from this study were collected as part of the NHEXAS Phase 1 field study.

D.2.2 Personal breathing Zone Monitoring Data

Concentrations of TCE (TCE) in the personal breathing zones of residents in the United States collected from seven studies identified during Systematic Review are summarized in Table_Apx D-2. Overall, the measured concentration dataset contains approximately 2,750 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. Only the 3-hr samples from Heavner et al. (1995) represent time inside the home only. Concentrations ranged from non-detect (limits varied) to 327.3 µg/m^3. The highest concentration was observed in samples collected in 2000 as part of the NHANES 1999-2000 study (Jia et al., 2008b). The study states that the top ten highest concentrations exceeded 300 µg/m^3, which they suggest may indicate exposure from immediate contact with solvents. The 95th percentile concentration in this study is 7.4 µg/m^3. All other studies showed maximum concentrations less than 10 µg/m^3. Median concentrations ranged from ND to 1.05 µg/m^3; and average concentrations ranged from 0.66 to 13 µg/m^3.

Data extracted for residential/general personal breathing zones studies conducted outside of North America, as well as studies conducted in schools and commercial establishments in the US and other

countries, is provided in [*Data Extraction Tables for Environmental Monitoring Data*. *Docket: EPA-HQ-OPPT-2019-0500*].

Table_Apx D-2. Personal Breathing Zone Concentrations (µg/m3) for TCE in the United States (General/Residential)

Study Info	Type	Site Description	LOD	Min.	Mean	Median	Max	Variance	Data Eval. Score
(Su et al., 2013)[a] US, 1999-2001 (n=544; DF = 0.23)	48-hr	Elizabeth, NJ; Houston, TX; and Los Angeles, CA; Adults (n=309) and children (n=118) from 310 non-smoking households.	--	ND	1.44	0.22	2.37 (95th)	10.74 (SD)	Medium
(Jia et al., 2008b)[b] US, 1999-2000 (n=665; DF = 0.229)	48-to 72-hr	Nation-wide; Adults (ages 20–59 years) in NHANES study	0.44	ND	0.4 (GM)	ND	327.3 (7.4 - 95th)	3.4 (GSD)	High
(Sexton et al., 2007) US, 1999 (n=333; DF = 0.925)	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	1	0.2	1.8 (90th)	--	High
(Clayton et al., 1999)[c] US, 1995-1997 (n=386; DF = 0.394)	6-day	IL, IN, OH, MI, MN, WI (Great Lakes Region); Non-institutionalized persons	--	ND	5.27	0.63	5.98 (90th)	--	High
(Heavner et al., 1995) US, 1991 (n=24; DF = NR)	3-hrs (in home only)	Columbus, OH; Non-smoking women (n=24) with non-smoking husbands	--	ND	1.84	1.05	9.08	2.39	Medium
(Heavner et al., 1995) US, 1991 (n=25; DF = NR)	3-hrs (in home only)	Columbus, OH; Non-smoking (n=25) women with smoking husbands	--	ND	0.66	ND	3.41	1.04	Medium
(Wallace, 1987)[d] US, 1981-1984 (n=772; DF = 0-0.97)	12-hrs	Elizabeth and Bayonne, NJ, Los Angeles, CA, and Contra Costa, CA; Adults in industrial/ chemical manufacturing and /or petroleum refining regions of the US.	--	--	3.8 to 13	--	--	--	High

Abbreviations: If a value was not reported, it is shown in this table as "--". LOD = level of detection. 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 were collected as part of the RIOPA study.
[b] Samples from this study were collected as part of the NHANES 1999-2000. The top ten highest concentrations exceeded 300 µg/m^3, which the authors suggest may be from immediate contact with solvents.
[c] Samples from this study were collected as part of the NHEXAS Phase 1 field study.
[d] Samples from this study were collected as part of the TEAMS study.

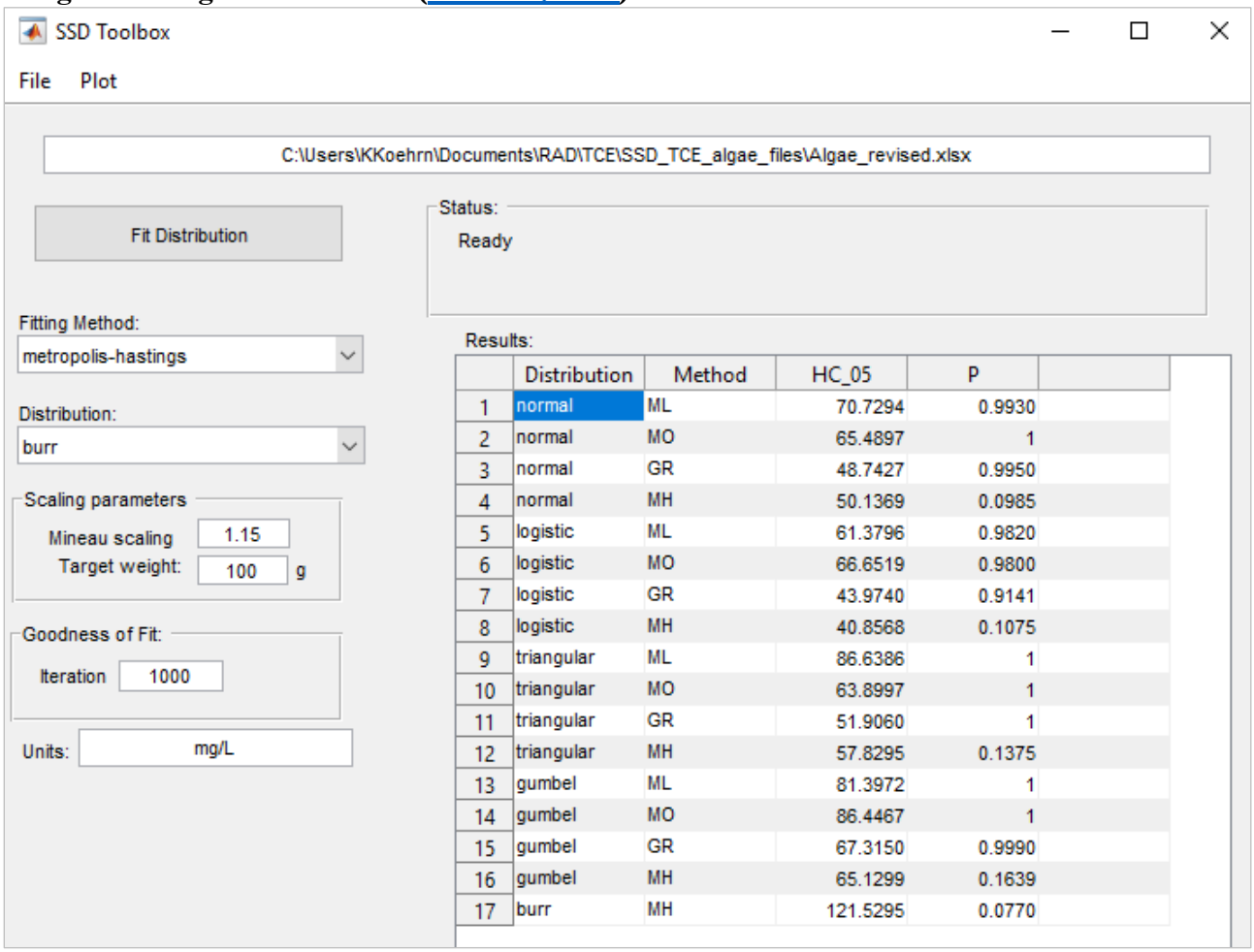
Appendix E ENVIRONMENTAL HAZARDS

E.1 Species Sensitivity Distribution (SSD) Methodology

The SSD Toolbox is a resource created by EPA's Office of Research and Development (ORD) that can fit SSDs to environmental hazard data (Etterson, 2019). It runs on Matlab 2018b (9.5) for Windows 64 bit. For this TCE Risk Evaluation, EPA created two SSDs with the SSD Toolbox, one using only algae hazard data and the other using acute hazard data for all other aquatic species. This appendix outlines the methodology used to create each.

For the algae SSD, algae hazard data were curated to prioritize study quality and to assure comparability between toxicity values (e.g., comparing EC_{50}s to EC_{50}s). The dataset included both saltwater and freshwater species, because the only saltwater species value was within the range of values reported for freshwater species. With this dataset, the Toolbox was used to apply a variety of algorithms to fit and visualize SSDs with different distributions. Figure_Apx E-1 shows the Toolbox interface after each distribution and fitting method was fit to the data. A hazardous concentration for 5% of species (HC_{05}) is calculated for each.

Figure_Apx E-1. SSD Toolbox interface and list of HC_{05}s for each distribution and fitting method using TCE's algae hazard data (Etterson, 2019)



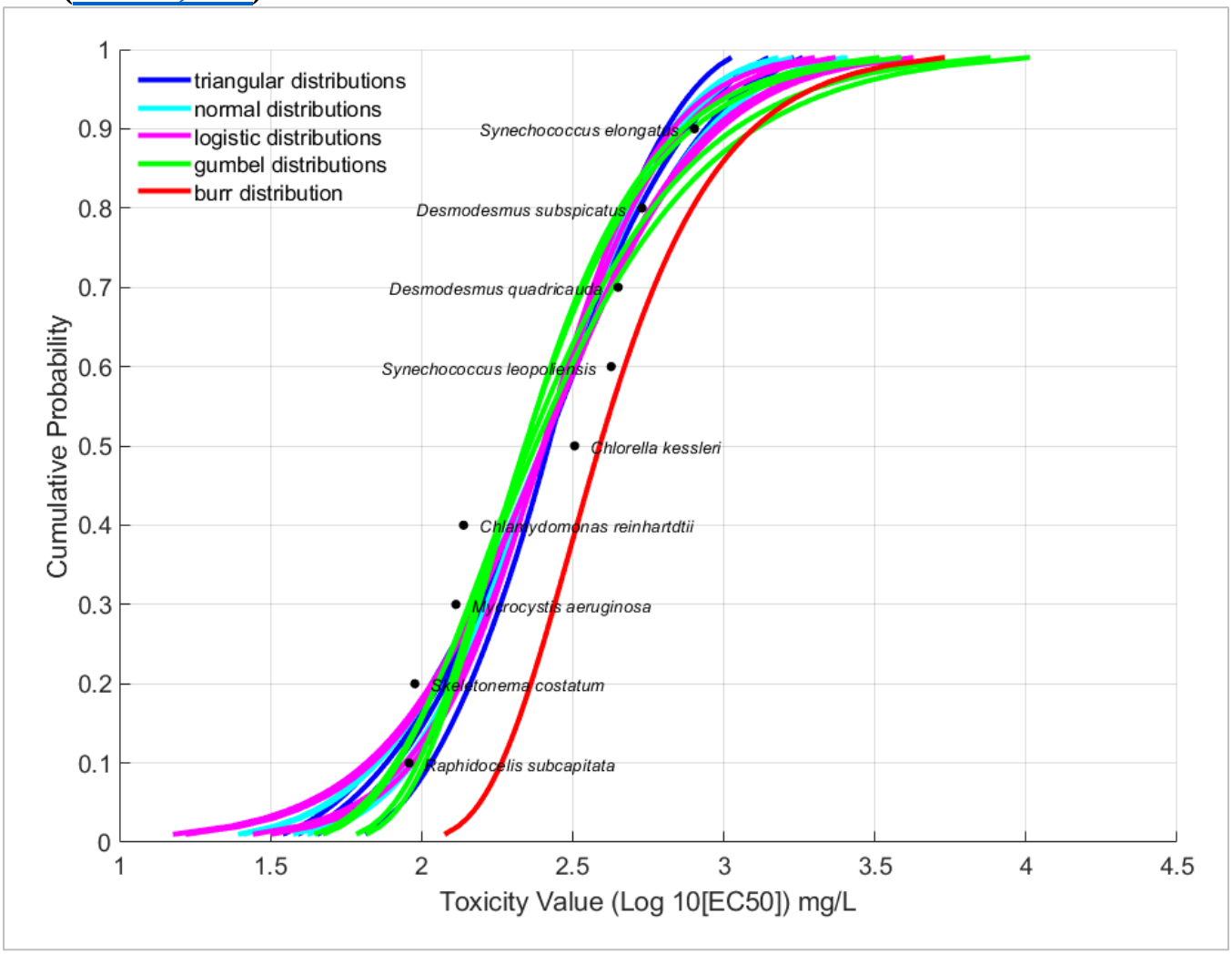
	Distribution	Method	HC_05	P
1	normal	ML	70.7294	0.9930
2	normal	MO	65.4897	1
3	normal	GR	48.7427	0.9950
4	normal	MH	50.1369	0.0985
5	logistic	ML	61.3796	0.9820
6	logistic	MO	66.6519	0.9800
7	logistic	GR	43.9740	0.9141
8	logistic	MH	40.8568	0.1075
9	triangular	ML	86.6386	1
10	triangular	MO	63.8997	1
11	triangular	GR	51.9060	1
12	triangular	MH	57.8295	0.1375
13	gumbel	ML	81.3972	1
14	gumbel	MO	86.4467	1
15	gumbel	GR	67.3150	0.9990
16	gumbel	MH	65.1299	0.1639
17	burr	MH	121.5295	0.0770

The SSD Toolbox's output contained several methods for choosing an appropriate distribution and fitting method, including goodness-of-fit and standard error among others. However, choosing the distribution with the best fit was challenging with a small dataset (e.g., hazard data for 9 algae species). P values for goodness-of-fit were all above 0.05, showing no evidence for lack of fit, and providing no help in discriminating among distributions (Figure_Apx E-1). Standard error was lowest across fitting methods for the Gumbel and Burr distributions (Table_Apx E-1). Because the ability for these measures to distinguish between distributions was limited, visual inspection of the distributions was used. For example, visual inspection showed Burr was not a good fit (Figure_Apx E-2).

Table_Apx E-1. Standard Error for all dsitributions and fitting methods using TCE's algae hazard data (Etterson, 2019)

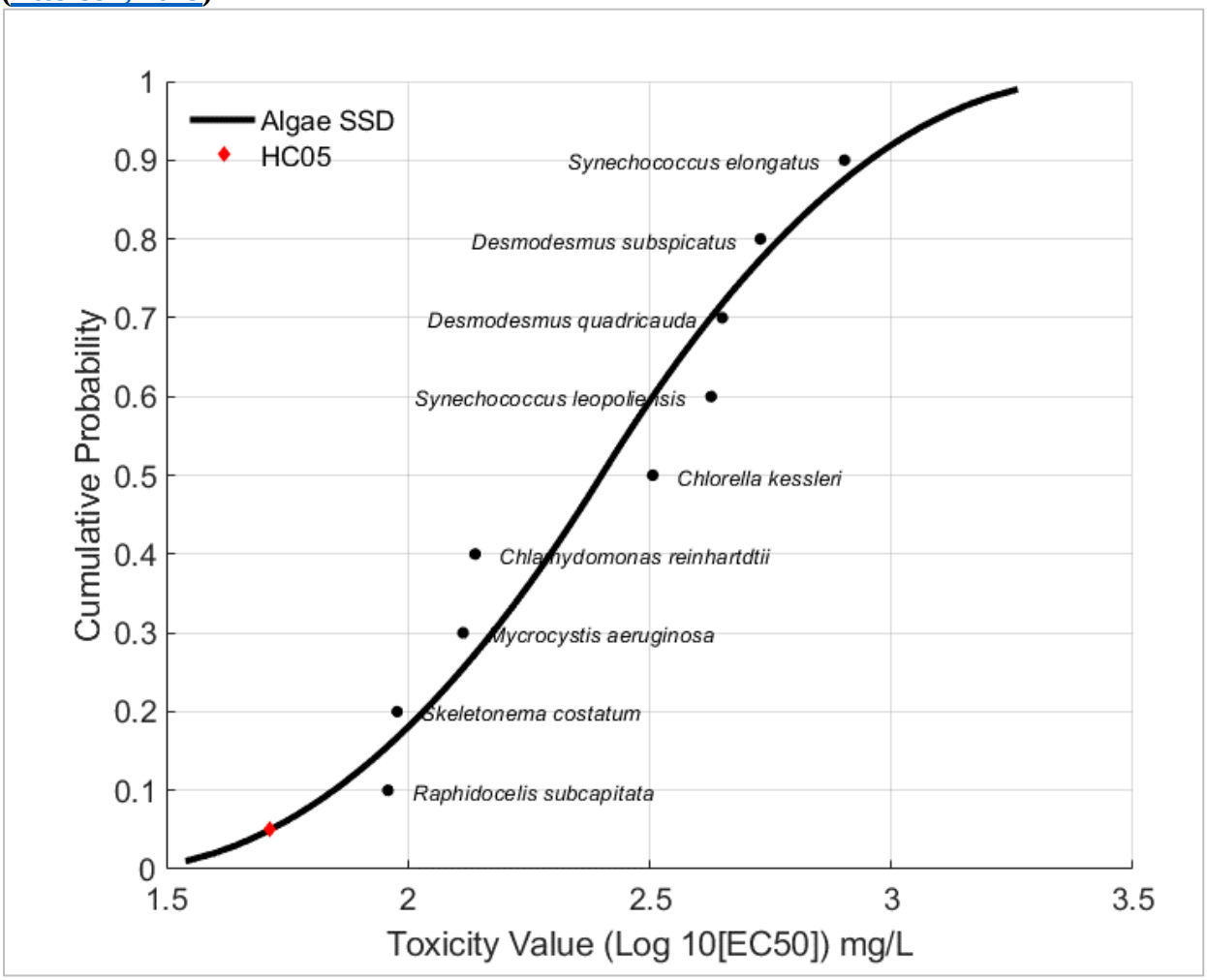
	Normal Distribution				Logistic Distribution				Triangular Distribution				Gumbel Distribution				Burr Distribution
	ML	MO	GR	MH	ML	MO	GR	MH	ML	MO	GR	MH	ML	MO	GR	MH	MH
Standard Error for HC_{05}	35.7	33.9	26.1	27.9	36.4	33.7	26.1	29.2	34.0	33.4	26.5	28.9	26.6	28.6	26.2	23.9	20.9

Figure_Apx E-2. All distributions and fitting methods in the SSD Toolbox for TCE's algae hazard data (Etterson, 2019)



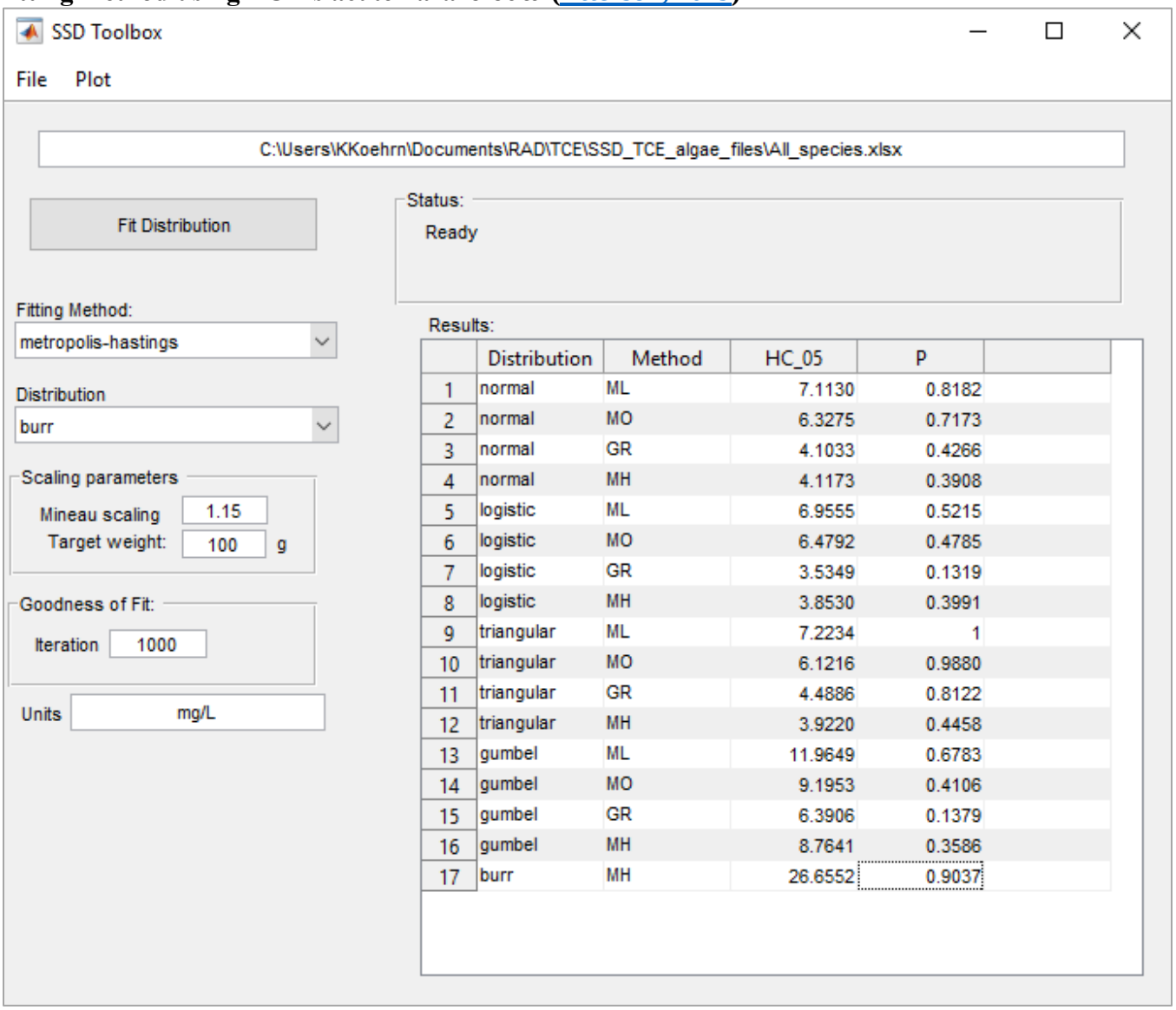
Using standard error and visual inspection, the distributions with the best fit for the most sensitive algae species included triangular and Gumbel. The triangular distribution with graphical methods fitting was the most protective, and was used as a line of evidence for assessing algae in this assessment (Figure_Apx E-3). The resulting SSD calculated an HC_{05} of 52 mg/L or 52,000 µg/L.

Figure_Apx E-3. TCE algae data fit with triangular distribution fit with graphical methods (Etterson, 2019)



For the acute SSD, acute hazard data for fish, amphibians, and invertebrates were curated to prioritize study quality and to assure comparability between toxicity values. For example, the dataset included only LC_{50}s for fish and amphibians, and EC_{50}s or LC_{50}s that measured immobilization and mortality for aquatic invertebrates. The dataset included both saltwater and freshwater species, because the toxicity values for saltwater species value were within the range of values reported for freshwater species in the same taxonomic group. Additionally, for fish and invertebrates, the mode of action for freshwater and saltwater species expected to be the same. With this dataset, the Toolbox was used to apply a variety of algorithms to fit and visualize SSDs with different distributions. Figure_Apx E-4 shows the Toolbox interface after each distribution and fitting method was fit to the data. An HC_{05} is calculated for each.

Figure_Apx E-4. SSD Toolbox interface showing HC_{05}s and P values for each distribution and fitting method using TCE's acute hazard data (Etterson, 2019)



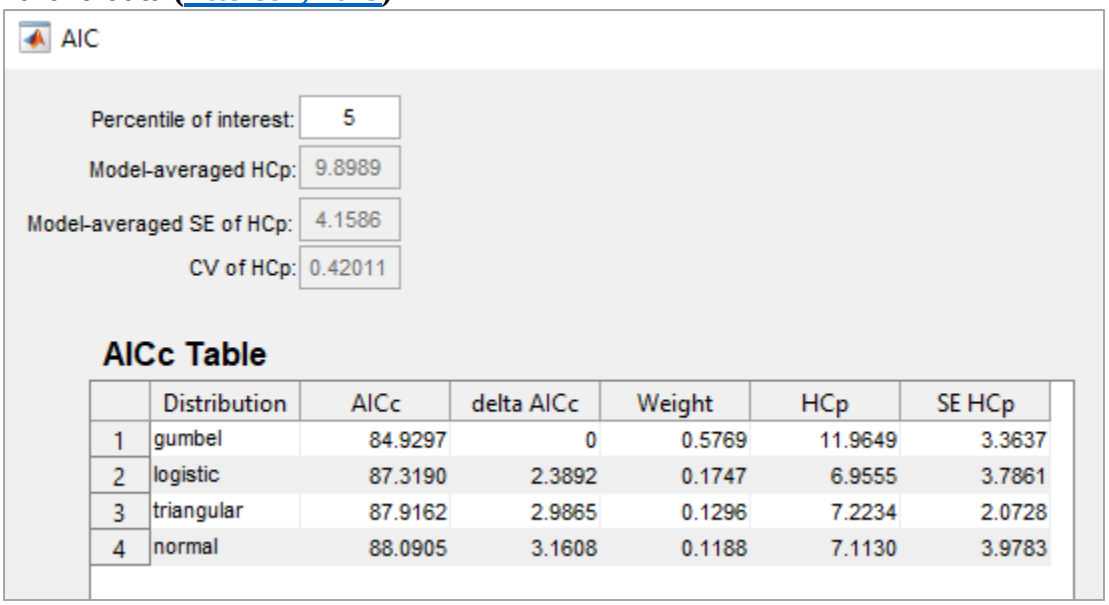
	Distribution	Method	HC_05	P
1	normal	ML	7.1130	0.8182
2	normal	MO	6.3275	0.7173
3	normal	GR	4.1033	0.4266
4	normal	MH	4.1173	0.3908
5	logistic	ML	6.9555	0.5215
6	logistic	MO	6.4792	0.4785
7	logistic	GR	3.5349	0.1319
8	logistic	MH	3.8530	0.3991
9	triangular	ML	7.2234	1
10	triangular	MO	6.1216	0.9880
11	triangular	GR	4.4886	0.8122
12	triangular	MH	3.9220	0.4458
13	gumbel	ML	11.9649	0.6783
14	gumbel	MO	9.1953	0.4106
15	gumbel	GR	6.3906	0.1379
16	gumbel	MH	8.7641	0.3586
17	burr	MH	26.6552	0.9037

Again the SSD Toolbox's output contained several methods for choosing an appropriate distribution and fitting method, including goodness-of-fit, standard error, and sample-size corrected Akaike Information Criterion (AIC_c, [Burnham and Anderson, 2002]). P values for goodness-of-fit were all above 0.05, showing no evidence for lack of fit, and providing no help in discriminating among distributions (Figure_Apx E-4). Standard error was mixed across fitting methods for some distributions but generally the lowest for the burr distribution (Table_Apx E-2). Figure_Apx E-5 shows that the gumbel distribution has the lowest AIC_c, indicating it may be the best distribution for this data though the relative AIC support compared to other distributions is weak. Because the ability for these measures to distinguish between distributions was limited, visual inspection of the distributions was also used. For example, visual inspection showed Burr was not a good fit (Figure_Apx E-6).

Table_Apx E-2. Standard Error for all distributions and fitting methods using TCE's acute hazard data (Etterson, 2019)

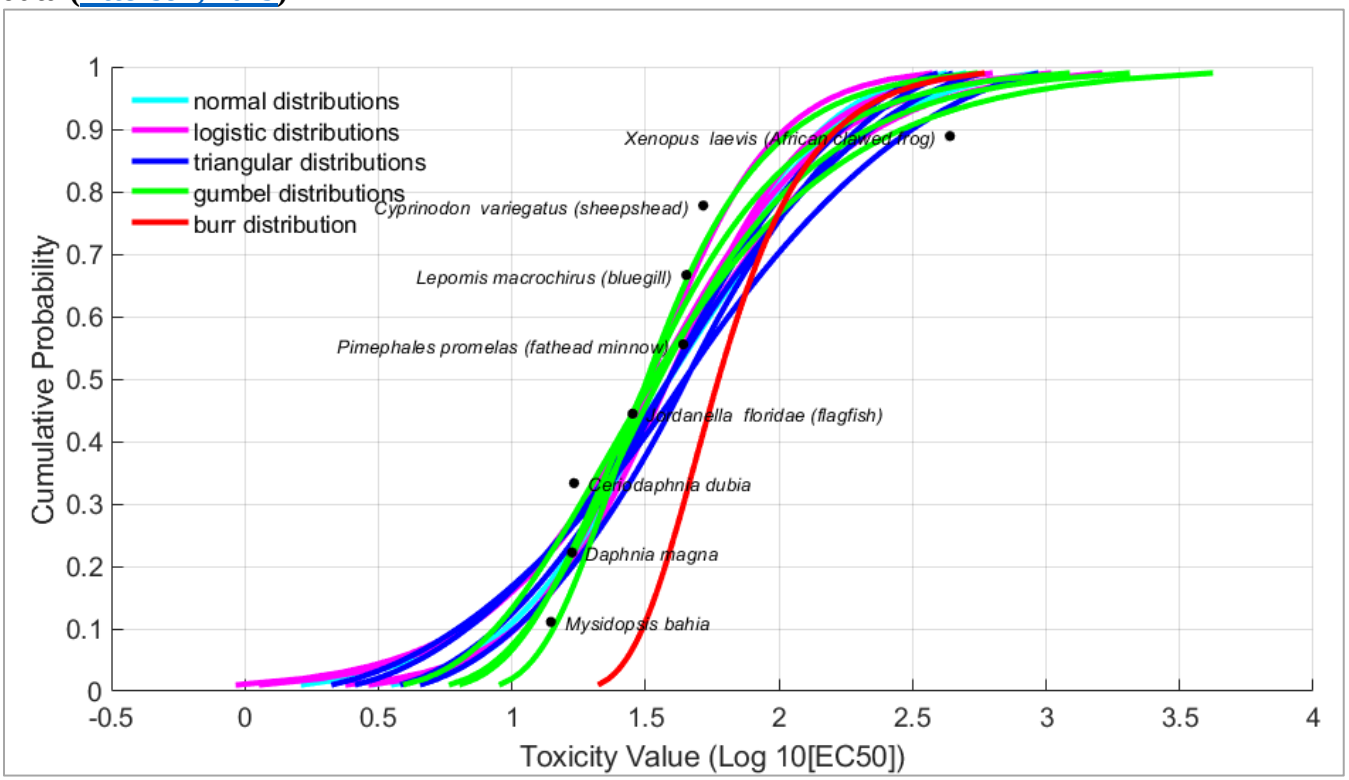
	Normal Distribution				Logistic Distribution				Triangular Distribution				Gumbel Distribution				Burr Distribution
	ML	MO	GR	MH	ML	MO	GR	MH	ML	MO	GR	MH	ML	MO	GR	MH	MH
Standard Error for HC05	5.8	5.2	3.7	3.7	4.8	5.9	3.4	3.8	6.9	5.0	3.9	4.1	4.1	4.6	3.6	3.9	2.9

Figure_Apx E-5. AIC_c for the four distribution options in the SSD Toolbox for TCE's acute hazard data (Etterson, 2019)



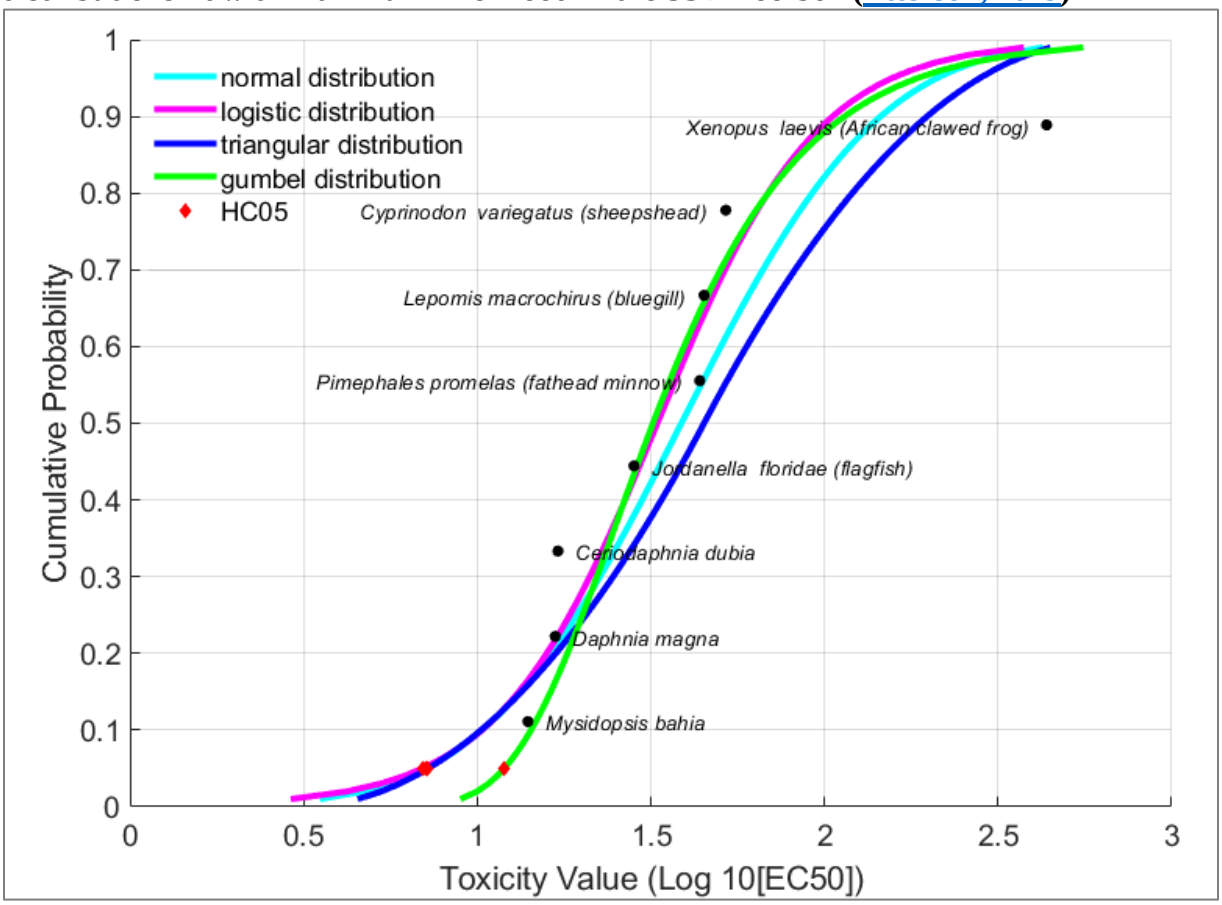
	Distribution	AICc	delta AICc	Weight	HCp	SE HCp
1	gumbel	84.9297	0	0.5769	11.9649	3.3637
2	logistic	87.3190	2.3892	0.1747	6.9555	3.7861
3	triangular	87.9162	2.9865	0.1296	7.2234	2.0728
4	normal	88.0905	3.1608	0.1188	7.1130	3.9783

Figure_Apx E-6. All distributions and fitting methods in the SSD Toolbox for TCE's acute hazard data (Etterson, 2019)



EPA used a model average of the Gumbel, logistic, triangular, and normal distributions, because it was not clear which distribution had the best fit after considering standard error, AIC_c, and visual inspection. The model-averaged HC_{05} from all four distributions was 9.9 mg/L or 9,900 µg/L, and the SSDs showed aquatic invertebrates were the most sensitive species (Figure_Apx E-7).

Figure_Apx E-7. TCE's acute hazard data fit with the normal, logistic, triangular, and Gumbel distributions fit with maximum likelihood in the SSD Toolbox (Etterson, 2019)



E.2 Environmental Risk Quotients (RQs) for Facilities Releasing TCE to Surface Water as Modeled in E-FAST

Table_Apx E-3. Environmental RQs by Facility (with RQs ≥ 1 in bold)

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
OES: Manufacturing										
Axiall Corporation, Westlake, LA NPDES: LA0007129	Surface Water	NPDES LA0007129	Surface water	350	1.266	0.0051	0.00	0.00	0.00	0.00
				20	22.15	0.0897	0.00	0.00	0.03	0.00
Olin Blue Cube, Freeport, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.069	2.42	0.00	0.00	0.81	0.00
				20	1.2	42.14	0.01	0.05	**14.05**	0.00
Solvents & Chemicals, Pearland, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.53	0.00	0.00	0.18	0.00
				20	0.265	9.48	0.00	0.01	**3.16**	0.00
	Surface Water	Organic Chemicals Manuf.	Surface water	350	0.015	2.77	0.00	0.00	0.92	0.00
				20	0.265	49.91	0.02	0.06	**16.64**	0.00
Occidental Chemical Corp Wichita, Wichita, KS NPDES: KS0096903 and Organic Chem MFG SIC	Surface Water	Surrogate NPDES KS0043036	Surface water	350	0.015	0.07	0.00	0.00	0.02	0.00
				20	0.265	1.33	0.00	0.00	0.44	0.00
	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.53	0.00	0.00	0.18	0.00
				20	0.265	9.48	0.00	0.01	**3.16**	0.00
OES: Processing as a Reactant										

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
440 unknown sites NPDES: Not applicable	Off-site Waste-water Treatment	Organic Chemicals Manufacture	Surface water	350	0.005	0.18	0.00	0.00	0.06	0.00
				20	0.089	3.13	0.00	0.00	**1.04**	0.00
	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.005	0.92	0.00	0.00	0.31	0.00
				20	0.089	16.45	0.01	0.02	**5.48**	0.00
Arkema Inc. Calvert City, KY NPDES: KY0003603	Surface Water	NPDES KY0003603	Surface water	350	0.017	0.000737	0.00	0.00	0.00	0.00
				20	0.295	0.128	0.00	0.00	0.04	0.00
Honeywell International - Geismar Complex, Geismar, LA NPDES: LA0006181	Surface Water	NPDES LA0006181	Surface water	350	0.0128	0.0000518	0.00	0.00	0.00	0.00
				20	0.224	0.000907	0.00	0.00	0.00	0.00
Praxair Technology Center, Tonawanda, NY NPDES: NY0000281	Surface Water	NPDES NY0000281	Still body	350	0.00169	169	0.05	0.21	**56.33**	0.00
				20	0.03	3000	0.94	**3.81**	**1000.00**	0.06
US DOE Paducah Site, Kevil, KY NPDES: KY0102083	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
GNF-A Wilmington-Castle Hayne, Wilmington NC NPDES: NC0001228	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: Repackaging										

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Hubbard-Hall Inc, Waterbury, CT NPDES: Unknown	Off-site Waste-water Treatment	Receiving Facility: Recycle Inc.; POTW (Ind.)	Surface water	250	1.108	27.18	0.01	0.03	**9.06**	0.00
				20	13.85	339.11	0.11	0.43	**113.04**	0.01
Oiltanking Houston Inc, Houston, TX NPDES: TX0091855	Surface Water	Surrogate NPDES TX0065943	Surface water	250	0.003	6.52	0.00	0.01	**2.17**	0.00
				20	0.041	89.13	0.03	0.11	**29.71**	0.00
St. Gabriel Terminal, Saint Gabriel, LA NPDES: LA0005487	Surface Water	NPDES LA0005487	Surface water	250	0.0055	0.0000223	0.00	0.00	0.00	0.00
				20	0.069	0.000279	0.00	0.00	0.00	0.00
Vopak Terminal Westwego Inc, Westwego, LA NPDES: LA0124583	Surface Water	Surrogate NPDES LA0042064	Surface water	250	0.00468	0.0000189	0.00	0.00	0.00	0.00
				20	0.058	0.000235	0.00	0.00	0.00	0.00
Research Solutions Group Inc, Pelham, AL NPDES: AL0074276	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Carlisle Engineered Products Inc, Middlefield, OH NPDES: OH0052370	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, and Metalworking Fluids)										
Texas Instruments, Inc.,				260	0.005	0.0188	0.00	0.00	0.01	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Attleboro, MA NPDES: MA0001791	Surface Water	NPDES MA0001791	Surface water	20	0.067	0.25	0.00	0.00	0.08	0.00
Accellent Inc/Collegeville Microcoax, Collegeville, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.002	0.0425	0.00	0.00	0.01	0.00
				20	0.029	0.62	0.00	0.00	0.21	0.00
Ametek Inc. U.S. Gauge Div., Sellersville, PA NPDES: PA0056014	Surface Water	Surrogate NPDES PA0020460	Surface water	260	0.001	0.0619	0.00	0.00	0.02	0.00
				20	0.011	0.68	0.00	0.00	0.23	0.00
Atk-Allegany Ballistics Lab (Nirop), Keyser, WV NPDES: WV0020371	Surface Water	NPDES WV0020371	Surface water	260	0.0005	0.00311	0.00	0.00	0.00	0.00
				20	0.0061	0.0373	0.00	0.00	0.01	0.00
Handy & Harman Tube Co/East Norriton, Norristown, PA NPDES: PA0011436	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
US Nasa Michoud Assembly Facility, New Orleans, LA NPDES: LA0052256	Surface Water	Surrogate NPDES LA0003280	Still body	260	1.96	765.63	0.24	0.97	**255.21**	0.01
				20	25.44	9937.5	**3.11**	**12.61**	**3312.50**	0.19
GM Components Holdings LLC,	Surface Water	NPDES NY0000558	Surface water	260	0.13	10.97	0.00	0.01	**3.66**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Lockport, NY NPDES: NY0000558				20	1.71	144.47	0.05	0.18	**48.16**	0.00
Akebono Elizabethtown Plant, Elizabethtown, KY NPDES: KY0089672	Surface Water	Surrogate NPDES KY0022039	Surface water	260	0.07	4.87	0.00	0.01	**1.62**	0.00
				20	0.897	62.38	0.02	0.08	**20.79**	0.00
Delphi Harrison Thermal Systems, Dayton, OH NPDES: OH0009431	Surface Water	NPDES OH0009431	Surface water	260	0.04	0.0752	0.00	0.00	0.03	0.00
				20	0.465	0.87	0.00	0.00	0.29	0.00
Chemours Company Fc LLC, Washington, WV NPDES: WV0001279	Surface Water	NPDES WV0001279	Surface water	260	0.03	0.00301	0.00	0.00	0.00	0.00
				20	0.334	0.0335	0.00	0.00	0.01	0.00
Equistar Chemicals Lp, La Porte, TX NPDES: TX0119792	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.02	2.22	0.00	0.00	0.74	0.00
				20	0.218	24.44	0.01	0.03	**8.15**	0.00
GE Aviation, Lynn, MA NPDES: MA0003905	Surface Water	NPDES MA0003905	Still water	260	0.01	0.0425	0.00	0.00	0.01	0.00
				20	0.128	0.54	0.00	0.00	0.18	0.00
Certa Vandalia LLC, Vandalia, OH NPDES: OH0122751	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.01	1.11	0.00	0.00	0.37	0.00
				20	0.107	11.89	0.00	0.02	**3.96**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
GM Components Holdings LLC Kokomo Ops, Kokomo, IN NPDES: IN0001830	Surface Water	NPDES IN0001830	Surface water	260	0.01	0.2	0.00	0.00	0.07	0.00
				20	0.086	1.73	0.00	0.00	0.58	0.00
Amphenol Corp-Aerospace Operations, Sidney, NY NPDES: NY0003824	Surface Water	NPDES NY0003824	Surface water	260	0.01	0.0486	0.00	0.00	0.02	0.00
				20	0.082	0.4	0.00	0.00	0.13	0.00
Emerson Power Trans Corp, Maysville, KY NPDES: KY0100196	Surface Water	Surrogate NPDES KY0020257	Surface water	260	0.01	0.0004	0.00	0.00	0.00	0.00
				20	0.081	0.00522	0.00	0.00	0.00	0.00
Olean Advanced Products, Olean, NY NPDES: NY0073547	Surface Water	Surrogate NPDES NY0027162	Surface water	260	0.01	0.0188	0.00	0.00	0.01	0.00
				20	0.068	0.13	0.00	0.00	0.04	0.00
Hollingsworth Saco Lowell, Easley, SC NPDES: SC0046396	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00469	0.52	0.00	0.00	0.17	0.00
				20	0.061	6.78	0.00	0.01	**2.26**	0.00
Trelleborg YSH Incorporated Sandusky Plant, Sandusky, MI NPDES: MI0028142	Surface Water	NPDES MI0028142	Surface water	260	0.0036	1.76	0.00	0.00	0.59	0.00
				20	0.047	23.04	0.01	0.03	**7.68**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Timken Us Corp Honea Path, Honea Path, SC NPDES: SC0047520	Surface Water	Surrogate NPDES SC0000698	Surface water	260	0.00355	1.06	0.00	0.00	0.35	0.00
				20	0.0462	13.77	0.00	0.02	**4.59**	0.00
Johnson Controls Incorporated, Wichita, KS NPDES: KS0000850	Surface Water	NPDES KS0000850	Surface water	260	0.00228	0.0548	0.00	0.00	0.02	0.00
				20	0.0296	0.72	0.00	0.00	0.24	0.00
National Railroad Passenger Corporation (Amtrak) Wilmington Maintenance Facility, Wilmington, DE NPDES: DE0050962	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00203	0.23	0.00	0.00	0.08	0.00
				20	0.026	2.89	0.00	0.00	0.96	0.00
Electrolux Home Products (Formerly Frigidaire), Greenville, MI NPDES: MI0002135	Surface Water	NPDES MI0002135	Surface water	260	0.00201	0.0171	0.00	0.00	0.01	0.00
				20	0.026	0.22	0.00	0.00	0.07	0.00
Rex Heat Treat Lansdale Inc, Lansdale, PA NPDES: PA0052965	Surface Water	Surrogate NPDES PA0026182	Surface water	260	0.00194	0.0523	0.00	0.00	0.02	0.00
				20	0.025	0.67	0.00	0.00	0.22	0.00
Carrier Corporation, Syracuse, NY NPDES: NY0001163	Surface Water	NPDES NY0001163	Still water	260	0.00177	0.22	0.00	0.00	0.07	0.00
				20	0.023	2.84	0.00	0.00	0.95	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Cascade Corp (0812100207), Springfield, OH NPDES: OH0085715	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00117	0.13	0.00	0.00	0.04	0.00
				20	0.015	1.67	0.00	0.00	0.56	0.00
USAF-Wurtsmith Afb, Oscoda, MI NPDES: MI0042285	Surface Water	Surrogate NPDES MI0028282	Surface water	260	0.00115	0.000753	0.00	0.00	0.00	0.00
				20	0.015	0.00983	0.00	0.00	0.00	0.00
AAR Mobility Systems, Cadillac, MI NPDES: MI0002640	Surface Water	Surrogate NPDES MI0020257	Surface water	260	0.00112	0.00916	0.00	0.00	0.00	0.00
				20	0.014	0.11	0.00	0.00	0.04	0.00
Eaton Mdh Company Inc, Kearney, NE NPDES: NE0114405	Surface Water	Surrogate NPDES NE0052647	Still water	260	0.00107	0.13	0.00	0.00	0.04	0.00
				20	0.014	1.69	0.00	0.00	0.56	0.00
Lake Region Medical, Trappe, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.0005	0.0106	0.00	0.00	0.00	0.00
				20	0.007	0.15	0.00	0.00	0.05	0.00
Motor Components L L C, Elmira, NY NPDES: NY0004081	Surface Water	NPDES NY0004081	Surface water	260	0.00096	0.0618	0.00	0.00	0.02	0.00
				20	0.0125	0.83	0.00	0.00	0.28	0.00
Salem Tube Mfg, Greenville, PA NPDES: PA0221244	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000897	0.0997	0.00	0.00	0.03	0.00
				20	0.012	1.33	0.00	0.00	0.44	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
GE (Greenville) Gas Turbines LLC, Greenville, SC NPDES: SC0003484	Surface Water	NPDES SC0003484	Surface water	260	0.000806	0.0821	0.00	0.00	0.03	0.00
				20	0.01	1.02	0.00	0.00	0.34	0.00
Parker Hannifin Corporation, Waverly, OH NPDES: OH0104132	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000747	0.083	0.00	0.00	0.03	0.00
				20	0.01	1.11	0.00	0.00	0.37	0.00
Mahle Engine Components Usa Inc, Muskegon, MI NPDES: MI0004057	Surface Water	NPDES MI0004057	Surface water	260	0.000742	0.0336	0.00	0.00	0.01	0.00
				20	0.01	0.45	0.00	0.00	0.15	0.00
General Electric Company - Waynesboro, Waynesboro, VA NPDES: VA0002402	Surface Water	NPDES VA0002402	Surface water	260	0.000733	0.00705	0.00	0.00	0.00	0.00
				20	0.01	0.0962	0.00	0.00	0.03	0.00
Globe Engineering Co Inc, Wichita, KS NPDES: KS0086703	Surface Water	Surrogate NPDES KS0043036	Surface water	260	0.00173	0.00853	0.00	0.00	0.00	0.00
				20	0.023	0.11	0.00	0.00	0.04	0.00
Gayston Corp, Dayton, OH NPDES: OH0127043	Surface Water	Surrogate NPDES OH0024881	Surface water	260	0.000643	0.00121	0.00	0.00	0.00	0.00
				20	0.008	0.015	0.00	0.00	0.01	0.00
Styrolution America LLC, Channahon, IL NPDES: IL0001619	Surface Water	NPDES IL0001619	Surface water	260	0.000637	0.000221	0.00	0.00	0.00	0.00
				20	0.008	0.00278	0.00	0.00	0.00	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Remington Arms Co Inc, Ilion, NY NPDES: NY0005282	Surface Water	NPDES NY0005282	Surface water	260	0.000612	0.000799	0.00	0.00	0.00	0.00
				20	0.008	0.0104	0.00	0.00	0.00	0.00
United Technologies Corporation, Pratt And Whitney Division, East Hartford, CT NPDES: CT0001376	Surface Water	NPDES CT0001376	Surface water	260	0.00048	0.0000822	0.00	0.00	0.00	0.00
				20	0.006	0.00103	0.00	0.00	0.00	0.00
Atk-Allegany Ballistics Lab (Nirop), Keyser, WV NPDES: WV0020371	Surface Water	NPDES WV0020371	Surface water	260	0.00047	0.00292	0.00	0.00	0.00	0.00
				20	0.006	0.0373	0.00	0.00	0.01	0.00
Sperry & Rice Manufacturing Co LLC, Brookville, IN NPDES: IN0001473	Surface Water	NPDES IN0001473	Surface water	260	0.000328	0.00569	0.00	0.00	0.00	0.00
				20	0.004	0.0694	0.00	0.00	0.02	0.00
Owt Industries, Pickens, SC NPDES: SC0026492	Surface Water	NPDES SC0026492	Surface water	260	0.000314	0.00213	0.00	0.00	0.00	0.00
				20	0.004	0.0272	0.00	0.00	0.01	0.00
Boler Company, Hillsdale, MI NPDES: MI0053651	Surface Water	Surrogate NPDES MI0022136	Surface water	260	0.000269	0.0204	0.00	0.00	0.01	0.00
				20	0.003	0.23	0.00	0.00	0.08	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Mccanna Inc., Carpentersville, IL NPDES: IL0071340	Surface Water	Surrogate NPDES IL0027944	Surface water	260	0.000268	0.000911	0.00	0.00	0.00	0.00
				20	0.003	0.0102	0.00	0.00	0.00	0.00
Cutler Hammer, Horseheads, NY NPDES: NY0246174	Surface Water	Surrogate NPDES NY0004081	Surface water	260	0.000238	0.0153	0.00	0.00	0.01	0.00
				20	0.003	0.19	0.00	0.00	0.06	0.00
US Air Force Offutt Afb Ne, Offutt A F B, NE NPDES: NE0121789	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000159	0.0177	0.00	0.00	0.01	0.00
				20	0.002	0.22	0.00	0.00	0.07	0.00
Troxel Company, Moscow, TN NPDES: TN0000451	Surface Water	NPDES TN0000451	Surface water	260	0.000134	0.000741	0.00	0.00	0.00	0.00
				20	0.002	0.0111	0.00	0.00	0.00	0.00
Austin Tube Prod, Baldwin, MI NPDES: MI0054224	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000114	0.0127	0.00	0.00	0.00	0.00
				20	0.001	0.11	0.00	0.00	0.04	0.00
LS Starrett Precision Tools, Athol, MA NPDES: MA0001350	Surface Water	NPDES MA0001350	Surface water	260	0.000102	0.00153	0.00	0.00	0.00	0.00
				20	0.001	0.015	0.00	0.00	0.01	0.00
Avx Corp, Raleigh, NC NPDES: NC0089494	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.0000883	0.00981	0.00	0.00	0.00	0.00
				20	0.001	0.11	0.00	0.00	0.04	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Indian Head Division, Naval Surface Warfare Center, Indian Head, MD NPDES: MD0003158	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
General Dynamics Ordnance Tactical Systems, Red Lion, PA NPDES: PA0043672	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Trane Residential Solutions - Fort Smith, Fort Smith, AR NPDES: AR0052477	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Lexmark International Inc., Lexington, KY NPDES: KY0097624	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Alliant Techsystems Operations LLC, Elkton, MD NPDES: MD0000078	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Daikin Applied America, Inc. (Formally Mcquay International), Scottsboro, AL NPDES: AL0069701	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Beechcraft Corporation, Wichita, KS NPDES: KS0000183	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Federal-Mogul Corp, Scottsville, KY NPDES: KY0106585	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Cessna Aircraft Co (Pawnee Facility), Wichita, KS NPDES: KS0000647	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
N.G.I, Parkersburg, WV NPDES: WV0003204	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Hyster-Yale Group, Inc, Sulligent, AL NPDES: AL0069787	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Hitachi Electronic Devices (Usa), Inc., Greenville, SC NPDES: SC0048411	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: Adhesives, Sealants, Paints, and Coatings										

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Able Electropolishing Co Inc, Chicago, IL NPDES: Not available	POTW	Adhesives and Sealants Manuf.	Surface water	250	0.298	7.28	0.00	0.01	**2.43**	0.00
Garlock Sealing Technologies, Palmyra, NY, NPDES: NY0000078	Surface Water	NPDES NY0000078	Surface water	250	0.00033	0.00716	0.00	0.00	0.00	0.00
				20	0.00407	0.0889	0.00	0.00	0.03	0.00
Ls Starrett Co, Athol, MA NPDES: MAR05B615	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Aerojet Rocketdyne, Inc., East Camden, AR NPDES: AR0051071, ARR00A521, ARR00A520	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Best One Tire & Service, Nashville, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Bridgestone Aircraft Tire (Usa), Inc., Mayodan, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Clayton Homes Inc, Oxford, NC	Surface Water		Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
NPDES: Not available	POTW	Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Cmh Manufacturing, Inc. Dba Schult Homes - Plant 958, Richfield, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Delphi Thermal Systems, Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	250	0.013	1.1	0.00	0.00	0.37	0.00
				20	0.16	13.5	0.00	0.02	**4.50**	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Green Bay Packaging Inc - Coon Rapids, Coon Rapids, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Mastercraft Boat Company, Vonore, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Michelin Aircraft Tire Company, Norwood, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

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M-Tek, Inc, Manchester, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Olin Corp, East Alton, IL NPDES: IL0000230	Surface Water	NPDES IL0000230	Surface water	250	0.013	0.18	0.00	0.00	0.06	0.00
				20	0.16	2.26	0.00	0.00	0.75	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Parker Hannifin Corp – Paraflex Division, Manitowoc, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Parrish Tire Company, Yadkinville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Republic Doors And Frames, Mckenzie, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Ro-Lab Rubber Company Inc.,	Surface Water		Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00

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Tracy, CA NPDES: Not available	POTW	Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Royale Comfort Seating, Inc. - Plant No. 1, Taylorsville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Snider Tire, Inc., Statesville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Snyder Paper Corporation, Hickory, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Stellana Us, Lake Geneva, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Thomas Built Buses - Courtesy Road, High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Unicel Corp, Escondido, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Acme Finishing Co Llc, Elk Grove Village, IL	Surface Water		Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00

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NPDES: Not available	POTW	Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Aerojet Rocketdyne, Inc., Rancho Cordova, CA NPDES: CA0004111	Surface Water	NPDES CA0004111	Surface water	250	0.013	0.000818	0.00	0.00	0.00	0.00
				20	0.16	0.0101	0.00	0.00	0.00	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Allegheny Cnty Airport Auth/ Pgh Intl Airport, Coroapolis Pittsburgh, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Amphenol Corp – Aerospace Operations, Sidney, NY NPDES: NY0003824	Surface Water	NPDES NY0003824	Surface water	250	0.013	0.0631	0.00	0.00	0.02	0.00
				20	0.16	0.78	0.00	0.00	0.26	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Aprotech Powertrain, Asheville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

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Coating & Converting Tech Corp/ Adhesive Coatings, Philadelphia, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Corpus Christi Army Depot, Corpus Christi, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Electronic Data Systems Camp Pendleton, Camp Pendleton, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Florida Production Engineering, Inc., Ormond Beach, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Goodrich Corporation, Jacksonville, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Kasai North America Inc, Madison Plant, Madison, MS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Kirtland Air Force Base,				250	0.013	1.67	0.00	0.00	0.56	0.00

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Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Marvin Windows & Doors, Warroad, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Mcneilus Truck & Manufacturing Inc, Dodge Center, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Metal Finishing Co. – Wichita (S Mclean Blvd), Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Murakami Manufacturing Usa Inc, Campbellsville, KY NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Peterbilt Motors Denton Facility, Denton, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Portsmouth Naval Shipyard, Kittery, ME NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
R.D. Henry & Co., Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Raytheon Company, Portsmouth, RI NPDES: RI0000281	Surface Water	NPDES RI0000281	Still body	250	0.013	10.83	0.00	0.01	**3.61**	0.00
				20	0.16	133.33	0.04	0.17	**44.44**	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Rehau Inc, Cullman, AL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Rotochopper Inc, Saint Martin, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Rubber Applications, Mulberry, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Sapa Precision Tubing Rockledge, Llc, Rockledge, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Thomas & Betts, Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Thomas Built Buses - Fairfield Road, High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Timco, Dba Haeco Americas Airframe Services, Greensboro, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Trelleborg Coated Systems Us, Inc – Grace Advanced Materials, Rutherfordton, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
U.S. Coast Guard Yard - Curtis Bay, Curtis Bay, MD NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Viracon Inc, Owatonna, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
OES: Other Industrial Uses										

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Eli Lilly And Company-Lilly Tech Ctr, Indianapolis, IN NPDES: IN0003310	Surface Water	NPDES IN0003310	Surface water	250	1.553	9.03	0.00	0.01	**3.01**	0.00
				20	19.41	113.09	0.04	0.14	**37.70**	0.00
Oxy Vinyls LP - Deer Park Pvc, Deer Park, TX NPDES: TX0007412	Surface Water	NPDES TX0007412	Surface water	250	0.148	0.49	0.00	0.00	0.16	0.00
				20	1.854	5.98	0.00	0.01	**1.99**	0.00
Washington Penn Plastics, Frankfort, KY NPDES: KY0097497	Surface Water	Surrogate NPDES KY0028410	Surface water	250	0.032	7.53	0.00	0.01	**2.51**	0.00
				20	0.399	94.12	0.03	0.12	**31.37**	0.00
Solvay - Houston Plant, Houston, TX NPDES: TX0007072	Surface Water	NPDES TX0007072	Surface water	350	0.024	4.44	0.00	0.01	**1.48**	0.00
				20	0.414	75.93	0.02	0.10	**25.31**	0.00
Natrium Plant, New Martinsville, WV NPDES: WV0004359	Surface Water	NPDES WV0004359	Surface water	250	0.022	0.00262	0.00	0.00	0.00	0.00
				20	0.274	0.0322	0.00	0.00	0.01	0.00
Leroy Quarry, Leroy, NY NPDES: NY0247189	Surface Water	Surrogate NPDES NY0030546	Surface water	250	0.019	0.71	0.00	0.00	0.24	0.00
				20	0.242	8.91	0.00	0.01	**2.97**	0.00
George C Marshall Space Flight Center, Huntsville, AL NPDES: AL0000221	Surface Water	Surrogate NPDES AL0025585	Surface water	250	0.01	0.2	0.00	0.00	0.07	0.00
				20	0.128	2.63	0.00	0.00	0.88	0.00

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Whelan Energy Center Power Plant, Hastings, NE NPDES: NE0113506	Surface Water	NPDES NE0113506	Surface water	250	0.009	2.92	0.00	0.00	0.97	0.00
				20	0.118	38.96	0.01	0.05	**12.99**	0.00
Akzo Nobel Surface Chemistry LLC, Morris, IL NPDES: IL0026069	Surface Water	NPDES IL0026069	Surface water	350	0.000329	0.000688	0.00	0.00	0.00	0.00
				20	0.006	0.0125	0.00	0.00	0.00	0.00
Solutia Nitro Site, Nitro, WV NPDES: WV0116181	Surface Water	Surrogate NPDES WV0023229	Surface water	350	0.000318	0.0000941	0.00	0.00	0.00	0.00
				20	0.006	0.00176	0.00	0.00	0.00	0.00
Amphenol Corporation - Columbia, Columbia, SC NPDES: SC0046264	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.000202	0.037	0.00	0.00	0.01	0.00
				20	0.004	0.74	0.00	0.00	0.25	0.00
Army Cold Regions Research & Engineering Lab, Hanover, NH NPDES: NH0001619	Surface Water	Surrogate NPDES NH0100099	Surface water	250	0.0002	0.000103	0.00	0.00	0.00	0.00
				20	0.0029	0.00154	0.00	0.00	0.00	0.00
Corning - Canton Plant, Canton, NY NPDES: NY0085006	Surface Water	Surrogate NPDES NY0034762	Surface water	250	0.0002	0.00034	0.00	0.00	0.00	0.00
				20	0.0028	0.0051	0.00	0.00	0.00	0.00

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Keeshan and Bost Chemical Co., Inc., Manvel, TX NPDES: TX0072168	Surface Water	NPDES TX0072168	Still body	350	0.000095	9.5	0.00	0.01	**3.17**	0.00
				20	0.002	200	0.06	0.25	**66.67**	0.00
Ames Rubber Corp Plant #1, Hamburg Boro, NJ NPDES: NJG000141	Surface Water	Surrogate NPDES NJ0000141	Surface water	250	0.00011	0.0149	0.00	0.00	0.00	0.00
				20	0.00133	0.18	0.00	0.00	0.06	0.00
Gorham, Providence, RI NPDES: RIG85E004	Surface Water	POTW (Ind.)	Surface water	250	0.0001	0.0129	0.00	0.00	0.00	0.00
				20	0.0012	0.13	0.00	0.00	0.04	0.00
Chemtura North and South Plants, Morgantown, WV NPDES: WV0004740	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Indorama Ventures Olefins, LLC, Sulphur, LA NPDES: LA0069850	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Emerson Power Transmission, Ithaca, NY NPDES: NY0002933	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
William E. Warne Power Plant,	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								

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Los Angeles County, CA NPDES: CA0059188										
Raytheon Aircraft Co(Was Beech Aircraft), Boulder, CO NPDES: COG315176	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: Spot Cleaning and Carpet Cleaning										
Boise State University, Boise, ID NPDES: IDG911006	Surface Water	Surrogate NPDES ID0023981	Surface water	300	0.00008	0.00388	0.00	0.00	0.00	0.00
				20	0.001	0.0485	0.00	0.00	0.02	0.00
Venetian Hotel And Casino, Las Vegas, NV NPDES: NV0022888	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
63,746 unknown sites NPDES: All POTW SIC	Surface Water or POTW	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: Industrial Processing Aid										
Occidental Chemical Corp Niagara Plant, Niagara Falls, NY NPDES: NY0003336	Surface Water	NPDES NY0003336	Still body	300	0.019	0.14	0.00	0.00	0.05	0.00
				20	0.292	2.2	0.00	0.00	0.73	0.00
Stepan Co Millsdale Road, Elwood, IL	Surface Water	NPDES IL0002453	Surface water	300	0.001	0.000419	0.00	0.00	0.00	0.00

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NPDES: IL0002453				20	0.008	0.00335	0.00	0.00	0.00	0.00
Entek International LLC, Lebanon, OR NPDES: N/A	Off-site Waste-water Treatment	No info on receiving facility; POTW (Ind.)	Surface water	300	0.38	9.3	0.00	0.01	**3.10**	0.00
				20	5.65	138.34	0.04	0.18	**46.11**	0.00
National Electrical Carbon Products Dba Morgan Adv Materials, Fostoria, OH NPDES: OH0052744	Off-site Waste-water Treatment	Receiving Facility: City of Fostoria; NPDES OH0052744	Surface water	300	0.008	0.15	0.00	0.00	0.05	0.00
				20	0.115	2.32	0.00	0.00	0.77	0.00
PPG Industries Inc Barberton, Barberton, OH NPDES: OH0024007	Off-site Waste-water Treatment	Receiving Facility: City of Barberton; NPDES OH0024007	Surface water	300	0.005	0.0141	0.00	0.00	0.00	0.00
				20	0.07	0.2	0.00	0.00	0.07	0.00
Daramic LLC, Corydon, IN NPDES: IN0020893	Surface Water	NPDES IN0020893	Surface water	300	0.008	0.0206	0.00	0.00	0.01	0.00
				20	0.114	0.29	0.00	0.00	0.10	0.00
OES: Commercial Printing and Copying										
Printing And Pub Sys Div, Weatherford, OK NPDES: OK0041785	Surface Water	Printing	Surface water	250	0.0002	0.00292	0.00	0.00	0.00	0.00
				20	0.0025	0.0365	0.00	0.00	0.01	0.00
OES: Other Commercial Uses										
Corning Hospital,				250	0.013	0.0271	0.00	0.00	0.01	0.00

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Corning, NY NPDES: NY0246701	Surface Water	Surrogate NPDES NY0025721	Surface water	20	0.159	0.33	0.00	0.00	0.11	0.00
Water Street Commercial Bldg, Dayton, OH NPDES: OH0141496	Surface Water	Surrogate NPDES OH0009521	Surface water	250	0.003	0.00564	0.00	0.00	0.00	0.00
				20	0.035	0.0658	0.00	0.00	0.02	0.00
Union Station North Wing Office Building, Denver, CO NPDES: COG315293	Surface Water	Surrogate NPDES CO0020095	Surface water	250	0.0004	0.0881	0.00	0.00	0.03	0.00
				20	0.00499	1.1	0.00	0.00	0.37	0.00
Confluence Park Apartments, Denver, CO NPDES: COG315339	Surface Water	Surrogate NPDES CO0020095	Surface water	250	0.00028	0.0617	0.00	0.00	0.02	0.00
				20	0.00354	0.77	0.00	0.00	0.26	0.00
Park Place Mixed Use Development, Annapolis, MD NPDES: MD0068861	Surface Water	Surrogate NPDES MD0052868	Still body	250	0.00027	9	0.00	0.01	**3.00**	0.00
				20	0.00334	110	0.03	0.14	**36.67**	0.00
Tree Top Inc Wenatchee Plant, Wenatchee, WA NPDES: WA0051527	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
Wynkoop Denver LLCP St, Denver, CO NPDES: COG603115	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								

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Greer Family Llc, South Burlington, VT NPDES: VT0001376	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
John Marshall III Site, Mclean, VA NPDES: VA0090093	Surface Water	Annual releases estimated to be <0.02 kg/year were not modeled, as they were determined to be unlikely to exceed the most sensitive COC using the most conservative input assumptions.								
OES: Process Solvent Recycling and Worker Handling of Wastes										
Clean Water Of New York Inc, Staten Island, NY NPDES: NY0200484	Surface Water	Surrogate NPDES NJ0000019	Still body	250	0.004	11.76	0.00	0.01	**3.92**	0.00
				20	0.047	138.24	0.04	0.18	**46.08**	0.00
Reserve Environmental Services, Ashtabula, OH NPDES: OH0098540	Surface Water						0.00	0.00	0.00	0.00
Veolia Es Technical Solutions LLC, Middlesex, NJ NPDES: NJ0020141	Off-site Waste-water Treatment	Receiving Facility: Middlesex Cnty UA; NPDES NJ0020141	Still body	250	24.1	2.85	0.00	0.00	0.95	0.00
				20	301.78	35.72	0.01	0.05	**11.91**	0.00
Clean Harbors Deer Park LLC,	Off-site Waste-	POTW (Ind.)	Surface water	250	0.35	8.57	0.00	0.01	**2.86**	0.00

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La Porte, TX NPDES: TX0005941	water Treatment			20	4.36	106.75	0.03	0.14	**35.58**	0.00
Clean Harbors El Dorado LLC, El Dorado, AR NPDES: AR0037800	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.04	0.98	0.00	0.00	0.33	0.00
				20	0.455	11.26	0.00	0.01	**3.75**	0.00
OES: Wastewater Treatment Plant (WWTP)										
New Rochelle STP, New Rochelle, NY NPDES: NY0026697	Surface Water	NPDES NY0026697	Still body	365	0.043	0.7	0.00	0.00	0.23	0.00
				20	0.786	12.79	0.00	0.02	**4.26**	0.00
Everett Water Pollution Control Facility, Everett, WA NPDES: WA0024490	Surface Water	NPDES WA0024490	Surface water	365	0.016	0.17	0.00	0.00	0.06	0.00
				20	0.299	3.11	0.00	0.00	**1.04**	0.00
Sullivan WWTP, Sullivan, MO NPDES: MO0104736	Surface Water	NPDES MO0104736	Surface water	365	0.01	0.61	0.00	0.00	0.20	0.00
				20	0.176	10.97	0.00	0.01	**3.66**	0.00
Sunnyside STP, Sunnyside, WA NPDES: WA0020991	Surface Water	NPDES WA0020991	Surface water	365	0.005	0.00673	0.00	0.00	0.00	0.00
				20	0.083	0.11	0.00	0.00	0.04	0.00
Port Of Sunnyside Industrial WWTF, Sunnyside, WA NPDES: WA0052426	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.26	0.00	0.00	0.09	0.00
				20	0.035	4.51	0.00	0.01	**1.50**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
U.S. Air Force Shaw AFB SC, Shaw AFB, SC NPDES: SC0024970	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.26	0.00	0.00	0.09	0.00
				20	0.032	4.12	0.00	0.01	**1.37**	0.00
Gnf-A Wilmington-Castle Hayne WWTP, Wilmington, NC NPDES: NC0001228	Surface Water	NPDES NC0001228	Surface water	365	0.0004	0.00194	0.00	0.00	0.00	0.00
				20	0.0067	0.034	0.00	0.00	0.01	0.00
Cameron Trading Post WWTP, Cameron, AZ NPDES: NN0021610	Surface Water	POTW (Ind.)	Surface water	365	0.0003	0.0387	0.00	0.00	0.01	0.00
				20	0.0047	0.64	0.00	0.00	0.21	0.00
Coal Grove WWTP, Coal Grove, OH NPDES: OH0104558	Surface Water	NPDES OH0029432	Surface water	365	0.0002	0.0000127	0.00	0.00	0.00	0.00
				20	0.0031	0.00019	0.00	0.00	0.00	0.00

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Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
OES: Adhesives, Sealants, Paints, and Coatings										
Able Electropolishing Co Inc, Chicago, IL NPDES: Not available	POTW	Adhesives and Sealants Manuf.	Surface water	250	0.298	7.28	0.00	0.01	**2.43**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Garlock Sealing Technologies, Palmyra, NY, NPDES: NY0000078	Surface Water	NPDES NY0000078	Surface water	250	0.00033	0.00716	0.00	0.00	0.00	0.00
				20	0.00407	0.0889	0.00	0.00	0.03	0.00
Ls Starrett Co, Athol, MA NPDES: MAR05B615	Surface Water	Not assessed (below the min risk level).								
Aerojet Rocketdyne, Inc., East Camden, AR NPDES: AR0051071, ARR00A521, ARR00A520	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Best One Tire & Service, Nashville, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Bridgestone Aircraft Tire (Usa), Inc., Mayodan, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Clayton Homes Inc, Oxford, NC	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
NPDES: Not available										
Cmh Manufacturing, Inc. Dba Schult Homes - Plant 958, Richfield, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Delphi Thermal Systems, Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	250	0.013	1.1	0.00	0.00	0.37	0.00
				20	0.16	13.5	0.00	0.02	**4.50**	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Green Bay Packaging Inc - Coon Rapids, Coon Rapids, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Mastercraft Boat Company, Vonore, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Michelin Aircraft Tire Company, Norwood, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
M-Tek, Inc, Manchester, TN	Surface Water		Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00

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NPDES: Not available	POTW	Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Olin Corp, East Alton, IL NPDES: IL0000230	Surface Water	NPDES IL0000230	Surface water	250	0.013	0.18	0.00	0.00	0.06	0.00
				20	0.16	2.26	0.00	0.00	0.75	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Parker Hannifin Corp – Paraflex Division, Manitowoc, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Parrish Tire Company, Yadkinville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Republic Doors And Frames, Mckenzie, TN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Ro-Lab Rubber Company Inc., Tracy, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Royale Comfort Seating, Inc. - Plant No. 1,	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

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Taylorsville, NC NPDES: Not available										
Snider Tire, Inc., Statesville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Snyder Paper Corporation, Hickory, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Stellana Us, Lake Geneva, WI NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Thomas Built Buses - Courtesy Road, High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Unicel Corp, Escondido, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Acme Finishing Co Llc, Elk Grove Village, IL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

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Aerojet Rocketdyne, Inc., Rancho Cordova, CA NPDES: CA0004111	Surface Water	NPDES CA0004111	Surface water	250	0.013	0.000818	0.00	0.00	0.00	0.00
				20	0.16	0.0101	0.00	0.00	0.00	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Allegheny Cnty Airport Auth/ Pgh Intl Airport, Coroapolis Pittsburgh, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Amphenol Corp – Aerospace Operations, Sidney, NY NPDES: NY0003824	Surface Water	NPDES NY0003824	Surface water	250	0.013	0.0631	0.00	0.00	0.02	0.00
				20	0.16	0.78	0.00	0.00	0.26	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Aprotech Powertrain, Asheville, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Coating & Converting Tech Corp/ Adhesive Coatings, Philadelphia, PA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
				250	0.013	1.67	0.00	0.00	0.56	0.00

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Corpus Christi Army Depot, Corpus Christi, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Electronic Data Systems Camp Pendleton, Camp Pendleton, CA NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Florida Production Engineering, Inc., Ormond Beach, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Goodrich Corporation, Jacksonville, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Kasai North America Inc, Madison Plant, Madison, MS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Kirtland Air Force Base, Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
				250	0.013	1.67	0.00	0.00	0.56	0.00

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Marvin Windows & Doors, Warroad, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Mcneilus Truck & Manufacturing Inc, Dodge Center, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Metal Finishing Co. – Wichita (S Mclean Blvd), Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Murakami Manufacturing Usa Inc, Campbellsville, KY NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Peterbilt Motors Denton Facility, Denton, TX NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Portsmouth Naval Shipyard, Kittery, ME NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

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R.D. Henry & Co., Wichita, KS NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Raytheon Company, Portsmouth, RI NPDES: RI0000281	Surface Water	NPDES RI0000281	Still body	250	0.013	10.83	0.00	0.01	**3.61**	0.00
				20	0.16	133.33	0.04	0.17	**44.44**	0.00
	POTW	No info on receiving facility; Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
Rehau Inc, Cullman, AL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Rotochopper Inc, Saint Martin, MN NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Rubber Applications, Mulberry, FL NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Sapa Precision Tubing Rockledge, Llc, Rockledge, FL	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
NPDES: Not available										
Thomas & Betts, Albuquerque, NM NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Thomas Built Buses - Fairfield Road, High Point, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Timco, Dba Haeco Americas Airframe Services, Greensboro, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Trelleborg Coated Systems Us, Inc – Grace Advanced Materials, Rutherfordton, NC NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
U.S. Coast Guard Yard - Curtis Bay, Curtis Bay, MD NPDES: Not available	Surface Water	Adhesives and Sealants Manuf.	Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00
	POTW			250	0.013	0.32	0.00	0.00	0.11	0.00
Viracon Inc, Owatonna, MN	Surface Water		Surface water	250	0.013	1.67	0.00	0.00	0.56	0.00
				20	0.16	20.57	0.01	0.03	**6.86**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
NPDES: Not available	POTW	Adhesives and Sealants Manuf.		250	0.013	0.32	0.00	0.00	0.11	0.00
OES: Commercial Printing and Copying										
Printing And Pub Sys Div, Weatherford, OK NPDES: OK0041785	Surface Water	Printing	Surface water	250	0.0002	0.00292	0.00	0.00	0.00	0.00
				20	0.0025	0.0365	0.00	0.00	0.01	0.00
OES: Industrial Processing Aid										
Occidental Chemical Corp Niagara Plant, Niagara Falls, NY NPDES: NY0003336	Surface Water	NPDES NY0003336	Still body	300	0.019	0.14	0.00	0.00	0.05	0.00
				20	0.292	2.2	0.00	0.00	0.73	0.00
Stepan Co Millsdale Road, Elwood, IL NPDES: IL0002453	Surface Water	NPDES IL0002453	Surface water	300	0.001	0.000419	0.00	0.00	0.00	0.00
				20	0.008	0.00335	0.00	0.00	0.00	0.00
Entek International LLC, Lebanon, OR NPDES: N/A	Off-site Waste-water Treatment	No info on receiving facility; POTW (Ind.)	Surface water	300	0.38	9.3	0.00	0.01	**3.10**	0.00
				20	5.65	138.34	0.04	0.18	**46.11**	0.00
National Electrical Carbon Products Dba Morgan Adv Materials, Fostoria, OH NPDES: OH0052744	Off-site Waste-water Treatment	Receiving Facility: City of Fostoria; NPDES OH0052744	Surface water	300	0.008	0.15	0.00	0.00	0.05	0.00
				20	0.115	2.32	0.00	0.00	0.77	0.00
PPG Industries Inc Barberton, Barberton, OH NPDES: OH0024007	Off-site Waste-water Treatment	Receiving Facility: City of Barberton; NPDES OH0024007	Surface water	300	0.005	0.0141	0.00	0.00	0.00	0.00
				20	0.07	0.2	0.00	0.00	0.07	0.00
Daramic LLC, Corydon, IN NPDES: IN0020893	Surface Water	NPDES IN0020893	Surface water	300	0.008	0.0206	0.00	0.00	0.01	0.00
				20	0.114	0.29	0.00	0.00	0.10	0.00

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OES: Manufacturing										
Axiall Corporation, Westlake, LA NPDES: LA0007129	Surface Water	NPDES LA0007129	Surface water	350	1.266	0.0051	0.00	0.00	0.00	0.00
				20	22.15	0.0897	0.00	0.00	0.03	0.00
Olin Blue Cube, Freeport, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.069	2.42	0.00	0.00	0.81	0.00
				20	1.2	42.14	0.01	0.05	**14.05**	0.00
Solvents & Chemicals, Pearland, TX NPDES: Not available	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.53	0.00	0.00	0.18	0.00
				20	0.265	9.48	0.00	0.01	**3.16**	0.00
	Surface Water	Organic Chemicals Manuf.	Surface water	350	0.015	2.77	0.00	0.00	0.92	0.00
				20	0.265	49.91	0.02	0.06	**16.64**	0.00
Occidental Chemical Corp Wichita, Wichita, KS NPDES: KS0096903 and Organic Chem MFG SIC	Surface Water	Surrogate NPDES KS0043036	Surface water	350	0.015	0.07	0.00	0.00	0.02	0.00
				20	0.265	1.33	0.00	0.00	0.44	0.00
	Off-site Waste-water Treatment	Organic Chemicals Manuf.	Surface water	350	0.015	0.53	0.00	0.00	0.18	0.00
				20	0.265	9.48	0.00	0.01	**3.16**	0.00
OES: Waste Water Treatment Plant (WWTP)										
New Rochelle STP, New Rochelle, NY NPDES: NY0026697	Surface Water	NPDES NY0026697	Still body	365	0.043	0.7	0.00	0.00	0.23	0.00
				20	0.786	12.79	0.00	0.02	**4.26**	0.00
Everett Water Pollution Control Facility, Everett, WA NPDES: WA0024490	Surface Water	NPDES WA0024490	Surface water	365	0.016	0.17	0.00	0.00	0.06	0.00
				20	0.299	3.11	0.00	0.00	**1.04**	0.00
Sullivan WWTP,				365	0.01	0.61	0.00	0.00	0.20	0.00

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Sullivan, MO NPDES: MO0104736	Surface Water	NPDES MO0104736	Surface water	20	0.176	10.97	0.00	0.01	**3.66**	0.00
Sunnyside STP, Sunnyside, WA NPDES: WA0020991	Surface Water	NPDES WA0020991	Surface water	365	0.005	0.00673	0.00	0.00	0.00	0.00
				20	0.083	0.11	0.00	0.00	0.04	0.00
Port Of Sunnyside Industrial WWTF, Sunnyside, WA NPDES: WA0052426	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.26	0.00	0.00	0.09	0.00
				20	0.035	4.51	0.00	0.01	**1.50**	0.00
U.S. Air Force Shaw AFB SC, Shaw AFB, SC NPDES: SC0024970	Surface Water	POTW (Ind.)	Surface water	365	0.002	0.26	0.00	0.00	0.09	0.00
				20	0.032	4.12	0.00	0.01	**1.37**	0.00
Gnf-A Wilmington-Castle Hayne WWTP, Wilmington, NC NPDES: NC0001228	Surface Water	NPDES NC0001228	Surface water	365	0.0004	0.00194	0.00	0.00	0.00	0.00
				20	0.0067	0.034	0.00	0.00	0.01	0.00
Cameron Trading Post WWTP, Cameron, AZ NPDES: NN0021610	Surface Water	POTW (Ind.)	Surface water	365	0.0003	0.0387	0.00	0.00	0.01	0.00
				20	0.0047	0.64	0.00	0.00	0.21	0.00
Coal Grove WWTP, Coal Grove, OH NPDES: OH0104558	Surface Water	NPDES OH0029432	Surface water	365	0.0002	0.0000127	0.00	0.00	0.00	0.00
				20	0.0031	0.00019	0.00	0.00	0.00	0.00
OES: Other Commercial Uses										
Corning Hospital,				250	0.013	0.0271	0.00	0.00	0.01	0.00

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Corning, NY NPDES: NY0246701	Surface Water	Surrogate NPDES NY0025721	Surface water	20	0.159	0.33	0.00	0.00	0.11	0.00
Water Street Commercial Bldg, Dayton, OH NPDES: OH0141496	Surface Water	Surrogate NPDES OH0009521	Surface water	250	0.003	0.00564	0.00	0.00	0.00	0.00
				20	0.035	0.0658	0.00	0.00	0.02	0.00
Union Station North Wing Office Building, Denver, CO NPDES: COG315293	Surface Water	Surrogate NPDES CO0020095	Surface water	250	0.0004	0.0881	0.00	0.00	0.03	0.00
				20	0.00499	1.1	0.00	0.00	0.37	0.00
Confluence Park Apartments, Denver, CO NPDES: COG315339	Surface Water	Surrogate NPDES CO0020095	Surface water	250	0.00028	0.0617	0.00	0.00	0.02	0.00
				20	0.00354	0.77	0.00	0.00	0.26	0.00
Park Place Mixed Use Development, Annapolis, MD NPDES: MD0068861	Surface Water	Surrogate NPDES MD0052868	Still body	250	0.00027	9	0.00	0.01	**3.00**	0.00
				20	0.00334	110	0.03	0.14	**36.67**	0.00
Tree Top Inc Wenatchee Plant, Wenatchee, WA NPDES: WA0051527	Surface Water	Not assessed (below the min risk level).								
Wynkoop Denver LLCP St, Denver, CO NPDES: COG603115	Surface Water	Not assessed (below the min risk level).								

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Greer Family Llc, South Burlington, VT NPDES: VT0001376	Surface Water	Not assessed (below the min risk level).								
John Marshall III Site, Mclean, VA NPDES: VA0090093	Surface Water	Not assessed (below the min risk level).								
OES: Other Industrial Uses										
Eli Lilly And Company-Lilly Tech Ctr, Indianapolis, IN NPDES: IN0003310	Surface Water	NPDES IN0003310	Surface water	250	1.553	9.03	0.00	0.01	**3.01**	0.00
				20	19.41	113.09	0.04	0.14	**37.70**	0.00
Oxy Vinyls LP - Deer Park Pvc, Deer Park, TX NPDES: TX0007412	Surface Water	NPDES TX0007412	Surface water	250	0.148	0.49	0.00	0.00	0.16	0.00
				20	1.854	5.98	0.00	0.01	**1.99**	0.00
Washington Penn Plastics, Frankfort, KY NPDES: KY0097497	Surface Water	Surrogate NPDES KY0028410	Surface water	250	0.032	7.53	0.00	0.01	**2.51**	0.00
				20	0.399	94.12	0.03	0.12	**31.37**	0.00
Solvay - Houston Plant, Houston, TX NPDES: TX0007072	Surface Water	NPDES TX0007072	Surface water	350	0.024	4.44	0.00	0.01	**1.48**	0.00
				20	0.414	75.93	0.02	0.10	**25.31**	0.00
Natrium Plant, New Martinsville, WV NPDES: WV0004359	Surface Water	NPDES WV0004359	Surface water	250	0.022	0.00262	0.00	0.00	0.00	0.00
				20	0.274	0.0322	0.00	0.00	0.01	0.00
Leroy Quarry,				250	0.019	0.71	0.00	0.00	0.24	0.00

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Leroy, NY NPDES: NY0247189	Surface Water	Surrogate NPDES NY0030546	Surface water	20	0.242	8.91	0.00	0.01	**2.97**	0.00
George C Marshall Space Flight Center, Huntsville, AL NPDES: AL0000221	Surface Water	Surrogate NPDES AL0025585	Surface water	250	0.01	0.2	0.00	0.00	0.07	0.00
				20	0.128	2.63	0.00	0.00	0.88	0.00
Whelan Energy Center Power Plant, Hastings, NE NPDES: NE0113506	Surface Water	NPDES NE0113506	Surface water	250	0.009	2.92	0.00	0.00	0.97	0.00
				20	0.118	38.96	0.01	0.05	**12.99**	0.00
Army Cold Regions Research & Engineering Lab, Hanover, NH NPDES: NH0001619	Surface Water	Surrogate NPDES NH0100099	Surface water	250	0.0002	0.000103	0.00	0.00	0.00	0.00
				20	0.0029	0.00154	0.00	0.00	0.00	0.00
Corning - Canton Plant, Canton, NY NPDES: NY0085006	Surface Water	Surrogate NPDES NY0034762	Surface water	250	0.0002	0.00034	0.00	0.00	0.00	0.00
				20	0.0028	0.0051	0.00	0.00	0.00	0.00
Ames Rubber Corp Plant #1, Hamburg Boro, NJ NPDES: NJG000141	Surface Water	Surrogate NPDES NJ0000141	Surface water	250	0.00011	0.0149	0.00	0.00	0.00	0.00
				20	0.00133	0.18	0.00	0.00	0.06	0.00
Gorham, Providence, RI NPDES: RIG85E004	Surface Water	POTW (Ind.)	Surface water	250	0.0001	0.0129	0.00	0.00	0.00	0.00
				20	0.0012	0.13	0.00	0.00	0.04	0.00
Akzo Nobel Surface Chemistry LLC, Morris, IL NPDES: IL0026069	Surface Water	NPDES IL0026069	Surface water	350	0.000329	0.000688	0.00	0.00	0.00	0.00
				20	0.006	0.0125	0.00	0.00	0.00	0.00
Solutia Nitro Site, Nitro, WV	Surface Water		Surface water	350	0.000318	0.0000941	0.00	0.00	0.00	0.00

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NPDES: WV0116181		Surrogate NPDES WV0023229		20	0.006	0.00176	0.00	0.00	0.00	0.00
Amphenol Corporation - Columbia, Columbia, SC NPDES: SC0046264	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.000202	0.037	0.00	0.00	0.01	0.00
				20	0.004	0.74	0.00	0.00	0.25	0.00
Keeshan and Bost Chemical Co., Inc., Manvel, TX NPDES: TX0072168	Surface Water	NPDES TX0072168	Still body	350	0.000095	9.5	0.00	0.01	**3.17**	0.00
				20	0.002	200	0.06	0.25	**66.67**	0.00
Chemtura North and South Plants, Morgantown, WV NPDES: WV0004740	Surface Water	Not assessed (below the min risk level).								
Indorama Ventures Olefins, LLC, Sulphur, LA NPDES: LA0069850	Surface Water	Not assessed (below the min risk level).								
Emerson Power Transmission, Ithaca, NY NPDES: NY0002933	Surface Water	Not assessed (below the min risk level).								
William E. Warne Power Plant, Los Angeles County, CA NPDES: CA0059188	Surface Water	Not assessed (below the min risk level).								
Raytheon Aircraft Co(Was Beech Aircraft), Boulder, CO	Surface Water	Not assessed (below the min risk level).								

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NPDES: COG315176										
OES: OTVD (Includes releases for Closed-Loop Degreasing, Conveyorized Degreasing, Web Degreasing, and Metalworking Fluids)										
Texas Instruments, Inc., Attleboro, MA NPDES: MA0001791	Surface Water	NPDES MA0001791	Surface water	260	0.005	0.0188	0.00	0.00	0.01	0.00
				20	0.067	0.25	0.00	0.00	0.08	0.00
Accellent Inc/Collegeville Microcoax, Collegeville, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.002	0.0425	0.00	0.00	0.01	0.00
				20	0.029	0.62	0.00	0.00	0.21	0.00
Ametek Inc. U.S. Gauge Div., Sellersville, PA NPDES: PA0056014	Surface Water	Surrogate NPDES PA0020460	Surface water	260	0.001	0.0619	0.00	0.00	0.02	0.00
				20	0.011	0.68	0.00	0.00	0.23	0.00
Atk-Allegany Ballistics Lab (Nirop), Keyser, WV NPDES: WV0020371	Surface Water	NPDES WV0020371	Surface water	260	0.0005	0.00311	0.00	0.00	0.00	0.00
				20	0.0061	0.0373	0.00	0.00	0.01	0.00
Handy & Harman Tube Co/East Norriton, Norristown, PA NPDES: PA0011436	Surface Water	Not assessed (below the min risk level).								
US Nasa Michoud Assembly Facility, New Orleans, LA NPDES: LA0052256	Surface Water	Surrogate NPDES LA0003280	Still body	260	1.96	765.63	0.24	0.97	**255.21**	0.01
				20	25.44	9937.5	**3.11**	**12.61**	**3312.50**	0.19
				260	0.13	10.97	0.00	0.01	**3.66**	0.00

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GM Components Holdings LLC, Lockport, NY NPDES: NY0000558	Surface Water	NPDES NY0000558	Surface water	20	1.71	144.47	0.05	0.18	**48.16**	0.00
Akebono Elizabethtown Plant, Elizabethtown, KY NPDES: KY0089672	Surface Water	Surrogate NPDES KY0022039	Surface water	260	0.07	4.87	0.00	0.01	**1.62**	0.00
				20	0.897	62.38	0.02	0.08	**20.79**	0.00
Delphi Harrison Thermal Systems, Dayton, OH NPDES: OH0009431	Surface Water	NPDES OH0009431	Surface water	260	0.04	0.0752	0.00	0.00	0.03	0.00
				20	0.465	0.87	0.00	0.00	0.29	0.00
Chemours Company Fc LLC, Washington, WV NPDES: WV0001279	Surface Water	NPDES WV0001279	Surface water	260	0.03	0.00301	0.00	0.00	0.00	0.00
				20	0.334	0.0335	0.00	0.00	0.01	0.00
Equistar Chemicals Lp, La Porte, TX NPDES: TX0119792	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.02	2.22	0.00	0.00	0.74	0.00
				20	0.218	24.44	0.01	0.03	**8.15**	0.00
GE Aviation, Lynn, MA NPDES: MA0003905	Surface Water	NPDES MA0003905	Still water	260	0.01	0.0425	0.00	0.00	0.01	0.00
				20	0.128	0.54	0.00	0.00	0.18	0.00
Certa Vandalia LLC, Vandalia, OH NPDES: OH0122751	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.01	1.11	0.00	0.00	0.37	0.00
				20	0.107	11.89	0.00	0.02	**3.96**	0.00
GM Components Holdings LLC Kokomo Ops,	Surface Water	NPDES IN0001830	Surface water	260	0.01	0.2	0.00	0.00	0.07	0.00
				20	0.086	1.73	0.00	0.00	0.58	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Kokomo, IN NPDES: IN0001830										
Amphenol Corp-Aerospace Operations, Sidney, NY NPDES: NY0003824	Surface Water	NPDES NY0003824	Surface water	260	0.01	0.0486	0.00	0.00	0.02	0.00
				20	0.082	0.4	0.00	0.00	0.13	0.00
Emerson Power Trans Corp, Maysville, KY NPDES: KY0100196	Surface Water	Surrogate NPDES KY0020257	Surface water	260	0.01	0.0004	0.00	0.00	0.00	0.00
				20	0.081	0.00522	0.00	0.00	0.00	0.00
Olean Advanced Products, Olean, NY NPDES: NY0073547	Surface Water	Surrogate NPDES NY0027162	Surface water	260	0.01	0.0188	0.00	0.00	0.01	0.00
				20	0.068	0.13	0.00	0.00	0.04	0.00
Hollingsworth Saco Lowell, Easley, SC NPDES: SC0046396	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00469	0.52	0.00	0.00	0.17	0.00
				20	0.061	6.78	0.00	0.01	**2.26**	0.00
Trelleborg YSH Incorporated Sandusky Plant, Sandusky, MI NPDES: MI0028142	Surface Water	NPDES MI0028142	Surface water	260	0.0036	1.76	0.00	0.00	0.59	0.00
				20	0.047	23.04	0.01	0.03	**7.68**	0.00
Timken Us Corp Honea Path, Honea Path, SC NPDES: SC0047520	Surface Water	Surrogate NPDES SC0000698	Surface water	260	0.00355	1.06	0.00	0.00	0.35	0.00
				20	0.0462	13.77	0.00	0.02	**4.59**	0.00
Johnson Controls Incorporated, Wichita, KS NPDES: KS0000850	Surface Water	NPDES KS0000850	Surface water	260	0.00228	0.0548	0.00	0.00	0.02	0.00
				20	0.0296	0.72	0.00	0.00	0.24	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
National Railroad Passenger Corporation (Amtrak) Wilmington Maintenance Facility, Wilmington, DE NPDES: DE0050962	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00203	0.23	0.00	0.00	0.08	0.00
				20	0.026	2.89	0.00	0.00	0.96	0.00
Electrolux Home Products (Formerly Frigidaire), Greenville, MI NPDES: MI0002135	Surface Water	NPDES MI0002135	Surface water	260	0.00201	0.0171	0.00	0.00	0.01	0.00
				20	0.026	0.22	0.00	0.00	0.07	0.00
Rex Heat Treat Lansdale Inc, Lansdale, PA NPDES: PA0052965	Surface Water	Surrogate NPDES PA0026182	Surface water	260	0.00194	0.0523	0.00	0.00	0.02	0.00
				20	0.025	0.67	0.00	0.00	0.22	0.00
Carrier Corporation, Syracuse, NY NPDES: NY0001163	Surface Water	NPDES NY0001163	Still water	260	0.00177	0.22	0.00	0.00	0.07	0.00
				20	0.023	2.84	0.00	0.00	0.95	0.00
Cascade Corp (0812100207), Springfield, OH NPDES: OH0085715	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.00117	0.13	0.00	0.00	0.04	0.00
				20	0.015	1.67	0.00	0.00	0.56	0.00
USAF-Wurtsmith Afb, Oscoda, MI NPDES: MI0042285	Surface Water	Surrogate NPDES MI0028282	Surface water	260	0.00115	0.000753	0.00	0.00	0.00	0.00
				20	0.015	0.00983	0.00	0.00	0.00	0.00
AAR Mobility Systems, Cadillac, MI NPDES: MI0002640	Surface Water	Surrogate NPDES MI0020257	Surface water	260	0.00112	0.00916	0.00	0.00	0.00	0.00
				20	0.014	0.11	0.00	0.00	0.04	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Eaton Mdh Company Inc, Kearney, NE NPDES: NE0114405	Surface Water	Surrogate NPDES NE0052647	Still water	260	0.00107	0.13	0.00	0.00	0.04	0.00
				20	0.014	1.69	0.00	0.00	0.56	0.00
Lake Region Medical, Trappe, PA NPDES: PA0042617	Surface Water	NPDES PA0042617	Surface water	260	0.0005	0.0106	0.00	0.00	0.00	0.00
				20	0.007	0.15	0.00	0.00	0.05	0.00
Motor Components L L C, Elmira, NY NPDES: NY0004081	Surface Water	NPDES NY0004081	Surface water	260	0.00096	0.0618	0.00	0.00	0.02	0.00
				20	0.0125	0.83	0.00	0.00	0.28	0.00
Salem Tube Mfg, Greenville, PA NPDES: PA0221244	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000897	0.0997	0.00	0.00	0.03	0.00
				20	0.012	1.33	0.00	0.00	0.44	0.00
GE (Greenville) Gas Turbines LLC, Greenville, SC NPDES: SC0003484	Surface Water	NPDES SC0003484	Surface water	260	0.000806	0.0821	0.00	0.00	0.03	0.00
				20	0.01	1.02	0.00	0.00	0.34	0.00
Parker Hannifin Corporation, Waverly, OH NPDES: OH0104132	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000747	0.083	0.00	0.00	0.03	0.00
				20	0.01	1.11	0.00	0.00	0.37	0.00
Mahle Engine Components Usa Inc, Muskegon, MI NPDES: MI0004057	Surface Water	NPDES MI0004057	Surface water	260	0.000742	0.0336	0.00	0.00	0.01	0.00
				20	0.01	0.45	0.00	0.00	0.15	0.00
General Electric Company - Waynesboro, Waynesboro, VA	Surface Water	NPDES VA0002402	Surface water	260	0.000733	0.00705	0.00	0.00	0.00	0.00
				20	0.01	0.0962	0.00	0.00	0.03	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
NPDES: VA0002402										
Globe Engineering Co Inc, Wichita, KS NPDES: KS0086703	Surface Water	Surrogate NPDES KS0043036	Surface water	260	0.00173	0.00853	0.00	0.00	0.00	0.00
				20	0.023	0.11	0.00	0.00	0.04	0.00
Gayston Corp, Dayton, OH NPDES: OH0127043	Surface Water	Surrogate NPDES OH0024881	Surface water	260	0.000643	0.00121	0.00	0.00	0.00	0.00
				20	0.008	0.015	0.00	0.00	0.01	0.00
Styrolution America LLC, Channahon, IL NPDES: IL0001619	Surface Water	NPDES IL0001619	Surface water	260	0.000637	0.000221	0.00	0.00	0.00	0.00
				20	0.008	0.00278	0.00	0.00	0.00	0.00
Remington Arms Co Inc, Ilion, NY NPDES: NY0005282	Surface Water	NPDES NY0005282	Surface water	260	0.000612	0.000799	0.00	0.00	0.00	0.00
				20	0.008	0.0104	0.00	0.00	0.00	0.00
United Technologies Corporation, Pratt And Whitney Division, East Hartford, CT NPDES: CT0001376	Surface Water	NPDES CT0001376	Surface water	260	0.00048	0.0000822	0.00	0.00	0.00	0.00
				20	0.006	0.00103	0.00	0.00	0.00	0.00
Atk-Allegany Ballistics Lab (Nirop), Keyser, WV NPDES: WV0020371	Surface Water	NPDES WV0020371	Surface water	260	0.00047	0.00292	0.00	0.00	0.00	0.00
				20	0.006	0.0373	0.00	0.00	0.01	0.00
Sperry & Rice Manufacturing Co LLC, Brookville, IN NPDES: IN0001473	Surface Water	NPDES IN0001473	Surface water	260	0.000328	0.00569	0.00	0.00	0.00	0.00
				20	0.004	0.0694	0.00	0.00	0.02	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Owt Industries, Pickens, SC NPDES: SC0026492	Surface Water	NPDES SC0026492	Surface water	260	0.000314	0.00213	0.00	0.00	0.00	0.00
				20	0.004	0.0272	0.00	0.00	0.01	0.00
Boler Company, Hillsdale, MI NPDES: MI0053651	Surface Water	Surrogate NPDES MI0022136	Surface water	260	0.000269	0.0204	0.00	0.00	0.01	0.00
				20	0.003	0.23	0.00	0.00	0.08	0.00
Mccanna Inc., Carpentersville, IL NPDES: IL0071340	Surface Water	Surrogate NPDES IL0027944	Surface water	260	0.000268	0.000911	0.00	0.00	0.00	0.00
				20	0.003	0.0102	0.00	0.00	0.00	0.00
Cutler Hammer, Horseheads, NY NPDES: NY0246174	Surface Water	Surrogate NPDES NY0004081	Surface water	260	0.000238	0.0153	0.00	0.00	0.01	0.00
				20	0.003	0.19	0.00	0.00	0.06	0.00
US Air Force Offutt Afb Ne, Offutt A F B, NE NPDES: NE0121789	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000159	0.0177	0.00	0.00	0.01	0.00
				20	0.002	0.22	0.00	0.00	0.07	0.00
Troxel Company, Moscow, TN NPDES: TN0000451	Surface Water	NPDES TN0000451	Surface water	260	0.000134	0.000741	0.00	0.00	0.00	0.00
				20	0.002	0.0111	0.00	0.00	0.00	0.00
Austin Tube Prod, Baldwin, MI NPDES: MI0054224	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.000114	0.0127	0.00	0.00	0.00	0.00
				20	0.001	0.11	0.00	0.00	0.04	0.00
LS Starrett Precision Tools, Athol, MA NPDES: MA0001350	Surface Water	NPDES MA0001350	Surface water	260	0.000102	0.00153	0.00	0.00	0.00	0.00
				20	0.001	0.015	0.00	0.00	0.01	0.00

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Avx Corp, Raleigh, NC NPDES: NC0089494	Surface Water	Primary Metal Forming Manuf.	Surface water	260	0.0000883	0.00981	0.00	0.00	0.00	0.00
				20	0.001	0.11	0.00	0.00	0.04	0.00
Indian Head Division, Naval Surface Warfare Center, Indian Head, MD NPDES: MD0003158	Surface Water	Not assessed (below the min risk level).								
General Dynamics Ordnance Tactical Systems, Red Lion, PA NPDES: PA0043672	Surface Water	Not assessed (below the min risk level).								
Trane Residential Solutions - Fort Smith, Fort Smith, AR NPDES: AR0052477	Surface Water	Not assessed (below the min risk level).								
Lexmark International Inc., Lexington, KY NPDES: KY0097624	Surface Water	Not assessed (below the min risk level).								
Alliant Techsystems Operations LLC, Elkton, MD NPDES: MD0000078	Surface Water	Not assessed (below the min risk level).								
Daikin Applied America, Inc. (Formally Mcquay International), Scottsboro, AL NPDES: AL0069701	Surface Water	Not assessed (below the min risk level).								

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Beechcraft Corporation, Wichita, KS NPDES: KS0000183	Surface Water	Not assessed (below the min risk level).								
Federal-Mogul Corp, Scottsville, KY NPDES: KY0106585	Surface Water	Not assessed (below the min risk level).								
Cessna Aircraft Co (Pawnee Facility), Wichita, KS NPDES: KS0000647	Surface Water	Not assessed (below the min risk level).								
N.G.I, Parkersburg, WV NPDES: WV0003204	Surface Water	Not assessed (below the min risk level).								
Hyster-Yale Group, Inc, Sulligent, AL NPDES: AL0069787	Surface Water	Not assessed (below the min risk level).								
Hitachi Electronic Devices (Usa), Inc., Greenville, SC NPDES: SC0048411	Surface Water	Not assessed (below the min risk level).								
OES: Process Solvent Recycling and Worker Handling of Wastes										
Clean Water Of New York Inc, Staten Island, NY NPDES: NY0200484	Surface Water	Surrogate NPDES NJ0000019	Still body	250	0.004	11.76	0.00	0.01	**3.92**	0.00
				20	0.047	138.24	0.04	0.18	**46.08**	0.00
Reserve Environmental Services, Ashtabula, OH NPDES: OH0098540	Surface Water						0.00	0.00	0.00	0.00
Veolia Es Technical Solutions LLC, Middlesex, NJ NPDES: NJ0020141	Off-site Waste-water Treatment	Receiving Facility: Middlesex Cnty UA;	Still body	250	24.1	2.85	0.00	0.00	0.95	0.00
				20	301.78	35.72	0.01	0.05	**11.91**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
		NPDES NJ0020141								
Clean Harbors Deer Park LLC, La Porte, TX NPDES: TX0005941	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.35	8.57	0.00	0.01	**2.86**	0.00
				20	4.36	106.75	0.03	0.14	**35.58**	0.00
Clean Harbors El Dorado LLC, El Dorado, AR NPDES: AR0037800	Off-site Waste-water Treatment	POTW (Ind.)	Surface water	250	0.04	0.98	0.00	0.00	0.33	0.00
				20	0.455	11.26	0.00	0.01	**3.75**	0.00
OES: Processing as a Reactant										
440 unknown sites NPDES: Not applicable	Off-site Waste-water Treatment	Organic Chemicals Manufacture	Surface water	350	0.005	0.18	0.00	0.00	0.06	0.00
				20	0.089	3.13	0.00	0.00	**1.04**	0.00
	Surface Water	Organic Chemicals Manufacture	Surface water	350	0.005	0.92	0.00	0.00	0.31	0.00
				20	0.089	16.45	0.01	0.02	**5.48**	0.00
Arkema Inc. Calvert City, KY NPDES: KY0003603	Surface Water	NPDES KY0003603	Surface water	350	0.017	0.000737	0.00	0.00	0.00	0.00
				20	0.295	0.128	0.00	0.00	0.04	0.00
US DOE Paducah Site, Kevil, KY NPDES: KY0102083	Surface Water	Not assessed (below the min risk level).								
GNF-A Wilmington-Castle Hayne, Wilmington NC NPDES: NC0001228	Surface Water	Not assessed (below the min risk level).								
Solvay - Houston Plant, Houston, TX NPDES: TX0007072	Surface Water	NPDES TX0007072	Surface water	350	0.024	4.44	0.00	0.01	**1.48**	0.00
				20	0.414	75.93	0.02	0.10	**25.31**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Honeywell International - Geismar Complex, Geismar, LA NPDES: LA0006181	Surface Water	NPDES LA0006181	Surface water	350	0.0128	0.0000518	0.00	0.00	0.00	0.00
				20	0.224	0.000907	0.00	0.00	0.00	0.00
Praxair Technology Center, Tonawanda, NY NPDES: NY0000281	Surface Water	NPDES NY0000281	Still body	350	0.00169	169	0.05	0.21	**56.33**	0.00
				20	0.03	3000	0.94	**3.81**	**1000.00**	0.06
US DOE Paducah Site, Kevil, KY NPDES: KY0102083	Surface Water	Not assessed (below the min risk level).								
GNF-A Wilmington-Castle Hayne, Wilmington NC NPDES: NC0001228	Surface Water	Not assessed (below the min risk level).								
OES: Repackaging										
Hubbard-Hall Inc, Waterbury, CT NPDES: Unknown	Off-site Waste-water Treatment	Receiving Facility: Recycle Inc.; POTW (Ind.)	Surface water	250	1.108	27.18	0.01	0.03	**9.06**	0.00
				20	13.85	339.11	0.11	0.43	**113.04**	0.01
				250	0.003	6.52	0.00	0.01	**2.17**	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Oiltanking Houston Inc, Houston, TX NPDES: TX0091855	Surface Water	Surrogate NPDES TX0065943	Surface water	20	0.041	89.13	0.03	0.11	**29.71**	0.00
St. Gabriel Terminal, Saint Gabriel, LA NPDES: LA0005487	Surface Water	NPDES LA0005487	Surface water	250	0.0055	0.0000223	0.00	0.00	0.00	0.00
				20	0.069	0.000279	0.00	0.00	0.00	0.00
Vopak Terminal Westwego Inc, Westwego, LA NPDES: LA0124583	Surface Water	Surrogate NPDES LA0042064	Surface water	250	0.00468	0.0000189	0.00	0.00	0.00	0.00
				20	0.058	0.000235	0.00	0.00	0.00	0.00
Research Solutions Group Inc, Pelham, AL NPDES: AL0074276	Surface Water	Not assessed (below the min risk level).								
Carlisle Engineered Products Inc, Middlefield, OH NPDES: OH0052370	Surface Water	Not assessed (below the min risk level).								
OES: Spot Cleaning and Carpet Cleaning										
Boise State University, Boise, ID NPDES: IDG911006	Surface Water	Surrogate NPDES ID0023981	Surface water	300	0.00008	0.00388	0.00	0.00	0.00	0.00
				20	0.001	0.0485	0.00	0.00	0.02	0.00

Name, Location, and ID of Active Releaser Facility [a]	Release Media [b]	Modeled Facility or Industry Sector in EFAST [c]	EFAST Waterbody Type [d]	Days of Release [e]	Release (kg/day) [f]	7Q10 SWC (ppb) [g]	Acute RQs (using COC of 3,200 ppb)	Chronic RQs (using fish COC of 788 ppb)	Algae RQs (using COC of 3 ppb)	Algae RQs (using COC of 52,000 ppb)
Venetian Hotel And Casino, Las Vegas, NV NPDES: NV0022888	Surface Water	Not assessed (below the min risk level).								
63,746 unknown sites NPDES: All POTW SIC	Surface Water or POTW	Not assessed (below the min risk level).								

a. Facilities actively releasing trichloroethylene 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 81% is applied to all indirect releases, as well as direct releases from WWTPs.

c. If a valid NPDES of the direct or indirect releaser was not available in EFAST, the release was modeled using either a surrogate representative facility in EFAST (based on location) or a representative generic industry sector. The name of the indirect releaser is provided, as reported in TRI.

d. EFAST uses ether the "surface water" model, for rivers and streams, or the "still water" model, for lakes, bays, and oceans.

e. Modeling was conducted with the maximum days of release per year expected. For direct releasing facilities, a minimum of 20 days was also modeled.

f. The daily release amount was calculated from the reported annual release amount divided by the number of release days per year.

g. For releases discharging to lakes, bays, estuaries, and oceans, the acute scenario mixing zone water concentration was reported in place of the 7Q10 SWC.

h. To determine the PDM days of exceedance for still bodies of water, the release days provided by the EPA Engineers should become the days of exceedance only if the predicted surface water concentration exceeds the COC. Otherwise, the days of exceedance can be assumed to be zero.

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Appendix F BENCHMARK DOSE ANALYSIS FOR (Selgrade and Gilmour, 2010)

F.1 BMDS Wizard Output Report - Mortality

The benchmark dose (BMD) modeling of dichotomous data was conducted with the EPA's BMD software (BMDS (version 2.7) via BMDS Wizard (version 1.11). All reasonably available dichotomous models (Gamma, Logistic, Dichotomous-Hill, Logistic, Log-Logistic, Probit, Log-Probit, Weibull, Multistage, and Quantal Linear) were fit to the incidence data for mortality due to introduced infection in mice following inhalation exposure to TCE. BMRs of 1%, 5%, and 10% extra risk were used in the BMD modeling, per technical direction. Adequacy of model fit was judged based on the χ^2 goodness-of-fit *p*-value ($p > 0.1$), magnitude of scaled residuals, and visual inspection of the model fit.

All models except for the Probit and Logistic provided adequate overall fit to the data, based on the $\chi 2$ goodness-of-fit p-value ($p > 0.1$). Among the remaining models, the Quantal Linear, Multistage, Weibull, Gamma and Log-Logistic models all showed poor fit at the 25 ppm data point, based on scaled residuals ranging from $> |1.5|$ to $> |2|$. This was the data point closest to the BMD for the Quantal Linear at BMR = 10% and for the rest of these models at BMR = 5%. Regardless of whether the models with poor fit at 25 ppm are included or not, the BMDLs at BMR = 10% or 5% are sufficiently close (within 3-fold), so that the model with the lowest AIC was selected; this is the Log-Probit. At BMR = 1%, however, the BMDLs are no longer within 3-fold; the results at this BMR show model-dependence. This reflects the lack of information reasonably available for the models to use in the data for the low-dose region of the dose-response curve (responses were similar in the control, 5, 10 and 25 ppm groups) and signifies increased uncertainty in selecting an appropriate BMDL at this BMR. Excluding the models with high scaled residuals at 25 ppm as less reliable leaves the Log-Probit and Dichotomous-Hill models. BMDLs for these models are sufficiently close, so the model with the lower AIC, the Log-Probit, was selected.

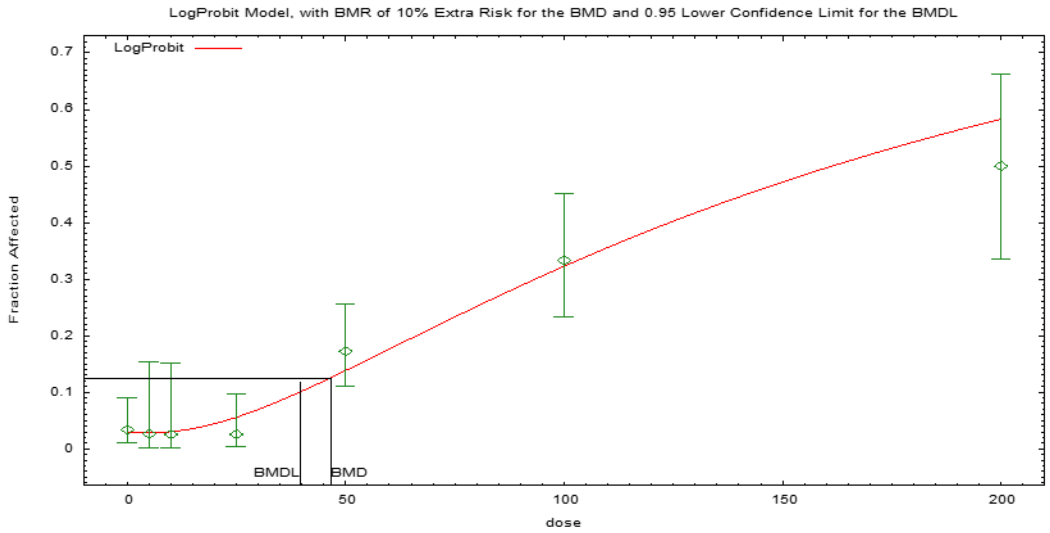
F.1.1 BMDS Summary of Mortality – BMR 10%

Table_Apx F-1. Summary of BMD Modeling Results for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 10% Extra Risk

Model[a]	Goodness of fit		BMD_{10Pct} (ppm)	$BMDL_{10Pct}$ (ppm)	Basis for model selection
	p-value	AIC			
Gamma	0.292	342.35	43.5	31.2	All models provided adequate overall fit to the data except for the Probit and Logistic models (based on the $\chi 2$ goodness-of-fit p-value). Although the Quantal Linear model provided adequate overall fit, the scaled residual nearest the BMD was $> |2|$, indicating poor fit in that part of the curve. With or without the Quantal Linear, the BMDLs are sufficiently close (< 3 fold), so the model with the lowest AIC was selected (Log-Probit).
Dichotomous-Hill	0.563	340.91	44.7	36.2	
Logistic	0.0074	351.35	66.2	57.6	
LogLogistic	0.370	341.62	43.3	31.6	
Probit	0.0211	348.55	61.1	53.3	
LogProbit	**0.582**	**338.72**	**46.6**	**39.6**	
Weibull	0.259	342.81	42.5	30.3	
Multistage 2°[b]	0.177	344.14	39.9	27.9	
Multistage 3°[c] Multistage 4°[d]	0.177	344.14	39.9	27.9	

Multistage 5°[e] Multistage 6°[f]					
Quantal-Linear	0.230	343.25	33.0	26.6	

[a] Selected model in bold; scaled residuals for selected model for doses 0, 5, 10, 25, 50, 100, and 200 ppm were 0.38, -0.08, -0.18, -1.16, 1.08, 0.22, -1.02, respectively.
[b] The Multistage 2° model may appear equivalent to the Multistage 3° model, however differences exist in digits not displayed in the table. This also applies to the Multistage 4° model. This also applies to the Multistage 5° model. This also applies to the Multistage 6° model.
[c] The Multistage 3° model may appear equivalent to the Multistage 2° model, however differences exist in digits not displayed in the table.
[d] For the Multistage 4° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 3° model.
[e] For the Multistage 5° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 4° model.
[f] For the Multistage 6° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 5° model.



Figure_Apx F-1. Plot of Incidence by Dose (ppm) with Fitted Curve for Log-Probit Model for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 10% Extra Risk

Probit Model. (Version: 3.4; Date: 5/21/2017)
The form of the probability function is: P[response] = Background + (1-Background) * CumNorm(Intercept+Slope*Log(Dose)),where CumNorm(.) is the cumulative normal distribution function
Slope parameter is restricted as slope >= 1

Benchmark Dose Computation.
BMR = 10% Extra risk
BMD = 46.6299
BMDL at the 95% confidence level = 39.5537

Parameter Estimates

Variable	Estimate	Default Initial Parameter Values
background	0.0281182	0.0338983
intercept	-5.1238E+00	-5.2930E+00
slope	1	1

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	p-value
Full model	-165.36	7			
Fitted model	-167.36	2	4.00401	5	0.55
Reduced model	-208.64	1	86.5627	6	<.0001

AIC: = 338.719

Goodness of Fit Table

Dose	Est. Prob.	Expected	Observed	Size	Scaled Resid
0	0.0281	3.318	4	118	0.38
5	0.0283	1.077	1	38	-0.08
10	0.0304	1.187	1	39	-0.18
25	0.0557	4.346	2	78	-1.16
50	0.1377	15.979	20	116	1.08
100	0.3216	25.088	26	78	0.22
200	0.5814	22.093	19	38	-1.02

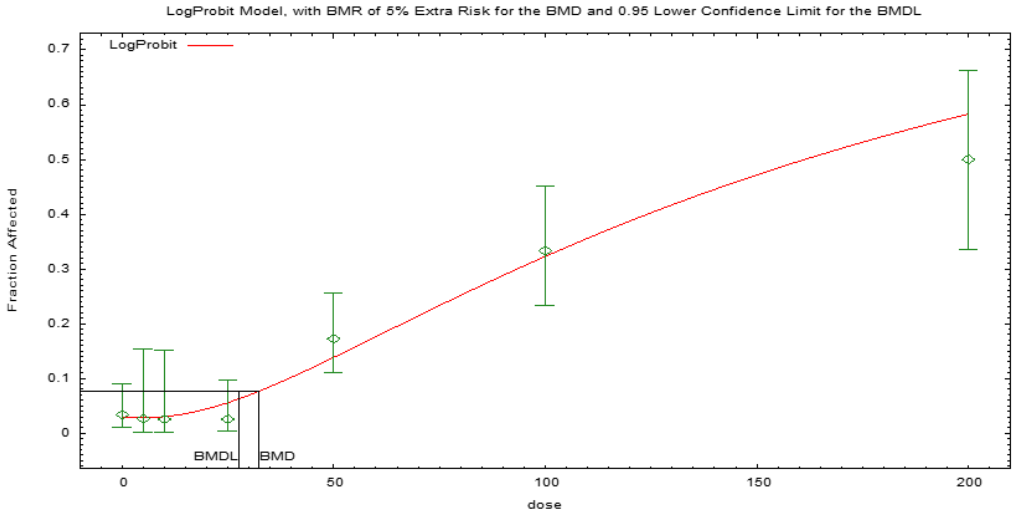
Chi^2 = 3.78 d.f = 5 P-value = 0.5818

F.1.2 BMDS Summary of Mortality – BMR: 5%

Table_Apx F-2. Summary of BMD Modeling Results for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 5% Extra Risk

Model[a]	Goodness of fit		BMD_{5Pct} (ppm)	$BMDL_{5Pct}$ (ppm)	Basis for model selection
	p-value	AIC			
Gamma	0.292	342.35	26.2	15.7	All models provided adequate overall fit to the data except for the Probit and Logistic models (based on the $\chi 2$ goodness-of-fit p-value). However, The Quantal Linear, Multistage, Weibull, Gamma and Log-Logistic models all showed poor fit at the 25 ppm data point, based on scaled residuals ranging from > |1.5| to > |2|. This was the data point closest to the BMD for all of these models except the Quantal Linear. With or without these models, the BMDLs are sufficiently close (< 3 fold), so the model with the lowest AIC was selected (Log-Probit).
Dichotomous-Hill	0.563	340.91	33.9	22.5	
Logistic	0.0074	351.35	40.3	34.4	
LogLogistic	0.370	341.62	26.8	17.0	
Probit	0.0211	348.55	36.6	31.4	
LogProbit	**0.582**	**338.72**	**32.4**	**27.5**	
Weibull	0.259	342.81	24.5	14.9	
Multistage 2° Multistage 3°[b] Multistage 4°[c] Multistage 5°[d] Multistage 6°[e]	0.177	344.14	20.6	13.6	
Quantal-Linear	0.230	343.25	16.0	12.9	

[a] Selected model in bold; scaled residuals for selected model for doses 0, 5, 10, 25, 50, 100, and 200 ppm were 0.38, -0.08, -0.18, -1.16, 1.08, 0.22, -1.02, respectively.
[b] For the Multistage 3° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 2° model.
[c] For the Multistage 4° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 3° model.
[d] For the Multistage 5° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 4° model.
[e] For the Multistage 6° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 5° model.



Figure_Apx F-2. Plot of Incidence by Dose (ppm) with Fitted Curve for Log-Probit Model for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 5% Extra Risk

Probit Model. (Version: 3.4; Date: 5/21/2017)

The form of the probability function is: P[response] = Background + (1-Background) * CumNorm(Intercept+Slope*Log(Dose)),where CumNorm(.) is the cumulative normal distribution function

Slope parameter is restricted as slope >= 1

Benchmark Dose Computation.

BMR = 5% Extra risk

BMD = 32.4253

BMDL at the 95% confidence level = 27.5047

Parameter Estimates

Variable	Estimate	Default Initial Parameter Values
background	0.0281182	0.0338983
intercept	-5.1238E+00	-5.2930E+00
slope	1	1

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	p-value
Full model	-165.36	7			
Fitted model	-167.36	2	4.00401	5	0.55
Reduced model	-208.64	1	86.5627	6	<.0001

AIC: = 338.719

Goodness of Fit Table

Dose	Est. Prob.	Expected	Observed	Size	Scaled Resid
0	0.0281	3.318	4	118	0.38
5	0.0283	1.077	1	38	-0.08
10	0.0304	1.187	1	39	-0.18
25	0.0557	4.346	2	78	-1.16
50	0.1377	15.979	20	116	1.08
100	0.3216	25.088	26	78	0.22
200	0.5814	22.093	19	38	-1.02

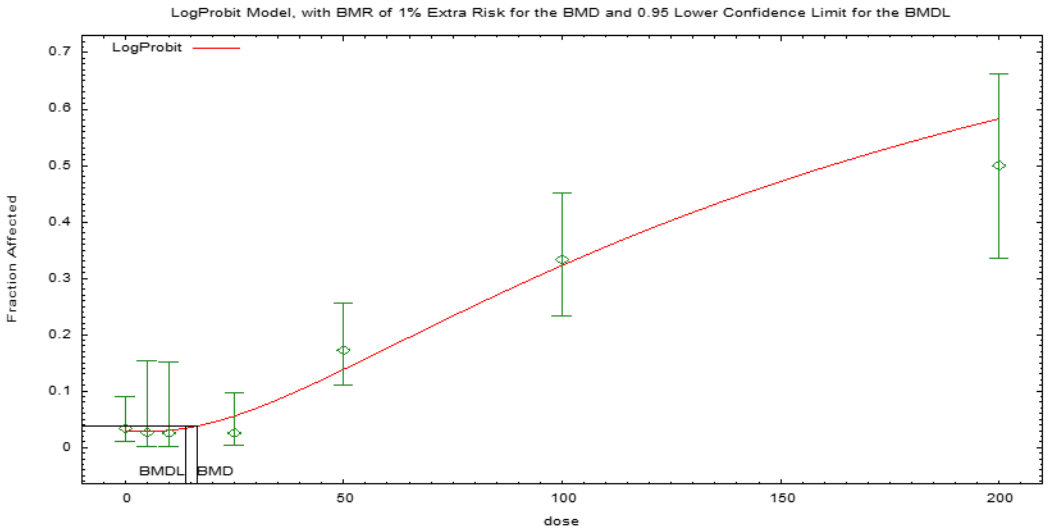
Chi^2 = 3.78 d.f = 5 P-value = 0.5818

F.1.3 BMDS Summary of Mortality – BMR: 1%

Table_Apx F-3. Summary of BMD Modeling Results for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 1% Extra Risk

Model[a]	Goodness of fit		BMD_{1Pct} (ppm)	$BMDL_{1Pct}$ (ppm)	Basis for model selection
	p-value	AIC			
Gamma	0.292	342.35	8.52	3.22	All models provided adequate overall fit to the data except for the Probit and Logistic models (based on the $\chi 2$ goodness-of-fit p-value). However, The Quantal Linear, Multistage, Weibull, Gamma and Log-Logistic models all showed poor fit at the 25 ppm data point, based on scaled residuals ranging from > |1.5| to > |2|. If all models are included, the BMDLs are not sufficiently close (> 3-fold). For this reason, the BMDS Wizard recommended selection of the Quantal Linear model, which had the lowest BMDL. The > 3-fold range of BMDLs is indicative of model dependence and signifies increased uncertainty in selecting an appropriate BMDL at this BMR. Excluding the models with high scaled residuals at 25 ppm as less reliable leaves the Log-Probit and Dichotomous-Hill models. BMDLs for these models are sufficiently close, so the model with the lower AIC, the Log-Probit, was selected.
Dichotomous-Hill	0.563	340.91	19.1	7.62	
Logistic	0.0074	351.35	10.2	8.35	
LogLogistic	0.370	341.62	9.29	4.17	
Probit	0.0211	348.55	9.14	7.52	
LogProbit	**0.582**	**338.72**	**16.4**	**13.9**	
Weibull	0.259	342.81	7.05	2.93	
Multistage 2°[b]	0.177	344.14	4.27	2.66	
Multistage 3°[c] Multistage 4°[d] Multistage 5°[e] Multistage 6°[f]	0.177	344.14	4.27	2.66	
Quantal-Linear	0.230	343.25	3.14	2.53	

[a] Selected model in bold; scaled residuals for selected model for doses 0, 5, 10, 25, 50, 100, and 200 ppm were 0.38, -0.08, -0.18, -1.16, 1.08, 0.22, -1.02, respectively.
[b] The Multistage 2° model may appear equivalent to the Multistage 3° model, however differences exist in digits not displayed in the table. This also applies to the Multistage 4° model. This also applies to the Multistage 5° model. This also applies to the Multistage 6° model.
[c] The Multistage 3° model may appear equivalent to the Multistage 2° model, however differences exist in digits not displayed in the table.
[d] For the Multistage 4° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 3° model.
[e] For the Multistage 5° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 4° model.
[f] For the Multistage 6° model, the beta coefficient estimates were 0 (boundary of parameters space). The models in this row reduced to the Multistage 5° model.



Figure_Apx F-3. Plot of Incidence by Dose (ppm) with Fitted Curve for Log-Probit Model for Mortality from Introduced Infection in Mice Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 1% Extra Risk

Probit Model. (Version: 3.4; Date: 5/21/2017)
The form of the probability function is: P[response] = Background + (1-Background) * CumNorm(Intercept+Slope*Log(Dose)),where CumNorm(.) is the cumulative normal distribution function
Slope parameter is restricted as slope >= 1

Benchmark Dose Computation.
BMR = 1% Extra risk
BMD = 16.4027
BMDL at the 95% confidence level = 13.9135

Parameter Estimates

Variable	Estimate	Default Initial Parameter Values
background	0.0281182	0.0338983
intercept	-5.1238E+00	-5.2930E+00
slope	1	1

Analysis of Deviance Table

Model	Log(likelihood)	# Param's	Deviance	Test d.f.	p-value
Full model	-165.36	7			
Fitted model	-167.36	2	4.00401	5	0.55
Reduced model	-208.64	1	86.5627	6	<.0001

AIC: = 338.719

Goodness of Fit Table

Dose	Est. Prob.	Expected	Observed	Size	Scaled Resid
0	0.0281	3.318	4	118	0.38
5	0.0283	1.077	1	38	-0.08
10	0.0304	1.187	1	39	-0.18
25	0.0557	4.346	2	78	-1.16
50	0.1377	15.979	20	116	1.08
100	0.3216	25.088	26	78	0.22
200	0.5814	22.093	19	38	-1.02

Chi^2 = 3.78 d.f = 5 P-value = 0.5818

F.2 BMDS Wizard Output Report - Number of Mice Infected

The benchmark dose (BMD) modeling of dichotomous data was conducted with the EPA's BMD software (BMDS (version 2.7) via BMDS Wizard (version 1.11). All reasonably available dichotomous models (Gamma, Logistic, Dichotomous-Hill, Logistic, Log-Logistic, Probit, Log-Probit, Weibull, Multistage, and Quantal Linear) were fit to the incidence data for mortality due to introduced infection in mice following inhalation exposure to TCE. BMRs of 1%, 5%, and 10% extra risk were used in the BMD modeling, per technical direction. Adequacy of model fit was judged based on the χ^2 goodness-of-fit *p*-value ($p > 0.1$), magnitude of scaled residuals, and visual inspection of the model fit.

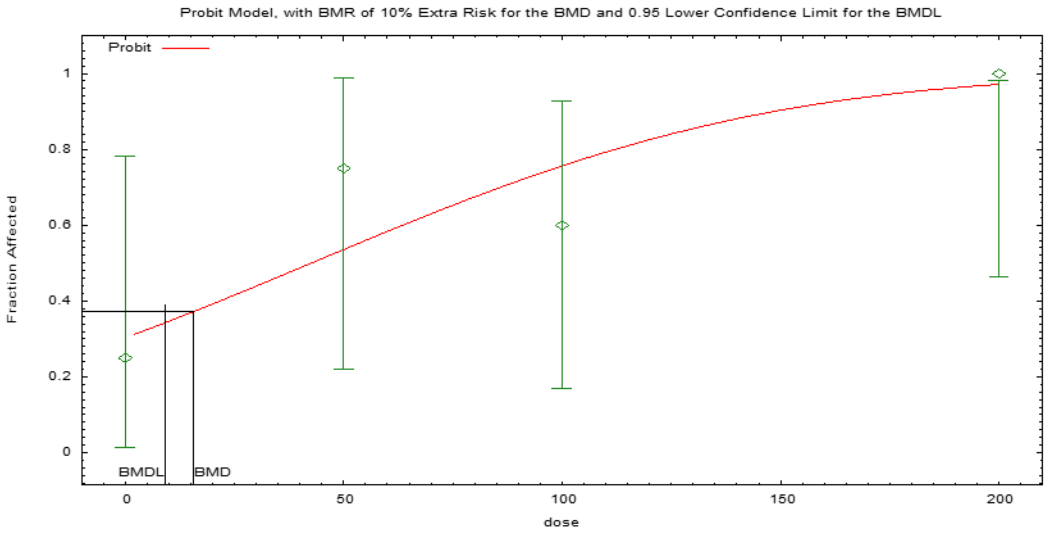
All models except for the Probit and Logistic provided adequate overall fit to the data, based on the $\chi 2$ goodness-of-fit p-value ($p > 0.1$). Among the remaining models, the Quantal Linear, Multistage, Weibull, Gamma and Log-Logistic models all showed poor fit at the 25 ppm data point, based on scaled residuals ranging from $> |1.5|$ to $> |2|$. This was the data point closest to the BMD for the Quantal Linear at BMR = 10% and for the rest of these models at BMR = 5%. Regardless of whether the models with poor fit at 25 ppm are included or not, the BMDLs at BMR = 10% or 5% are sufficiently close (within 3-fold), so that the model with the lowest AIC was selected; this is the Log-Probit. At BMR = 1%, however, the BMDLs are no longer within 3-fold; the results at this BMR show model-dependence. This reflects the lack of information reasonably available for the models to use in the data for the low-dose region of the dose-response curve (responses were similar in the control, 5, 10 and 25 ppm groups) and signifies increased uncertainty in selecting an appropriate BMDL at this BMR. Excluding the models with high scaled residuals at 25 ppm as less reliable leaves the Log-Probit and Dichotomous-Hill models. BMDLs for these models are sufficiently close, so the model with the lower AIC, the Log-Probit, was selected.

F.2.1 BMDS Summary of Infected at 72 hours – BMR – 10%

Table_Apx F-4. Summary of BMD Modeling Results for Number of Mice Infected at 72 Hours after Infection Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 10% Extra Risk

Model[a]	Goodness of fit		BMD_{10Pct} (ppm)	$BMDL_{10Pct}$ (ppm)	Basis for model selection
	p-value	AIC			
Gamma	0.190	23.637	~~9.77~~	~~4.24~~	All models provided adequate fit to the data (based on the $\chi 2$ goodness-of-fit p-value), although a BMDL could not be calculated for the Dichotomous-Hill model. The BMDS Wizard recommended the Probit model because it had the lowest AIC. BMDs and BMDLs from all models are well below the lowest data point and cannot be considered reliable.
Dichotomous-Hill	0.164	23.965	~~12.7~~	~~error~~[b]	
Logistic	0.428	21.584	~~15.6~~	~~8.36~~	
LogLogistic	0.164	23.965	~~12.7~~	~~1.13~~	
Probit	**0.448**	**21.445**	~~15.7~~	~~9.11~~	
LogProbit	0.383	21.877	~~15.6~~	~~6.86~~	
Weibull	0.189	23.606	~~14.3~~	~~4.25~~	
Multistage 2°	0.202	23.480	~~13.6~~	~~4.32~~	
Multistage 3°	0.228	23.267	~~13.8~~	~~4.43~~	
Quantal-Linear	0.425	21.639	~~8.56~~	~~4.24~~	

[a] Selected model in bold; scaled residuals for selected model for doses 0, 50, 100, and 200 ppm were -0.23, 0.86, -0.82, 0.38, respectively.
[b] BMD or BMDL computation failed for this model.



Table_Apx F-5. Plot of Incidence by Dose (ppm) with Fitted Curve for Probit Model for Number of Mice Infected at 72 Hours after Infection Following Inhalation Exposure to TCE (Selgrade and Gilmour 2010); BMR = 10% Extra Risk

Appendix G WEIGHT OF EVIDENCE FOR CONGENITAL HEART DEFECTS

G.1 EPA Review of the Charles River (2019) Study

G.1.1 Study Methodology and Results

In a study sponsored by the Halogenated Solvents Industry Alliance (HSIA), Charles River Laboratories Ashland, LLC performed "An Oral (Drinking Water) Study of the Effects of Trichloroethylene (TCE) on Fetal Heart Development in Sprague Dawley Rats". The study was based on general accordance with OPPTS 870.3700 and OECD Test Guideline 414 with the stated purpose of replicating the findings of (Dawson et al., 1993) and (Johnson et al., 2003), which observed increased cardiac malformations in the fetuses of pregnant female Sprague Dawley rats administered TCE in drinking water.

The study utilized 6 test groups, including negative and positive controls. Retinoic acid (RA) served as a positive control and was administered daily via gavage. TCE was administered via drinking water. See details in Table_Apx G-1, which is adapted from Text Table 4 in the study.

Table_Apx G-1. Experimental Design

Group	Treatment	Target Concentration	Route of Administration	Number of Females (Dams)
1	Vehicle (water)	0 ppm	Drinking Water	25
2	Retinoic Acid	3 mg/ml	Gavage	25
3	TCE	0.25 ppm	Drinking Water	25
4	TCE	1.5 ppm	Drinking Water	25
5	TCE	500 ppm	Drinking Water	25
6	TCE	1000 ppm	Drinking Water	25

In order to reduce TCE loss due to evaporation, drinking water formulations were prepared at volumes large enough to minimize headspace and a connected nitrogen source was used to backfill headspace during dosing. Despite this effort, 24-hour loss monitoring indicated that 30% to 49% of average measured TCE concentration was lost over the course of a day.

Interventricular septal defects (VSDs) were the only cardiac malformation observed in TCE-treated groups. Additional types of defects were observed in the positive control RA-treated group, including malformations of the aorta and arteries, small ventricle, and situs inversus (transposition of the heart and great/major vessels). Situs inversus was also observed in a single vehicle control fetus. The study authors did not observe a statistically significant increase in VSDs among TCE-treated fetuses compared to vehicle. Additionally, all VSDs observed in TCE-exposed fetuses were smaller than 1mm, in contrast with vehicle and RA-treated groups. Results are shown in Table_Apx G-2 below, which is adapted from Text Table 14 in the study, with a few small edits. The Charles River study described the statistical estimate used as "summation per group (%)", which appears to be the sum of viable fetuses affected per litter (%) / number of litters per group". EPA determined that while this method is appropriate, the description is unclear and would be better described as "Mean % Affected / Litter per Group". EPA therefore replaced the descriptor "% per litter" with the above descriptor. EPA also identified that the

RA-treated group actually had 41.2% affected, as opposed to 42.2% as was presented in Text Table 14 of the study.

Table_Apx G-2. Summary of Observed Interventricular Defects

Dosage:	0 ppm (Vehicle)	15 mg/kg-day RA	0.25 ppm TCE	1.5 ppm TCE	500 ppm TCE	1000 ppm TCE
# Affected Fetuses (Litters)	7 (5)	112 (23)	4 (4)	5 (3)	13 (8)	12 (6)
Mean % Affected / Litter per Group	2.4%	41.2% ($p < 0.01$)	1.4%	1.5%	3.8%	3.7%
Size of Opening (Number of Fetuses)	<1mm (6) 1mm (1)	<1mm (103) 1mm (8) >2mm (1)	<1mm (All)	<1mm (All)	<1mm (All)	<1mm (All)
Defect Location	Membranous	Membranous (111); Muscular (1)	Membranous	Membranous	Membranous	Membranous

VSDs were not statistically significantly increased in TCE-treated groups compared to vehicle control, while RA treatment resulted in a substantially increased incidence of cardiac defects. The authors additionally highlighted the fact that all identified VSDs in TCE-treated groups were smaller than 1mm. The study states that these would be expected to resolve postnatally and are therefore unlikely to be adverse.

G.1.2 EPA Review

G.1.2.1 Comparing Results Between Charles River and Johnson Studies

The Charles River study calculated observed defects differently than was done for the Dawson and Johnson studies. The calculation for mean % affected / litter per group results in different values than the "% fetuses affected" and "% litters affected" metrics used in the Dawson and Johnson studies, which simply divided the amount of affected fetuses or litters by the total (multiplied by 100 to create a percentage). For comparison, Table_Apx G-3 below presents the data from both the Johnson and Charles River studies calculated as the % fetuses and % litters affected.

Table_Apx G-3. Incidence of total heart malformations in Johnson and Charles River studies.

	Johnson 2003			Charles River 2019		
Dose	**% fetuses affected**	**% litters affected**	**Source**	**% fetuses affected**	**% litters affected**	**Source/Notes**
0 ppm	13/606 (2.2%)	9/55 (16.4%)	(Johnson et al., 2003)	8/308 (2.5%)	6/24 (25.0%)	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
2.5 ppb	0/44 (0.0%)	0/12 (0.0%)	(Johnson et al., 2003)	N/A	N/A	N/A
0.25 ppm	5/110 (4.5%)	4/9 (44.4)	(Johnson et al., 2003)	4/275 (1.4%)	4/22 (18.2%)	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86

1.5 ppm	9/181 (5.0%)	5/13 (38.5%)	(Johnson et al., 2003)	5/321 (1.5%)	3/24 (12.5%)	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
500 ppm	N/A	N/A	N/A	13/330 (3.9%)	8/24 (33.3%)	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
1000 (Charles River) or 1100 (Johnson) ppm	11/105 (10.5%)	6/9 (66.7%)	(Johnson et al., 2003)	12/342 (3.5%)	6/24 (25.0%)	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86

The Johnson study clearly shows greater incidences of cardiac defects at 0.25 ppm, 1.5 ppm, and 1100 ppm compared to the same or similar doses (1000 ppm in Charles River). Of note however, VSDs, and specifically only membranous VSDs, were the only type of heart malformation identified by the Charles River study in TCE-treated fetuses. In contrast, the Johnson study identified a broad variety of defects in exposed fetuses. The Johnson study observed VSDs at only a slightly greater incidence per fetus than by Charles River at higher doses, while (peri)membranous VSDs were observed at a similar or lower incidence than by Charles River. Additionally, Charles River observed substantially higher incidences of VSDs in the control and 0.25 ppm groups. The data comparing the incidence of VSDs only is presented in Table_Apx G-4, with the incidence of membranous VSDs displayed in parentheses.

Table_Apx G-4. Incidence of VSDs in Johnson and Charles River studies.

	Johnson 2003		Charles River 2019	
Dose	**% fetuses affected (mem. only)**	**Source**	**% fetuses affected**	**Source/Notes**
0 ppm	0.66% (0.33%)	(Johnson et al., 2003), Table 2	2.5%	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
2.5 ppb	0%	(Johnson et al., 2003), Table 2	N/A	N/A
0.25 ppm	0%	(Johnson et al., 2003), Table 2	1.4%	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
1.5 ppm	2.21% (1.66%)	(Johnson et al., 2003), Table 2	1.5%	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
500 ppm	N/A	N/A	3.9%	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86
1000 (Charles River) or 1100 (Johnson) ppm	3.81% (2.86%)	(Johnson et al., 2003), Table 2	3.5%	(Charles River Laboratories, 2019), Table 15 (soft tissue), p. 86

G.1.2.2 Differences in Types of Malformations Observed

The majority of cardiac malformations observed in the Johnson study were not VSDs (see Table 2 in (Johnson et al., 2003), while the Charles River study only identified VSDs in controls and TCE-treated offspring. Of note, two major categories of heart malformations identified in the Johnson study that are absent from even the positive control group of the Charles River study are atrial septal defects and valve defects. The Charles River study methodology appeared to be focused primarily on identification of VSDs over other heart defects, which may explain the observed positive bias toward detection of VSDs in vehicle control and low-dose fetuses as compared to both the Johnson study and historical control data. Table_Apx G-5 compares the heart defects observed across all *in vivo* oral studies. Fisher at al. (2001), a gavage study that also did not find a statistically significant association of TCE exposure with congenital cardiac defects, is also included for comparison. Of note, the (Fisher et al., 2001) study utilized the same dissection and evaluation methodology as the (Johnson et al., 2003) studies. There is substantial overlap in the many type of defects identified in the three studies, while only membranous VSDs were observed in TCE-treated animals in (Charles River Laboratories, 2019) (great blood vessel variation was identified in a few TCE-treated pups but was considered incidental by the study authors). When comparing the results from (Fisher et al., 2001) and (Charles River Laboratories, 2019), EPA acknowledges that differences in dosing method, vehicle volume, and other variables may also contribute to any observed differences.

Table_Apx G-5. Heart and Cardiovascular Defects Observed in Oral TCE studies

Cardiac Malformations Observed Across Select Oral TCE and Retinoic Acid (RA) Developmental Toxicity Studies in Rats				
Trichloroethylene (TCE)			**Retinoic Acid (RA)**	
Johnson et al. (2003) [a]	**Charles River (2019)**	**Fisher et al. (2001)**	**Charles River (2019)**	**Fisher et al. (2001)**
Septal defects				
Ventricular septal defect (VSD) (perimembranous, subaortic, muscular)	**Ventricular septal defect (VSD) (membranous)**		**Ventricular septal defect (VSD) (membranous, subaortic, muscular)**	**Ventricular septal defect (VSD) (membranous, aortic, muscular)**
Atrial septal defect (ASD)		**Atrial septal defect (ASD)**		**Atrial septal defect (ASD)**
Valve defects				
Mitral valve defect		**Mitral valve defect**		**Mitral valve defect**
Tricuspid valve defect		**Tricuspid valve defect**		**Tricuspid valve defect**
Pulmonary valve defect				**Pulmonary valve defect**
Aortic valve defects (multiple)			**Aortic stenosis**	**Aortic stenosis**
Atrium, ventricle, and miscellaneous structural abnormalities				
Atrioventricular septal defect (**endocardial cushion defects**)		**Endocardial cushion defects**		
		Right ventricle enlarged		**Right ventricle enlarged**
		Left ventricle aneurysm dissecting	Heart ventricle, small	Left atrial hypertrophy
				Cleft, apex of heart

Cardiac Malformations Observed Across Select Oral TCE and Retinoic Acid (RA) Developmental Toxicity Studies in Rats				
Great vessel structural abnormalities				
			Transposition of the great vessels	**Transposition of the great vessels**
			Aortic arch effects	**Aortic arch effects**
			Major blood vessel variation	**Major blood vessel variation**
Pulmonary artery hypoplasia				**Pulmonary artery hypoplasia**
Aortic hypoplasia				
		Innominate artery short		**Innominate artery effect**
Coronary artery/sinus			Stenotic carotid	Truncus dilated
Positional abnormalities of the heart and great vessels				
		Situs inversus	**Situs inversus**	Dextrocardia
Abnormal looping				Overriding aorta

[a] Includes data from Dawson et al. (1993).
Bold text indicates defects observed across multiple studies (both TCE and RA treatment).
Red bold text indicates defects only observed with RA treatment across multiple studies.

EPA's conclusion that the Charles River study insufficiently sensitive to non-VSD defects was supported by the limited variety of malformations observed in the RA positive control based on a compiled literature search:

1. EPA searched HERO and PubMed for studies investigating heart defects and malformations that occur during prenatal exposure to all-trans retinoic acid (RA). Of the 37 studies reviewed, 12 studies were excluded from analysis because they were abstracts, book chapters, reviews, or studies that did not expose animals to all-trans RA. Thus, EPA reviewed 25 studies and compared the results of these studies to those reported by the Charles River and Johnson studies.
2. In all species examined, a total of 35 heart defects were associated with prenatal exposure to RA in the identified literature.
3. The Charles River study reported 10 types of heart defects in animals exposed to RA.
4. Heart defects associated with TCE exposure partially overlap defects associated with RA exposure. The Johnson study identified 10 types of cardiac defects in TCE-exposed fetuses. Charles River only identified one defect (membranous VSDs) associated with TCE exposure (major blood vessel variation was observed in 1-2 TCE-treated fetuses, but this effect was not considered treatment-related).
5. All 35 defects associated with RA exposure were observed in rodents in the literature review. If we limit the analysis to studies examining only rats, 31 of the total 35 defects were observed. Only 6 of the 35 defects were noted in chickens, and 2 of the 35 were noted in zebrafish. Therefore, the differences between defects captured in the Charles River study and the general literature cannot be explained simply by inclusion of additional experimental species in the general literature.

EPA therefore concludes that Charles River did not capture the entirety of cardiac defects that were expected upon exposure to RA.

EPA searched HERO using the following keywords:

- Retinoic Acid
- Retinoic Acid + Cardiac

EPA also searched PubMed using the following keywords:

- retinoic acid (RA)-induced cardiac defects
- retinoic acid AND (cardiac defects OR cardiac malformations OR heart defects OR heart malformations OR cardiac teratogenesis OR aorta OR ventricle OR endocardial cushion OR pulmonary valve OR mitral valve OR aortic valve OR ventricular septum OR atrial septum OR tricuspid valve OR aneurysm).

Table_Apx G-6 presents all of the cardiac defects found in the literature search.
Table_Apx G-7 compares the types of defects observed across the Johnson and Charles River studies with those identified in the literature search. Several defects associated with TCE exposure as well as several RA-induced defects in the Charles River study were not associated with RA exposure in the literature. Overall, the spectrum of heart defects observed upon RA exposure in the literature largely, but not entirely, overlaps with heart defects associated with TCE exposure. Of note, atrial septal defects, which were the most common type of malformation identified in the Johnson study, were identified in 5 other RA studies but not in the Charles River study.

Table_Apx G-6. Cardiac Defects Observed in Literature

Cardiac Defect	Number of Studies
VSD	12
ASD	5
Tetralogy_Fallot	1
Hypoplastic_Left_Heart_Syndrome	1
Tricuspid_Atresia	1
Aortic_Valve_Stenosis	1
Pulmonary_Trunk_Stenosis	3
Right_Ventricular_Hypertrophy	2
Left_Ventricular_Hypertrophy	1
Right_Atrial_Hypertrophy	2
Left_Atrial_Hypertrophy	1
CAVC	1
Situs_Inversus	2
Dextrocardia	5
d_Transposition	12
I_Transposition	1
Cleft_Apex	1
CoA	1
ARSA	2
IAA	1
Left_Circumflex_Aorta	1
Right aortic arch defect (RAA)	4
Double_Aortic_Arch	1
Cervical_Aortic_Arch	1
Hypoplastic_Aortic_Arch	1
Truncus_Arteriosus	7
PDA	1
Innominate_Artery_Absent	1

Innominate_Artery_Short	1
Right_Carotid_Off_Aorta	1
Right_Subclavian_Artery_Absent	1
DORV	10
Endocardial_Cushion_Defect	3
Abnormal_Heart_Looping	7
Other*	14

628
629

Table_Apx G-7. Cardiac Defects Observed After Exposure to RA or TCE

Chemical:		TCE	TCE	RA	RA	RA
Malformation Class	**Malformation Name**	**Charles River 2019**	**Johnson 2003**	**Charles River 2019**	**Other Literature (No. Studies)**	**Other Literature Species[1]**
Atrium, Ventricle and Valve Defects	VSDs[2]	√	√	√	√ (12)	C, H, M, R
Atrium, Ventricle and Valve Defects	Atrial Septal Defect		√		√ (5)	H, R
Atrium, Ventricle and Valve Defects	Double outlet ventricle (DORV)				√ (10)	C, H, M, R
Atrium, Ventricle and Valve Defects	Tetralogy of Fallot				√ (1)	M
Atrium, Ventricle and Valve Defects	Hypoplastic Left Heart Syndrome				√ (1)	R
Atrium, Ventricle and Valve Defects	Tricuspid defects		√		√ (1)	H
Atrium, Ventricle and Valve Defects	Aortic valve defects		√[3]		√ (1)	R
Atrium, Ventricle and Valve Defects	Mitral valve defects		√			
Atrium, Ventricle and Valve Defects	Right ventricular hypertrophy				√ (2)	R
Atrium, Ventricle and Valve Defects	Left ventriclular hypertrophy				√ (1)	R
Atrium, Ventricle and Valve Defects	Right atrial hypertrophy				√ (2)	R
Atrium, Ventricle and Valve Defects	Left atrial hypertrophy				√ (1)	R
Atrium, Ventricle and Valve Defects	Small ventricle			√		
Atrium, Ventricle and Valve Defects	Complete Atrioventricular Canal defect (CAVC)		√		√ (1)	R
Symmetry	Situs Inversus			√	√ (2)	C, R
Symmetry	Dextrocardia				√ (5)	M, R
Symmetry	d-Transposition of the great arteries			√	√ (12)	C, H, M, R
Symmetry	l-Transposition of the Great Arteries				√ (1)	R
Symmetry	Cleft, apex of heart				√ (1)	R
Aortic Arch Defects	Coarctation of the Aorta (CoA)			√	√ (1)	R
Aortic Arch Defects	Left aortic arch with aberrant right subclavian artery (ARSA)			√[4]	√ (2)	R
Aortic Arch Defects	left circumflex aorta				√ (1)	M

Chemical:		TCE	TCE	RA	RA	RA
Malformation Class	**Malformation Name**	**Charles River 2019**	**Johnson 2003**	**Charles River 2019**	**Other Literature (No. Studies)**	**Other Literature Species[1]**
Aortic Arch Defects	Right aortic arch defects (RAA)		√		√ (4)	H, M, R
Aortic Arch Defects	Double aortic arch				√ (1)	R
Aortic Arch Defects	Cervical aortic arch				√ (1)	R
Aortic Arch Defects	Interruption of the aortic arch			√	√ (1)	M
Aortic Arch Defects	Hypoplastic aortic arch				√ (1)	R
Aortic Arch Defects	Stenotic aortic arch			√		
Other vessel defects	Pulmonary trunk stenosis				√ (3)	H, R
Other vessel defects	Truncus Arteriosus (dilated truncus)				√ (7)	H, M, R
Other vessel defects: incomplete postnatal development	Patent Ductus Arteriosus				√ (1)	R
Other vessel defects	Innominate artery absent				√ (1)	R
Other vessel defects	Innominate artery short				√ (1)	R
Other vessel defects	Right carotid off aorta				√ (1)	R
Other vessel defects	Stenotic carotid			√		
Other vessel defects	Right subclavian artery absent				√ (1)	R
Other vessel defects	Pulmonary artery hypoplasia		√			
Other vessel defects	Coronary artery/sinus defects		√			
Other early developmental defect	Endocardial cushion defects				√ (3)	M, R
Other early developmental defect	Abnormal heart looping		√		√ (7)	C, H, R, Z
Other[5]				√[7]	√ (14)	C, H, M, R, Z

[1] Chicken (C), Hamster, (H), Mouse (M), Rat (R), Zebrafish (Z).
[2] Most studies reviewed did not specify among perimembranous, muscular or subarterial VSDs, so these were included all as "VSDs" for the literature review comparison.
[3] Aortic valve defects included aortic valve defect with fenestrated leaflets and aortic valve stenosis described as aortic valve defect with fused leaflets creating aortic valvular stenosis.
[4] Chicken (C), Hamster, (H), Mouse (M), Rat (R), Zebrafish (Z).
[4]Retroesophageal aortic arch described in Charles River study was tagged as ARSA defect.
[5] Major blood vessel variation (right carotid and subclavian arteries arose independently from the aortic arch [no brachiocephalic trunk] or right subclavian artery coursed retroesophageal and joined the aortic arch adjacent to ductus arteriosus [no brachiocephalic trunk]) tagged to RAA defects.
[5] If EPA was unsure of the general malformation class, the defect was categorized as "other".
[6] "Other" defect in HSIA study (RA exposure groups) was a major blood vessel variation (an elongated brachiocephalic trunk or a missing brachiocephalic trunk due to right carotid and right subclavian arising independently from the aortic arch, or due to a retroesophageal right subclavian; or (right carotid and subclavian arteries arose independently from the aortic arch [no brachiocephalic trunk] or right subclavian artery coursed retroesophageal and joined the aortic arch adjacent to ductus arteriosus [no brachiocephalic trunk]).

G.1.2.3 Methodology Differences

There are likely several contributing factors explaining why the Charles River study failed to identify atrial or valve defects. In the Johnson study, the materials and methods section described examination of the internal structure of the heart for all fetuses. The dissection methodology allows detailed examination of the atrial septum. In contrast, the Charles River study states that the fetal evaluation methods were conducted according to Stuckhardt and Poppe (1984), which does not include

examination of atrial septal defects. Therefore, the methodology used by the Charles River study was likely to miss this important category of cardiac malformations. As shown in Table_Apx G-7, five other studies were identified in the literature that observed atrial septal defects following RA exposure, while none were observed in the Charles River study.

The Stuckhardt and Poppe method (1984) does includes visualization of the valves (the tricuspid, mitral, aortic, and pulmonary valves) but the methods as described in the Johnson study and supporting information are more likely to reveal valvular defects as compared to the Stuckhardt and Poppe methodology. The Stuckhardt and Poppe method specifies that two cuts are made in the fresh fetal heart. This allows visualization of the tricuspid valve, between the right atrium and right ventricle, the three cusps of the semilunar valve of the pulmonary artery, and the interventricular septum. In comparison, the Johnson study clearly specified that the fetal hearts were to be examined in situ for external defects and then excised, preserved with glutaraldehyde, and dissected. The examination of the internal structure of the heart for all fetuses specifically included removing tissue to expose the pulmonary, aortic, tricuspid, and mitral valves. The location of the coronary ostium was noted, each valve was probed for patency, and the formation of each valve leaflet was examined.

EPA believes that there is a certain amount of tissue elasticity in fresh fetal hearts that can obscure the detection of valvular defects during fetal morphological evaluation. Because the Johnson study evaluated the internal structure of the fetal hearts post-fixation, examination of the valvular structures would have been facilitated. Additionally, valve defects may be overlooked during examination unless the technician is directly focusing on evaluating the cardiac valves in all fetuses (not just those, for example, in which external cardiac morphological differences, such as a collapsed ventricle, might suggest a potential valve problem). No indication is given in the Charles River report whether a directed effort was made to identify valvular abnormalities.

Other identified differences and uncertainties in the methodology between the two studies may or may not have contributed to the differences in results. These factors could potentially make either the Johnson or the Charles River data more precise. These include the following:

1. Variations in TCE loss over time. While the Charles River study made extensive efforts to minimize TCE loss, the 24-hour loss monitoring indicated that average loss across all measurements was actually greater than that in the Johnson study (42% vs 35%). The Johnson study did not provide analytical measurements for close comparison, but it is possible that on average the delivered dose was greater in the Johnson study.
2. Possible differences in criteria for fetuses selected for examination. In the Johnson study, it is not explicitly stated whether all or only viable fetuses were examined. The Charles River study indicates that only viable fetuses were examined. For the Charles River study, this is a moot point as there were no dead fetuses in the entire study. However, this aspect of study design is not documented in the Dawson or Johnson studies.
3. Randomization methods. Differences in incidences at the litter level could potentially result from non-randomized groups of animals at different dose levels. Different randomization strategies were used in Johnson 2003 compared to the HSIA study. Dam assignments to exposure groups was randomized in Johnson 2003, whereas the HSIA study used stratified randomization. Details of the stratified randomization strategy were not presented, except to indicate that the goal was to achieve similar group mean body weights. Given that there were six treatment groups and many racks have six cages per row, it raises the possibility that treatment group was confounded with cage position, i.e., Group 1 in one column, Group 2 in the next column, etc. The Dawson and

Johnson methods of randomization did not include consideration of, or stratification by, body weight.

4. Husbandry differences. the Charles River study individually housed the pregnant females, whereas the Dawson and Johnson studies group-housed the females, so several dams were consuming treated drinking water from the same bottle. Thus, there would be greater precision in the Charles River dose calculations.
5. Source and strain of rats. The rats used in all the studies conducted as part of the TCE research program at the University of Arizona that included (Dawson et al., 1993) and (Johnson et al., 2003) were Harlan Sprague-Dawley rats purchased from Harlan Laboratories Inc., Indianapolis, IN. The Charles River rats were Crl:CD(SD) Sprague-Dawley rats from Charles River Laboratories in Raleigh, NC. It is unknown what influence the source or strain differences might have had on the response to treatment with TCE. Additional information from both groups of researchers would be needed to ascertain whether the source, sub strain or genetic drift of the test animals influenced the incidences of cardiac malformations.
6. Technical confirmation of diagnosis. The Charles River report did not specify whether cardiac abnormalities were confirmed by other technical staff or the Study Director. There is no opportunity to re-examine fetuses because the report states that all carcasses were discarded following completion of the internal examination of the fetuses. In comparison, the three principle authors of the Dawson and Johnson studies (P. Johnson, S. Goldberg, and B. Dawson), each examined every identified fetal cardiac anomaly, and they only included findings for which there was unanimous agreement on diagnosis (as described in (Makris et al., 2016)). Therefore, there is high confidence in the determination of observed defects in the Dawson and Johnson studies. Of note, neither study was designed to confirm diagnoses of normal fetal morphology.

G.1.2.4 Adversity of Small VSDs

In addition to the lack of a statistically significant increase in cardiac defects, the Charles River study claims that the <1mm VSDs induced by TCE are non-adverse because "…similar to humans, small spontaneous interventricular septal defects in rats close postnatally and hence should not be considered adverse. Based on these data, the interventricular septal defects observed in the TCE-treated groups were considered to be spontaneous background occurrences and unrelated to TCE exposure." This claim is confounding and internally inconsistent however, because the vast majority (92%) of VSDs observed in the RA-treated positive control group were also <1mm. If VSDs <1mm are truly non-adverse, then this positive control data provides additional indication that the study is insufficiently sensitive for detecting adverse cardiac defects.

The Charles River study cites (Fleeman et al., 2004), which based on results of trimethadione exposure concluded: "…some treatment-induced membranous VSD will close during postnatal development similar to spontaneously occurring membranous VSD." The authors then state that "small, isolated VSD do not seem to impact postnatal viability and growth; however, large VSD are likely to affect postnatal survival." Importantly, the presence of a VSD was associated with reduced survival, so observing reduced incidence of VSDs postnatally may be selecting for those pups that were less adversely affected. Nonetheless, the data does demonstrate that some, but not all, VSDs are compatible with postnatal life. However, as there is no information provided in this paper to characterize the size range of VSD in those pups that died compared to the size of the VSD in those that survive, one cannot rule out the possibility that any VSD may be a potential adverse effect of chemical exposure.

A review of the literature on spontaneous closure of VSDs (Zhang, 2015) summarized that both defect size and location can influence the likelihood of postnatal closure. The author reports that studies have found defects <3-6mm are more likely to close but acknowledges the controversy over the significance

of defect size. More significantly, the study concluded that muscular VSDs are much more likely to close spontaneously than membranous VSDs (which were the only VSD type associated with TCE exposure in the Charles River study). The incidence in humans of spontaneous closure in cited studies examining only muscular VSDs ranges from 22% to 84%, while for studies examining only membranous or perimembranous VSDs the incidence ranges from only 4% to 47%. Additionally, the morphological characterization of closure of the membranous VSD seems to most commonly involve the use of a leaflet of the tricuspid valve, which would be expected to impact the functional ability of that heart valve. Therefore, even if a membranous VSD is able to spontaneously close, there are likely functional impacts of that closer, resulting in an adverse health effect.

Overall, it is impossible to speculate whether the specific VSDs identified in these studies would have closed during lactation. Congenital heart defects of any kind are considered to be an adverse medical event in humans, whether they eventually close naturally or need to be surgically repaired. When considering the uncertainty over the likelihood of VSD closure and the preponderance of additional types of defects observed in other studies, this consideration is not relevant to the significance of this endpoint.

G.2 WOE Analysis for Congenital Cardiac Defects

G.2.1 Methodology

1) EPA identified, collected and reviewed a sampling of recent literature on systematic approaches to performing weight-of-evidence evaluation. Relevant articles were identified by simple Google searches and by tree searching references listed in these publications. References included the following:
    a. Weed. 2005. Weight of Evidence: A Review of Concept and Methods. Risk Anal 25(6): 1545-1557 (Weed, 2005).
    b. Gough. 2007. Weight of Evidence: A Framework for the Appraisal of the Quality and Relevance of Evidence. Research Papers in Education 22(2): 213-228 (Gough, 2007).
    c. Rhomberg et al. 2013. A survey of frameworks for best practices in weight-of-evidence analyses. Crit Rev Toxicol 43(9): 753–784 (Rhomberg et al., 2013).
    d. Rooney et al. 2014. Systematic Review and Evidence Integration for Literature-Based Environmental Health Science Assessments. Env Health Perspect 122 (7): 711-718 (Rooney et al., 2014).
    e. NTP. 2015. Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration (NTP, 2015).
    f. EPA. 2016. Weight of Evidence in Ecological Assessment. Risk Assessment Forum. EPA/100/R16/001 (U.S. EPA, 2016i).
    g. EPA. 2015. EDSP: Weight of Evidence Analysis of Potential Interaction with the Estrogen, Androgen or Thyroid Pathways. Chemical: Glyphosate. Office of Pesticide Programs (U.S. EPA, 2015a).
    h. US Army Corps of Engineers. 2018. Weight-of-Evidence Concepts: Introduction and Application to Sediment Management (Engineers, 2018).
    i. European Commission. 2018. Memorandum on weight of evidence and uncertainties. Revision 2018. Scientific Committee on Health, Environmental and Emerging Risks (SCHEER) (EC, 2018).
    j. EFSA. 2017. Guidance on the use of the weight of evidence approach in scientific assessments. EFSA Journal 15(8): 4971 (1-69) (EFSA, 2017).

k. Linkov et al. 2015. From "Weight of Evidence" to Quantitative Data Integration using Multicriteria Decision Analysis and Bayesian Methods. Altex 32(1): 3-8 (Linkov et al., 2015).
l. Smith et al. 2002. Weight of Evidence (WOE): Quantitative Estimation of Probability of Impact. Manuscript (Smith et al., 2002).
m. Bridges et al. 2017. Framework for the quantitative weight-of-evidence analysis of 'omics data for regulatory purposes. Reg Tox Pharm 91: S46-S60 (Bridges et al., 2017).
n. Dekant and Bridges. 2016. Assessment of reproductive and developmental effects of DINP, DnHP and DCHP using quantitative weight of evidence. Reg Tox Pharm 81: 397-406 (Dekant and Bridges, 2016).
o. Bridges and Solomon. 2016. Quantitative weight-of-evidence analysis of the persistence, bioaccumulation, toxicity, and potential for long-range transport of the cyclic volatile methyl siloxanes. J Toxicol Environ Health Part B 19(8): 345-379 (Bridges and Solomon, 2016).
p. Gangwal et al. 2012. Incorporating exposure information into the toxicological prioritization index decision support framework. Sci Total Environ 435-436: 316-325 (Gangwal et al., 2012).
q. Reif et al. 2013. ToxPi GUI: an interactive visualization tool for transparent integration of data from diverse sources of evidence. Bioinformatics 29(3): 402-403 (Reif et al., 2013).
r. Klimisch et al. 1997. A Systematic Approach for Evaluating the Quality of Experimental Toxicological and Ecotoxicological Data. Reg Tox Pharm 25: 1-5 (Klimisch et al., 1997).

2) Upon review of the various weight-of-evidence approaches that have been proposed, EPA chose to adopt the method presented by EPA Risk Assessment Forum (U.S. EPA, 2016i). This method was originally designed for ecological assessment and offers some flexibility in its recommendations, so it has been adapted as fit-for-purpose to perform the weight-of-evidence analysis for TCE cardiac defects. Benefits of this method are as follows:
   a. The distinguishing feature of this method is that pieces of evidence are scored not just for reliability (quality) and relevance, as in most methods reviewed, but also strength of the evidence. EPA concurs with (U.S. EPA, 2016i) that explicitly scoring the strength of the individual pieces of evidence (e.g., magnitude, dose-response, etc.) is crucial to performing a weight-of-evidence assessment.
   b. The scoring system presented is qualitative and uses intuitive and easily understood symbols to convey both the implication of a piece of evidence (+, -, 0 for positive, negative, none, or supports, weakens, neutral/ambiguous) and the weight attached to it (+, ++, +++ or -, --, --- for low, medium and high). EPA believes that symbols are preferable to numerical scores because their use correctly implies that they cannot be numerically combined. They simply signify semi-quantitative levels of confidence, strength, and directionality of the results for the different qualitative properties.
   c. Assessment results are presented as weight-of evidence tables that show a visual picture of the findings. The tables capture nuances in the evidence being weighed and yet remain understandable. Seeing patterns in the frequencies of +, − and 0 symbols that indicate the weight of evidence is easier than if words or numbers are used to score evidence.
   d. The method is flexible. Although developed for use in ecological assessment, it is easily adaptable to use in human health assessment and to different approaches (e.g., individual pieces of evidence can be assessed and weighed for a line or type of evidence based on

source, such as inhalation toxicity studies, or for a line of evidence for a particular property (e.g., temporal association or other Hill consideration).

3) For our implementation of the (U.S. EPA, 2016i) weight-of-evidence method, EPA developed an Excel spreadsheet *[EPA, 2019. Data Table for Congenital Heart Defects Weight of Evidence Analysis. Docket: EPA-HQ-OPPT-2019-0500]*, as follows:
    a. The pieces of evidence are studies (or distinct experiments within studies). They are organized into lines of evidence based on study type: epidemiological, *in vivo* animal), and mechanistic. Within each line of evidence, pieces of evidence are further organized into subsets based on route of exposure (oral, inhalation, other) and test material (TCE or metabolite) for toxicological studies or vertebrate class of tissue, embryo or animal studied (mammalian, avian, fish) for mechanistic studies. WOE determinations are made in succession, first for subsets of a line of evidence, then for the full lines of evidence, and then for the overall database, each building on the assessments that came before.
    b. Each piece of evidence (study) was graded in 3 areas: reliability (quality), outcome/strength, and relevance. The rationale for each grade was recorded.
        i. Reliability is defined in (U.S. EPA, 2016i) as inherent properties that make evidence convincing. For our implementation, because each piece of evidence is a study, this refers primarily to aspects of study design, execution, and transparency.
            1. Possible scores for reliability were 0, +, ++, or +++ for unusable, low, medium and high.
            2. In contrast to the study quality evaluations performed in Distiller, which included >20 specific quality criteria for each study, here each study was given only a single overall grade. We considered the same issues, but we did not formally go through and assign grades on each one individually. Instead, focus was on key attributes. Noteworthy deficiencies were recorded and grades were assigned based on the number and nature of the specific deficiencies identified.
        ii. Outcome/strength is defined in (U.S. EPA, 2016i) as degree of differentiation from control, reference, or randomness. This is based on study results and may be influenced by magnitude, dose-response, number of related elements changed (e.g., consistent changes in histopathology and serum chemistry), temporal concordance, etc.
            1. Possible scores for outcome/strength were ---, --, -, 0, +, ++, or +++ for results ranging from strongly negative to no effect/ambiguous to strongly positive.
        iii. Relevance is defined in (U.S. EPA, 2016i) as degree of correspondence between the evidence and the assessment endpoint. This can be thought of as the degree of extrapolation that would be needed to use the data in question for developing a toxicity value.
            1. Possible scores for relevance were 0, +, ++, or +++ for none, low, medium and high.
            2. Maximum values based on study type were +++ for epidemiology studies, ++ for in vivo animal studies by natural route of exposure, and + for in vivo animal studies by other route of exposure and in vitro studies. Starting from these maximum scores, deductions were made for issues

such as testing of TCE metabolites rather than TCE for in vivo animal studies and poorly defined exposures in epidemiology studies.

iv. The grades for reliability, outcome/strength, and relevance for each piece of evidence (study) were integrated across each area (horizontally) into an overall grade for that study. In deriving the overall grade, low area scores were considered to have more weight than higher scores, as per (U.S. EPA, 2016i). In other words, if any one of the three grading areas was low, then even if other aspects of the study were rated highly, the study still contributed lower weight overall to the WOE analysis (e.g., a great study with a compelling result performed using DCA rather than TCE). Based on this methodology, overall grades for each study were always in the same direction as the strength score (i.e. + vs -) at a value defined by the lowest amplitude (+ vs ++ vs +++) of the three factors. Rationale for the overall grade was provided, as it was for the individual area grades.

c. When integrating overall study scores from all studies within a line of evidence (or subset of a line of evidence) or across lines of evidence (vertically), overall summary scores were determined as a the best semi-quantitative representation of all overall study grades within that line of evidence, with considerations given to both the amplitude of the overall study grades along with the consistency of the strength direction across studies. When results were mixed, overall summary scores for a line of evidence gave greater weight to overall study grades of greater amplitude (e.g., ++ vs +). Similarly, studies with non-ambiguous results (not a strength score of 0) were considered more informative than ambiguous studies. Additionally, consistent overall study grades of lower amplitude (e.g., all +) may have resulted in a summary score of a higher amplitude (++). In this way, WOE determination was most influenced by studies with the strongest, clearest effects and/or lines of evidence with the most consistent results. This differs from how the individual area grades were combined into overall study grades (See Section b(iv), above), where the lowest amplitude value determined the overall weight.

d. Evidence areas were also integrated as a mathematical average (e.g., ++ = 2, 0/- = -0.5), in order to summarize the evidence areas for all studies. In contrast with the overall summary score however, for individual evidence areas, the integrated area scores represented a true average and were not adjusted upward for consistency or in order to favor non-ambiguous results (which was specific to strength score). Of note, these are included for presentation purposes only and were not used to determine the overall summary score for a line of evidence. The overall summary scores were determined by integrating the overall grades for each study, in the manner as described in Section c. Because of these different methodologies and the fact that overall study grades are defined by the lowest amplitude evidence area, the overall summary score may differ from the integrated area scores.

Note: This analysis was performed in parallel with the systematic review data evaluation of the individual studies. The WOE analysis had a greater focus on relevance to the specific endpoint while the data evaluation metrics aimed to evaluate the utility of a study for dose-response analysis. Therefore, the conclusions of the WOE analysis for individual studies occasionally differed from the results of the systematic review data evaluation. The results of both are presented together in *[EPA, 2019. Data Table for Congenital Heart Defects Weight of Evidence Analysis. Docket: EPA-HQ-OPPT-2019-0500.]*. Of note, studies that scored Unacceptable in data quality evaluation were not considered in the WOE analysis. Their evaluation is included for reference, but their scores had no impact on the overall grades

for each line of evidence or subset. Unacceptable studies are indicated by red text in the below tables and the supplemental data table. Studies that were not rated (NR) because EPA determined that they were not pertinent are indicated by blue text in the supplemental data table, however they are not included in the tables below.

G.2.2 WOE Results By Study Type

Data evaluated to assess the weight-of-evidence for congenital heart defects from exposure to TCE include studies from three lines of evidence: epidemiology studies, *in vivo* animal toxicity studies, and mechanistic studies. For this analysis, the three lines of evidence will be considered both individually and collectively.

Table_Apx G-8 shows the weight-of-evidence for the various epidemiology studies that were considered in this review. Ruckart et al. (2013) was identified in previous reviews but was graded as NR (not relevant) and dropped from the analysis because the study did not include cardiac defects as an assessed endpoint. All of the other TCE studies were considered to be of (++) relevance scores because they examined associations of TCE exposure in humans, however quantitative exposure to TCE was assessed indirectly in all of them. One study that examined exposure to TCE degradants (Wright et al., 2017) scored only (+) for relevance because the degradants may also have originated from a different source. The high potential for misclassification of exposure was a limiting factor for all of these studies, which were otherwise generally adequate ecological or case-control studies (reliability rated as + for all studies). Of the relevant studies, four reported results suggestive of a positive association between maternal TCE exposure and congenital cardiac defects in offspring, one reported a lack of an association, and two reported ambiguous results. Of the three studies with a positive association, (Goldberg et al., 1990) was rated Unacceptable in data quality evaluation and therefore did not contribute to the WOE. The Bove reports (1996; 1995) (considered here as a single study because the two papers contain the same data on cardiac defects) reported elevated but nonsignificant increases in odds ratios. Yauck et al. (2004) reported a positive association between congenital heart defects and TCE exposure only in older mothers, while younger mothers and the overall population had a null association. The finding of a negative association in the study by (Lagakos et al., 1986) has some ambiguity because it was based on a very small number of cases, exposure was not classified based on TCE specifically, and there was atypical directionality of confounder effects. Gilboa et al. (2012) did not find any positive association with TCE exposure in a large but limited study. Three studies showing positive associations of varying strength (Brender et al., 2014; Forand et al., 2012; Wright et al., 2017) also had some limitations but collectively provide suggestive evidence for an association between maternal TCE exposure and cardiac defects in offspring. In evaluating all studies and giving greater weight to studies with non-ambiguous results, the resulting overall summary score for epidemiology is (+), indicating a positive association between TCE exposure and congenital cardiac defects.

Table_Apx G-8. Weight-of-Evidence Table for Epidemiology Studies

Evidence Area	Reliability	Strength	Relevance	Overall Grade
TCE				
(Lagakos et al., 1986)	+	0/-	++	0/-
(Bove, 1996; Bove et al., 1995)	+	0	++	0
(Yauck et al., 2004)	+	0/+	++	0/+
(Forand et al., 2012)	+	++	++	+

Evidence Area	Reliability	Strength	Relevance	Overall Grade
(Gilboa et al., 2012)	+	-	++	-
(Brender et al., 2014)	+	+	++	+
(Goldberg et al., 1990)	0	+	++	0
METABOLITES (TCA, DCA)				
(Wright et al., 2017)	++	+	+	+
Integrated Area Scores (all epidemiology)	+	0/+	++	
Summary Score (all epidemiology)				+

Possible scores for reliability and relevance were 0, +, ++, or +++ for unusable, low, medium and high.
Possible scores for strength and overall weight were ---, --, -, 0, +, ++, or +++, with ranges inbetween, for results ranging from strongly negative to ambiguous to strongly positive.
Red text identifies studies that scored Unacceptable in data quality evaluation and a 0 for reliability. The WOE scores are provided for reference but were not incorporated into the overall score for the line of evidence.

Table_Apx G-9 shows the weight-of-evidence for the various *in vivo* animal studies that were considered in this review. The four TCE oral studies were considered of (++) relevance because they used a natural route of exposure (drinking water or gavage) in a mammallian study. Dawson et al. (1993) and the Charles River Laboratories study (2019) were rated as (++) reliability, while Fisher et al. (2001) and Johnson et al. (2003) were rated as (+) reliability. The score was downgraded for (Fisher et al., 2001) because only a single dose group was used and the negative control for TCE demonstrated a very elevated prevalence of heart and cardiovascular defects. Johnson et al. (2003) was rated as lower reliability due to the small group sizes, poor data reporting (somewhat mitigated by subsequent errata and personal communications), and the pooling of data from multiple trials into a single experiment. Increased incidence of cardiac defects were observed in pups from the (Dawson et al., 1993) and (Johnson et al., 2003) studies. The Strength scores for these studies were characterized as (++) for (Johnson et al., 2003) and (+) for (Dawson et al., 1993), influenced by the low magnitude of effect in the high dose groups and uncertainty surrounding the precision of estimated doses. The incidence of cardiac defects were not increased by TCE oral gavage in the (Fisher et al., 2001) study; however, this study used only a single dose group and the incidence of heart defects was elevated in the soybean oil controls compared to drinking water controls, therefore the strength score was (0/-). The recent study by Charles River Laboratories (2019) also did not find any statistically significant increase in developmental cardiac defects following TCE administration in drinking water, however this study appeared to be of reduced sensitivity in its ability to detect all types of cardiac defects (see Appendix G.1). It therefore also scored (0/-) for Strength. The overall summary for the TCE oral studies was characterized as ambiguous to weakly positive (0/+) due to conflicting study results, with a lean toward positive based on the ambiguity of the negative studies.

Six oral experiments using TCE metabolites (TCA or DCA) were rated as lower relevance (+), because a metabolite was administered (not TCE) and the relevance of these effects to humans likely dependent upon individual toxicokinetic variability and the administered dose. These studies were considered mostly reliable with ratings of (+++) (Smith et al., 1989) and (++) (Fisher et al., 2001; Epstein et al., 1992). Only (Johnson et al., 1998) received a lower reliability score (0/+) due to concerns about source of the test substance and sharing of bottles among animals. Both TCA and DCA were convincingly shown to produce strong dose-related cardiac defects (strength score of ++) in the (Smith et al., 1992, 1989) studies (downgraded for use of relatively high doses that produced other embryo/fetotoxic effects

or even maternal effects), with weaker positive strength scores (+) in the (Johnson et al., 1998) and (Epstein et al., 1992) studies. The (Fisher et al., 2001) study (also reviewed separately for TCE administration) only showed a small, non-statistically significant increase in cardiac defects for both TCA and DCA, but the single dose level used in these studies was too low to rule out effects at higher doses based on results of the other studies. The overall summary score for the oral metabolite studies was (+).

Three inhalation studies using TCE were considered relevant (natural exposure route) and reliable. Reliability ratings were reduced for studies with a single exposure group and poor reporting (+, (Schwetz et al., 1975)) in addition to small group sizes and high negative control responses with a lack of dose-responsiveness (0/+, (Dorfmueller et al., 1979)). These studies were also reduced in relevancy score (+) because they were general teratology studies and the focus on cardiac effects was unclear. Two studies scored an Unacceptable in data quality and a 0 in reliability for limited reporting of study details (Hardin et al., 1981) and use of a nonstandard exposure duration with insufficient details on exposure method (Healy et al., 1982). These studies did not contribute to the WOE. Among acceptable inhalation studies, the results were consistently negative, however with varying scores in the three evidence areas. Carney et al. (2006) was the best inhalation study, scoring the maximum (+++) for reliability and showing a strong negative response (--). Based on these results, the summary score for the inhalation studies was (-), primarily driven by the weight of the (Carney et al., 2006) data but reduced by the weaknesses of the other studies and the limited number of acceptable studies with non-ambiguous results.

As for other exposure routes, Dawson et al. (1990) administered TCE via intrauterine instillation in rats. This relevance of this study was rated as lower (+) due to the unnatural exposure route and the study reliability was low (0/+), because of sampling inadequacy, small group sizes, and poor reporting. The strength of this study was (+) due to several factors, including the use of fetuses (not litters) as the experimental unit, the small magnitude of the response seen in the high dose group only (which was a very high dose considering the exposure route). The overall summary score for animal studies across all exposure routes suggests an unclear/ambiguous relationship between TCE exposure during gestation and the incidence of cardiac defects in offspring. This ambiguity is based on weakly positive evidence from oral or intrauterine TCE administration, positive evidence from oral TCE metabolites, and a negative evidencewith TCE inhalation. The WOE from *in vivo* animal toxicity studies therefore does not either support or refute the association of TCE exposure with developmental cardiac defects.

Table_Apx G-9. Weight-of-Evidence Table for *In Vivo* Animal Toxicity Studies

Evidence Area	Reliability	Strength	Relevance	Overall Grade
ORAL				
TCE				
(Dawson et al., 1993)	++	+	++	+
(Johnson et al., 2003)	+	++	++	+
(Fisher et al., 2001)	+	0/-	++	0/-
(Charles River Laboratories, 2019)	++	0/-	++	0/-
Integrated Area Scores	+/++	0/+	++	
Summary Score (TCE)				0/+

Evidence Area	Reliability	Strength	Relevance	Overall Grade
METABOLITES (TCA, DCA)				
(Smith et al., 1989)	+++	++	+	+
(Smith et al., 1992)	+++	++	+	+
(Johnson et al., 1998)	0/+	+	+	0/+
(Fisher et al., 2001)	++	-	+	-
(Epstein et al., 1992)	++	+	+	+
Integrated Area Scores	++	+	+	
Summary Score (Metabolites)				+
Integrated Area Scores (all oral studies)	++	+	++	
Summary Score (all oral studies)				+
INHALATION				
TCE				
(Schwetz et al., 1975)	+	0/-	+	0/-
(Dorfmueller et al., 1979)	0/+	0/-	+	0/-
(Carney et al., 2006)	+++	--	++	--
(Hardin et al., 1981)	0	-	++	0
(Healy et al., 1982)	0	-	++	0
Integrated Area Scores (all inhalation studies)	+/++	-	+/++	
Summary Score (all inhalation studies)				-
OTHER ROUTES (Uterine Infusion)				
(Dawson et al., 1990)	0/+	+	+	0/+
Integrated Area Scores (*in vivo* - all routes)	+/++	0/+	+/++	
Summary Score (*in vivo* - all routes)				0

Possible scores for reliability and relevance were 0, +, ++, or +++, with ranges inbetween, for unusable, low, medium and high. Possible scores for strength and overall weight were ---, --, -, 0, +, ++, or +++, with ranges inbetween, for results ranging from strongly negative to ambiguous to strongly positive.
Red text identifies studies that scored Unacceptable in data quality evaluation. The WOE scores are provided for reference but were not incorporated into the overall score for the line of evidence.

Mechanistic studies that inform the weight-of-evidence for developmental heart defects include evaluations of cardiac structure and function in chick and rodent embryos and mode-of-action or key event data focused on processes and pathways that contribute to the observed valvulo-septal defects (e.g., altered calcium flux, inhibition of stem cell differentiation and endothelial cell proliferation) as well as altered expression of oxidative metabolism enzymes. A mechanistic study from Palbykin et al. (2011) was graded as not relevant and was dropped from the analysis because it merely examined

molecular mechanisms underlying the results observed in (Caldwell et al., 2008) without contributing any additional WOE to the endpoint. The remaining mechanistic studies in mammalian cells/tissues, chick embryos and zebrafish embryos were generally rated as lower relevance in comparison to human studies and *in vivo* animal studies using a natural route of administration except for studies on *ex vivo* whole rat embryos or *in vivo* data from rodents or humans, which were assigned a relevance score of (+/++). All other studies were rated as (+) relevance.

Mechanistic studies in mammalian systems included an occupational worker study (Green et al., 2004), *in vivo* rat studies (Collier et al., 2003; Dow and Green, 2000), studies using rat and mouse whole embryo cultures (Hunter et al., 1996; Saillenfait et al., 1995) and *in vitro* studies using cell lines (Jiang et al., 2015; Caldwell et al., 2008; Selmin et al., 2008; Ou et al., 2003). Ou et al. (2003) and Jiang et al. (2015) were rated as highly reliable (+++) because they were well-designed and well-conducted studies with a full reporting of the results. Most of the remaining mammalian studies were rated as (++) for reliability, because there were minor deficiencies noted in study design, performance or reporting. Dow and Green (2000) was rated as low (0/+) for reliability, with flaws including pooling of experiments, poor data reporting, and insufficient justification of dose selection. In mammalian systems, higher strength (++) was ascribed to studies that demonstrated structural changes in the embryonic heart (Hunter et al., 1996), suppression of endothelial cell proliferation in cell culture (Ou et al., 2003), and inhibition of cardiac differentiation from embryonic stem cells (Jiang et al., 2015). Studies that demonstrated precursor events that contribute to altered cardiac development (i.e., changes in gene expression, altered calcium flux, folate deficiency) were rated as weakly positive (+) for strength. These included changes in gene expression relating to cardiac development and calcium flux (Jiang et al., 2015; Caldwell et al., 2008; Selmin et al., 2008; Collier et al., 2003) and *in vivo* folate deficiency (Green et al., 2004; Dow and Green, 2000) (which has been associated with congenital heart defects in humans (Mao et al., 2017)). Saillenfait et al. (1995) did not observe morphological cardiac changes in whole rat embryos exposed to TCE in culture, although only morphological features were examined and the results were not explicitly discussed in the text. This study was rated as moderately negative (-/--) for strength.

With the exception of the Saillenfait study (which did not describe its procedure for evaluation of malformations in whole rat embryos), the other mammalian mechanistic studies all reported positive results. Several of these studies demonstrated a clear dose-response, although in others the results were less clear (e.g., suggestive of a biphasic dose-response, with change at the lower doses but not the higher doses, see discussion in Section 3.2.4.1.6). The overall summary score for mammalian mechanistic studies was (+).

The chick embryo is a valid model system for studying embryonic development, and in particular, cardiac development. Eight studies investigated development of cardiac defects and associated effects in chick embryos exposed to TCE and metabolites. These were all generally well-designed, conducted and reported. All chick embryo studies received a (++) rating for reliability except for (Loeber et al., 1988), which was downgraded slightly to (+/++) due to missing reporting details and a potentially insensitive evaluation procedure. Two studies reported significant increases in incidences of a variety of cardiac defects (Rufer et al., 2010; Loeber et al., 1988), resulting in a a strength rating of (++). The remaining studies showed various mechanistic changes thought to be involved in cardiac development or function and scored less positive for strength, (+). The only study that did not produce a clear positive result featured an earlier exposure window than the others and obtained ambiguous results with mixed results on endocardiocyte proliferation and no changes in cardiac output was rated as (0) for

strength (Drake et al., 2006b). The overall summary score for chick embryo studies was (++) based on the relatively large number of studies demonstrating consistently positive effects.

The zebrafish embryo is also a valid model for evaluating cardiac development. Two of the three zebrafish embryo studies were well designed and well documented with few notable limitations (rated as highly reliable, +++). The reliability rating for (Williams et al., 2006) was reduced to (++) due to the use of a single exposure level. All three studies gave positive results indicating the potential for TCE (or its metabolite DCA) to effect cardiac development in zebrafish. The study by Wirbisky et al. (2016) was the most comprehensive study of the three (rated as +++ for strength), identifying multiple dose-responsive cardiovascular effects as well as associated gene changes. The other two studies received a (++) for strength because of observed severe changes in heart rate but at concentrations associated with other toxicities (Hassoun et al., 2005) or because only a single, elevated dose was used (Williams et al., 2006). The overall summary score for zebrafish embryo studies was (+). The overall summary score for mechanistic studies across all species and study designs was (++) due to consistent positive outcomes observed in all study types. The WOE from mechanistic studies therefore provides stronger positive evidence of an association between TCE exposure and congenital cardiac defects.

Table_Apx G-10. Weight-of-Evidence Table for Mechanistic Studies

Evidence Area	Reliability	Strength	Relevance	Overall Grade
MAMMALIAN CELLS/TISSUE				
TCE				
(Saillenfait et al., 1995)	++	-/--	+/++	-/--
(Collier et al., 2003)	++	+	+	+
(Selmin et al., 2008)	++	+	+	+
(Caldwell et al., 2008)	++	+	+	+
(Ou et al., 2003)	+++	++	+	+
(Jiang et al., 2015)	+++	++	+	+
(Dow and Green, 2000)	0/+	+	+/++	0/+
(Green et al., 2004)	++	+	+/++	+
METABOLITES (TCA, DCA, Trichloroethanol, Chloral)				
(Saillenfait et al., 1995)	++	-/--	+/++	-/--
(Collier et al., 2003)	++	+	+/++	+
(Hunter et al., 1996)	++	++	+/++	+
(Selmin et al., 2008)	++	+	+	+
(Dow and Green, 2000)	++	+	+	+
Integrated Area Scores	++	+	+	
Summary Score (all mammalian tissue studies)				+
CHICK EMBRYO				
TCE				

Evidence Area	Reliability	Strength	Relevance	Overall Grade
(Loeber et al., 1988)	+/++	++	+	+
(Boyer et al., 2000)	++	+	+	+
(Mishima et al., 2006)	++	+	+	+
(Drake et al., 2006a)	++	+	+	+
(Drake et al., 2006b)	++	0	+	0
(Rufer et al., 2010)	++	++	+	+
(Makwana et al., 2010)	++	+	+	+
(Makwana et al., 2013)	++	+	+	+
METABOLITES (TCA)				
(Harris et al., 2018)	++	+	+	+
(Drake et al., 2006a)	++	+	+	+
(Drake et al., 2006b)	++	0	+	0
Integrated Area Scores	++	+	+	
Summary Score (all chick studies)				++
ZEBRAFISH EMBRYO				
TCE				
(Wirbisky et al., 2016)	+++	+++	+	+
METABOLITES (DCA)				
(Hassoun et al., 2005)	+++	++	+	+
(Williams et al., 2006)	++	++	+	+
Integrated Area Scores	+++	++/+++	+	
Summary Score (all zebrafish studies)				+
Integrated Area Scores (all mechanistic studies)	+++	+/++	+	
Summary Score (all mechanistic studies)				++

Possible scores for reliability and relevance were 0, +, ++, or +++, with ranges inbetween, for unusable, low, medium and high. Possible scores for strength and overall weight were ---, --, -, 0, +, ++, or +++, with ranges inbetween, for results ranging from strongly negative to ambiguous to strongly positive.

In summary, the database contains a large and diverse set of studies pertinent to assessing congenital heart defects from TCE exposure (overall relevance was rated as ++). Well-designed, conducted and reported studies were located for all categories, although the epidemiology studies were limited to ecological or case-control study designs with high potential for misclassification of exposure and the many of the *in vivo* animal studies contained at least one major limitation (overall reliability rating of +/++). The integrated strength area score was (+), indicating a suggestive positive association of TCE with congenital cardiac defects. The epidemiology studies as a group provide suggestive evidence for an effect of TCE on cardiac defects in humans (summary score of +). Oral *in vivo* studies provided

ambiguous to weakly positive (0/+) results for TCE itself, but positive results for its TCA and DCA metabolites (+), while inhalation studies contributed negative evidence (-). Mechanistic studies provided solid, consistent supporting information for effects of TCE and metabolites on cardiac development and precursor effects (summary score of ++). Overall, the database is both reliable and relevant and provides positive overall evidence that TCE may produce cardiac defects in humans (based on positive evidence from epidemiology studies, mixed evidence from animal toxicity studies, and stronger positive evidence from mechanistic studies).

Table_Apx G-11. Overall Weight-of-Evidence Table and Summary Scores

Evidence Area	Reliability	Strength	Relevance	Summary Score
Epidemiology studies	+	+	++	+
In vivo animal toxicity studies	+/++	0/+	+/++	0
Mechanistic studies	+++	+/++	+	++
Integrated Area Scores	++	+	++	+

Possible scores for reliability and relevance were 0, +, ++, or +++, with ranges inbetween, for unusable, low, medium and high. Possible scores for strength and overall weight were ---, --, -, 0, +, ++, or +++, with ranges inbetween, for results ranging from strongly negative to ambiguous to strongly positive.

Appendix H META-ANALYSIS FOR CANCER

H.1 Study Screening and Selection

All epidemiologic studies included in the U.S. EPA 2011 IRIS assessment of TCE (Appendix C, (U.S. EPA, 2011b) were considered to be informative and carried forward for meta-analysis. Informative epidemiologic studies of non-Hodgkin lymphoma (NHL), kidney cancer or liver cancer and exposure to TCE published since the 2011 IRIS assessment were identified through a systematic literature search. Studies examining only other cancer types were excluded from consideration.

H.1.1 Data Quality and Inclusion/Exclusion Criteria Screening

Relevant studies were evaluated for data quality and were additionally screened through inclusion/exclusion criteria developed based on the criteria established in the 2011 IRIS assessment (Appendix C, (U.S. EPA, 2011b)), as described in Table_Apx H-1. Results of this criteria screening are presented in
Table_Apx H-2.

Table_Apx H-1. Meta-Analysis Inclusion/Exclusion Criteria for Considering Cancer Studies Identified in EPA's Literature Search

Inclusion Criteria	Exclusion Criteria
Study Design	
Cohort and case control studies.	Geographic-based, ecological, or proportionate mortality ratio (PMR) study design.
Participant Selection	
Adequate selection in cohort studies of exposure and control groups and of cases and controls in case-control studies.	Inadequate selection in cohort studies (exposed and control groups were not similar, and differences were not controlled for in the statistical analysis). Controls were drawn from a very dissimilar population than cases or recruited within very different time frames (case control studies).
Exposure	
TCE exposure potential inferred to each subject and quantitative assessment of TCE exposure for each subject by reference to industrial hygiene records indicating a high probability of TCE use, individual biomarkers, job exposure matrices (JEMs), water distribution models, or obtained from subjects using questionnaire (case-control studies).	TCE exposure potential not assigned to individual subjects using JEM, individual biomarkers, water distribution models, or industrial hygiene data indicating a high probability of TCE use (cohort studies).
Reports as least 2 levels of exposure (e.g., exposed/unexposed).	The range and distribution of exposure are not adequate to determine an exposure-response relationship. No description is provided on the levels or range of exposure.
Outcome Assessment	
Evaluation of incidence or mortality from kidney cancer, liver cancer, or NHL. RR estimates and corresponding CIs (or information to allow calculation).	Data for non-cancer health outcomes or incidence or mortality reported for cancers other than kidney, liver, or NHL. All hemato- and lymphopoietic cancer reported as broad category.
Statistical Power (sensitivity)	
The number of participants or cases and controls are adequate to detect an effect in the exposed population and/or subgroups of the total population.	The number of participants or cases and controls are inadequate to detect an effect in the exposed population and/or subgroups of the total population.

Table_Apx H-2. Screening Results of Cancer Studies Identified in EPA's Literature Search Based on Inclusion/Exclusion Criteria

Studies recommended for inclusion in quantitative meta-analysis:	
Studies	**Primary reason(s)**
(Bove et al., 2014a) (Bove et al., 2014b) (Buhagen et al., 2016) (Christensen et al., 2013) (Cocco et al., 2013) (Hansen et al., 2013) (Lipworth et al., 2011) (Purdue et al., 2016) (Silver et al., 2014) (Vlaanderen et al., 2013)	Analytical study designs of cohort or case-control; evaluation of incidence or mortality; adequate selection in cohort studies of exposure and control groups and of cases and controls in case-control studies; TCE exposure potential inferred to each subject and quantitative assessment of TCE exposure assessment for each subject by reference to industrial hygiene records indicating a high probability of TCE use, individual biomarkers, JEMs, water distribution models, or obtained from subjects using questionnaire (case-control studies); RR estimates for kidney cancer, liver cancer, or NHL with confidence intervals

Studies NOT recommended for inclusion in quantitative meta-analysis:	
Studies	**Primary reason(s)**
(Alanee et al., 2015)	Weakness with respect to analytical study design (i.e., geographic-based, ecological or PMR design).
(Alanee et al., 2015)	TCE exposure potential not assigned to individual subjects using JEM, individual biomarkers, water distribution models, or industrial hygiene data from other process indicating a high probability of TCE use (cohort studies).
(Bassig et al., 2016) (Ruckart et al., 2013)	Examined noncancer health outcomes or cancer incidence or mortality for cancers other than kidney, liver, or NHL. All hemato- and lymphopoietic cancer reported as broad category.
(Bahr et al., 2011)	EPA reviewer scored the study as Unacceptable (Rationale: Repeated examples of poor quality, study design and execution and ignorance of potential biases that went unmentioned even in the discussion indicate inexperience and poor quality control).

H.1.2 Screening results

Data quality and inclusion/exclusion criteria screening identified ten studies suitable for use in meta-analysis. Of these, there were nine new studies with suitable informative data on the association of exposure to TCE and NHL (Bove et al., 2014a; Bove et al., 2014b; Christensen et al., 2013; Cocco et al., 2013; Hansen et al., 2013; Lipworth et al., 2011; Purdue et al., 2016; Silver et al., 2014; Vlaanderen et al., 2013), eight new studies with informative data for kidney cancer (Bove et al., 2014a; Buhagen et al., 2016; Christensen et al., 2013; Hansen et al., 2013; Lipworth et al., 2011; Purdue et al., 2016; Silver et al., 2014; Vlaanderen et al., 2013), and six new studies with informative data for liver cancer (Bove et al., 2014a; Christensen et al., 2013; Hansen et al., 2013; Lipworth et al., 2011; Silver et al., 2014; Vlaanderen et al., 2013). All of these studies scored Acceptable for data quality except (Bahr et al., 2011), which was excluded for scoring Unacceptable. Every study scored at least a Medium except for (Buhagen et al., 2016), which scored a Low but was recommended for inclusion by inclusion/exclusion criteria. The respective data quality scores were considered in sensitivity analyses of the meta-analyses results (see Appendix H.2.2.2).

All studies from the 2011 IRIS meta-analysis were Acceptable in data quality and scored at least a Medium. Therefore, data from the ten new studies that passed the criteria screening were extracted along

with results from previous studies identified in the 2011 IRIS assessment (U.S. EPA, 2011e). When more than one report was available for a single study population, only the most recent publication or the publication reporting the most informative data for TCE was selected for inclusion in the meta-analysis (see Table_Apx H-3). This resulted in a smaller set of data included in the meta-analysis as compared to the total list of studies.

H.1.3 Pooled Cohorts

Two of the new papers pooled data from earlier studies included in the 2011 IRIS meta-analysis. (Hansen et al., 2013) pooled and updated three Nordic national cohort studies of workers biologically monitored for exposure to TCE (Anttila et al., 1995; Axelson et al., 1994; Hansen et al., 2001). Similarly, (Cocco et al., 2013) pooled earlier case-control studies of NHL including (Cocco et al., 2010), (Miligi et al., 2006), and (Purdue et al., 2011). Two other new studies provided updated data on populations included in the U.S. EPA 2011 IRIS assessment: (Lipworth et al., 2011) updated a cohort study of aircraft workers (Boice et al., 1999) and (Christensen et al., 2013) updated an earlier population-based case-control study (Siemiatycki, 1991). After removing these overlapping and superseded studies, a total of 18 studies of NHL, 18 studies of kidney cancer, and 11 studies of liver cancer were available for meta-analysis.

Among the included studies, up to about 800 of the approximately 40,000 Danish workers studied by (Raaschou-Nielsen et al., 2003) may have also been included in the Nordic pooled study of 5553 biomonitored workers (Hansen et al., 2013). However, both studies were retained in the analysis because any overlap would have been minor. There was also minor overlap between the cohorts studied by (Zhao et al., 2005) and (Boice et al., 2006), but those papers reported data for different outcomes. These results are summarized in Table_Apx H-3.

Table_Apx H-3. Cancer Studies Covering the Same Cohort as Previous Studies from either the 2011 IRIS Assessment or EPA Literature Search

Study reviewed	Other assessed studies with participants from the same cohort
2011 IRIS Assessment	
(Anttila et al., 1995) (Finland only)	Included in (Hansen et al., 2013)
(Axelson et al., 1994) (Sweden only)	Included in (Hansen et al., 2013)
(Boice et al., 1999)	Updated in (Lipworth et al., 2011)
(Boice et al., 2006)	(Zhao et al., 2005) (partial)
(Brüning et al., 2003)	None
(Charbotel et al., 2006)	None
(Cocco et al., 2010)	Included in (Cocco et al., 2013)
(Dosemeci et al., 1999)	None
(Greenland et al., 1994)	None
(Hansen et al., 2001) (Denmark only)	(Raaschou-Nielsen et al., 2003) (partial); Included in (Hansen et al., 2013)
(Hardell et al., 1994)	None
(Miligi et al., 2006)	Included in (Cocco et al., 2013)
(Moore et al., 2010)	None

Study reviewed	Other assessed studies with participants from the same cohort
(Morgan et al., 1998)	None
(Nordström et al., 1998)	None
(Persson and Fredrikson, 1999)	None
(Pesch et al., 2000)	None
(Purdue et al., 2011)	Included in (Cocco et al., 2013)
(Raaschou-Nielsen et al., 2003)	Partial overlap with (Hansen et al., 2001)
(Radican et al., 2008)	None
(Siemiatycki, 1991)	Updated in (Christensen et al., 2013)
(Wang et al., 2009)	None
(Zhao et al., 2005)	(Boice et al., 2006) (partial)
New Studies Identified in EPA Literature Search	
(Bove et al., 2014a)	None
(Bove et al., 2014b)	None
(Buhagen et al., 2016)	None
(Cocco et al., 2013)	(Cocco et al., 2010); (Miligi et al., 2006); (Purdue et al., 2011)
(Christensen et al., 2013)	(Siemiatycki, 1991)
(Hansen et al., 2013)	(Hansen et al., 2001); (Anttila et al., 1995); (Raaschou-Nielsen et al., 2003) (partial)
(Lipworth et al., 2011)	(Boice et al., 1999)
(Purdue et al., 2016)	None
(Silver et al., 2014)	None
(Vlaanderen et al., 2013)	None

H.2 Meta-Analysis Methods and Results

H.2.1 Methods

Data abstraction

Data for each pertinent study identified, including measures of the association (including rate ratio (RR), odds ratio (OR), hazard ratio (HR), etc.) of each cancer of interest with exposure to TCE, their confidence intervals (CI) and if reasonably available, standard errors, identification of the type of measure (RR, OR, etc), the study design and the exposure metric (ever/never exposed, cumulative exposure, duration of exposure, etc.) were abstracted for meta-analysis. All types of epidemiologic ratio measures of association, including RR, OR, HR and standardized mortality or incidence ratios (SMR, SIR), were considered to be equivalent and are collectively referred to below as RRs. The preferred estimates of association for meta-analysis were based on contrasts within the study population and were either 1) comparisons of groups exposed and not exposed to perchloroethylene or 2) comparisons of groups with the highest and lowest level of exposure to perchloroethylene, in that order. For NHL, estimates of association for the most highly exposed group were also abstracted, when they were reasonably available. For each comparison, the most fully adjusted risk estimate was selected.

Estimates of association based on cumulative exposure were preferred to those based on other exposure metrics.

Data for studies included in the U.S. EPA 2011 IRIS assessment (U.S. EPA, 2011e) were abstracted from tables in Appendix C of that assessment. The measures of association, confidence limits and estimates of SE listed in those tables were utilized for consistency with the previous assessment.

For newer studies not included in the IRIS assessment, log-relative risks and their standard errors were estimated from the extracted data; the data for the newer studies are provided in tables in Section H.2.3. If the standard error (SE) of RR was reported in the publication, the standard error of ln(RR) was taken as ln(SE). If SE was not reported and the CI was reasonably symmetric around the point estimate (< 5% difference between upper and lower half CI), it was approximated as (ln(upper bound CI)-ln(lower bound CI))/3.92. Different approaches in the event of more substantial CI asymmetry. If the measure of RR was a SMR or SIR, SE was approximated by $(1/O)^{1/2}$, where O is the observed number of cases (Greenland & O'Rourke, 2008). If RR was 1 or >1, SE was estimated from the upper half CI, as (ln(upper bound CI) – ln(RR))/1.96. For RR < 1, SE was estimated from the lower half CI in an equivalent manner. Despite these varying approaches, differences in the method of estimating SE are unlikely to substantially affect the point estimate or CI of a meta-RR.

Data analysis

Meta-analyses were performed using the metan procedure in Stata (Stata Corp, College Station TX). The metan procedure also provides options for utilizing a user-provided estimate of SE or estimating SE from input confidence intervals assuming approximate symmetry.

For each cancer type of interest, the initial analysis included all of the selected studies in a fixed-effects model. Models were specified using the logs of RR and SE as input parameters, allowing the software to estimate study-specific and overall 95% CIs. Heterogeneity was assessed using the I^2 statistic (Higgins et al., 2003) and visual inspection of the plots. If no important heterogeneity was indicated, the fixed-effects meta-estimate was taken as the measure of overall association. Fixed effects models are preferred for this purpose, as they are generally unbiased (Poole and Greenland, 1999). Where notable heterogeneity was indicated, a random-effects model using the DerSimonian-Laird estimators was applied to estimate the overall association. EPA's preferred approach is to estimate SE according to the methods described above. With this procedure, the study-specific CIs displayed on forest plots were estimated by the software and may differ slightly from those reported in the original publications.

The influence of individual studies was assessed in a "leave one out" meta-analysis using the metaninf procedure in Stata. Each study was omitted in turn and the meta-estimate was re-calculated without that study to gauge its effect on the overall association. Meta-analyses stratified by the quality score assigned in the initial reviewer were carried out to assess whether effects differed in high versus medium- or low-quality studies.

The potential for publication bias was assessed by visual inspection of funnel plots.

Sample Stata commands are provided in Section H.2.4.

H.2.2 Results

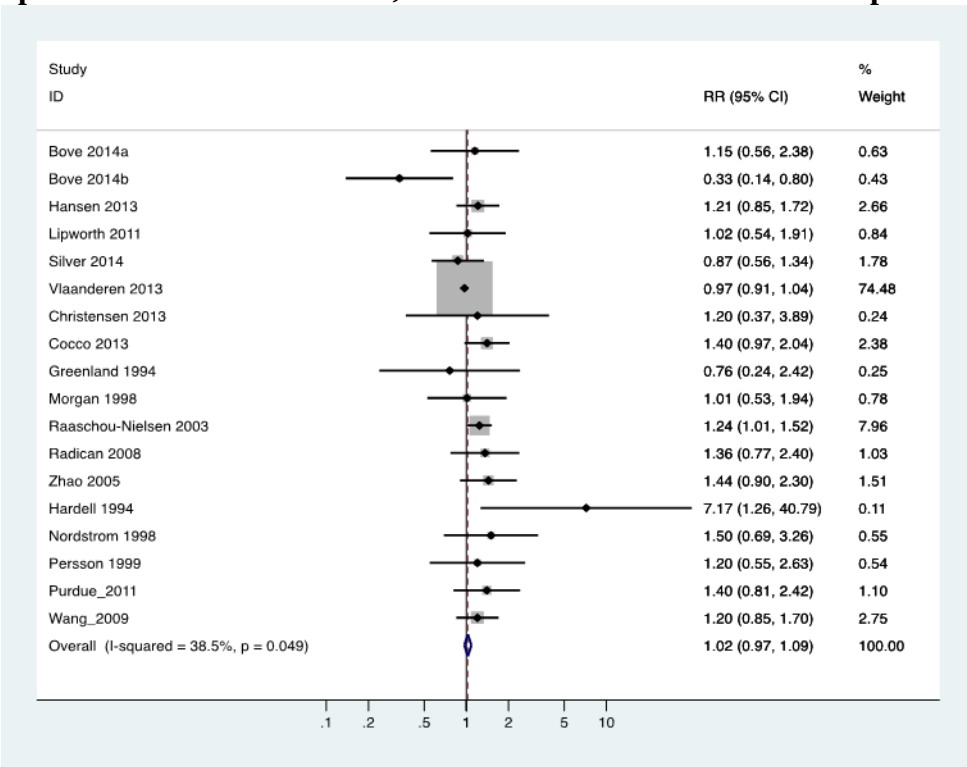
H.2.2.1 Initial Meta-Analyses

Non-Hodgkin lymphoma

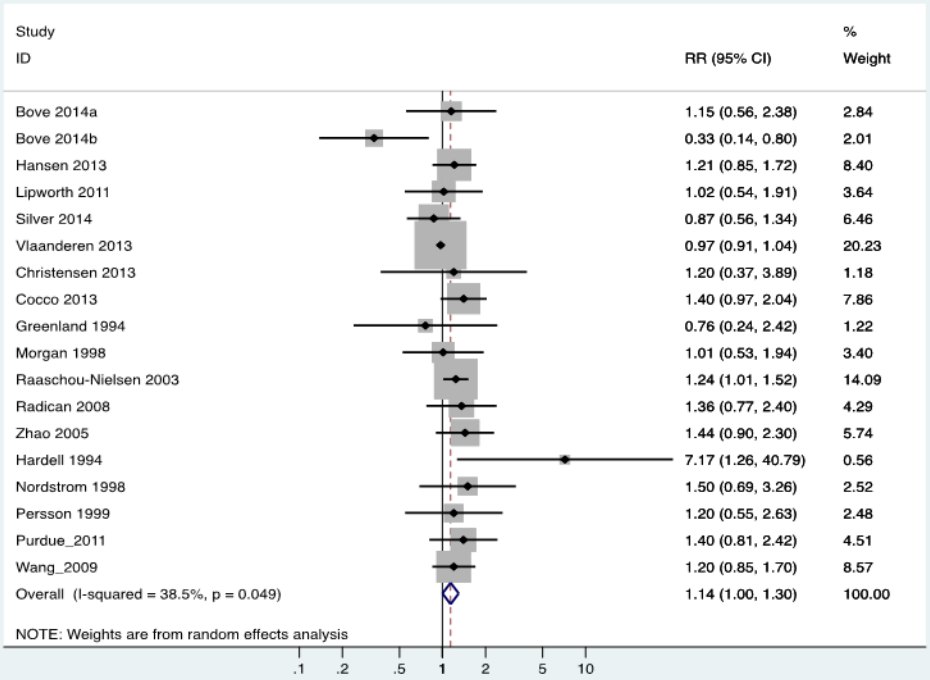
In the fixed-effects model for NHL (Figure_Apx H-1), the meta-RR for overall exposure to TCE was 1.02 (95% CI 0.97-1.08) with moderate heterogeneity between studies (I^2 38.4%, p 0.05). The large study by Vlaanderen et al. (2013) was heavily weighted in the fixed-effects model. Fitting a random-effects model (Figure_Apx H-2) to the same set of studies reduced the weight of the (Vlaanderen et al., 2013) study and gave a meta-estimate of 1.14 (95% CI 1.00-1.30).

In the 2011 TCE meta-analysis of NHL, there was some indication of heterogeneity (I^2-value was 26%, suggesting low-to-moderate heterogeneity). Little to no heterogeneity was found for kidney or renal cancers. Additional analyses focused on the studies with the highest exposure, because if TCE exposure increases the risk of NHL, the effects should be more apparent in the highest exposure groups. Analysis showed that the summary effect estimate of the highest exposed groups was stronger, a finding that lent support to the conclusion that TCE exposure increased the risk of NHL. Since moderate heterogeneity (greater than in 2011) was identified for the overall set of studies, EPA additionally analyzed results from populations identified as receiving "high exposure" to TCE in order to parallel the analyses performed in the 2011 IRIS Assessment. Fixed- and random-effects models comparing the highest to lowest exposure groups in each study also weighted the (Vlaanderen et al., 2013) study heavily and produced meta-RRs of 1.03 (95% CI 0.93-1.15) and 1.33 (95% CI 0.98-1.80), respectively (Figure_Apx H-3 and Figure_Apx H-4). Extracted RR estimates and confidence intervals from each NHL study are presented in Table_Apx H-7, Table_Apx H-8, and Table_Apx H-9.

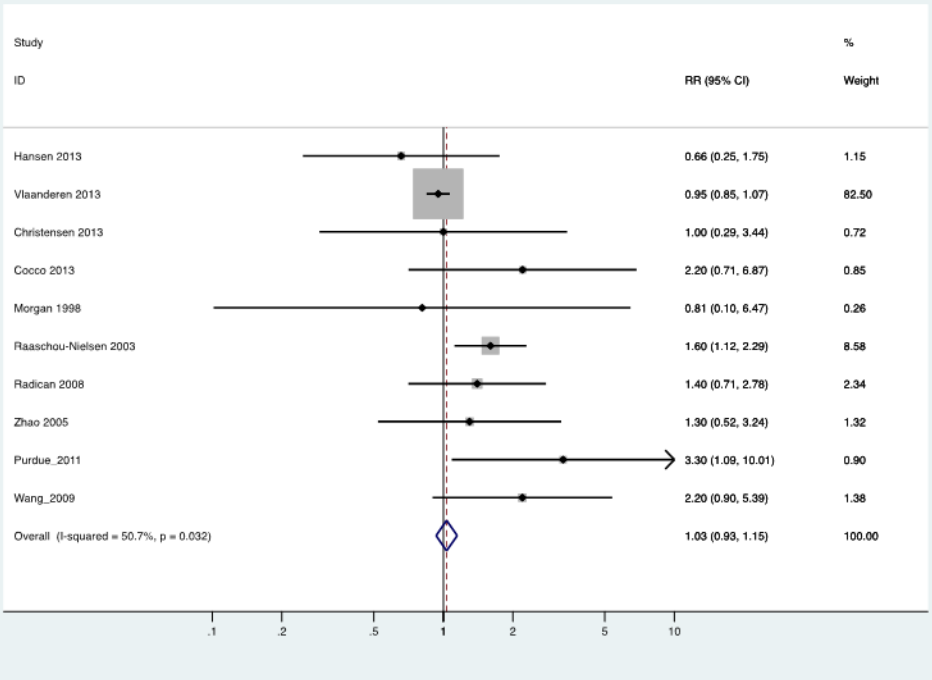
Figure_Apx H-1. Fixed-effects model, overall association of NHL and exposure to TCE.



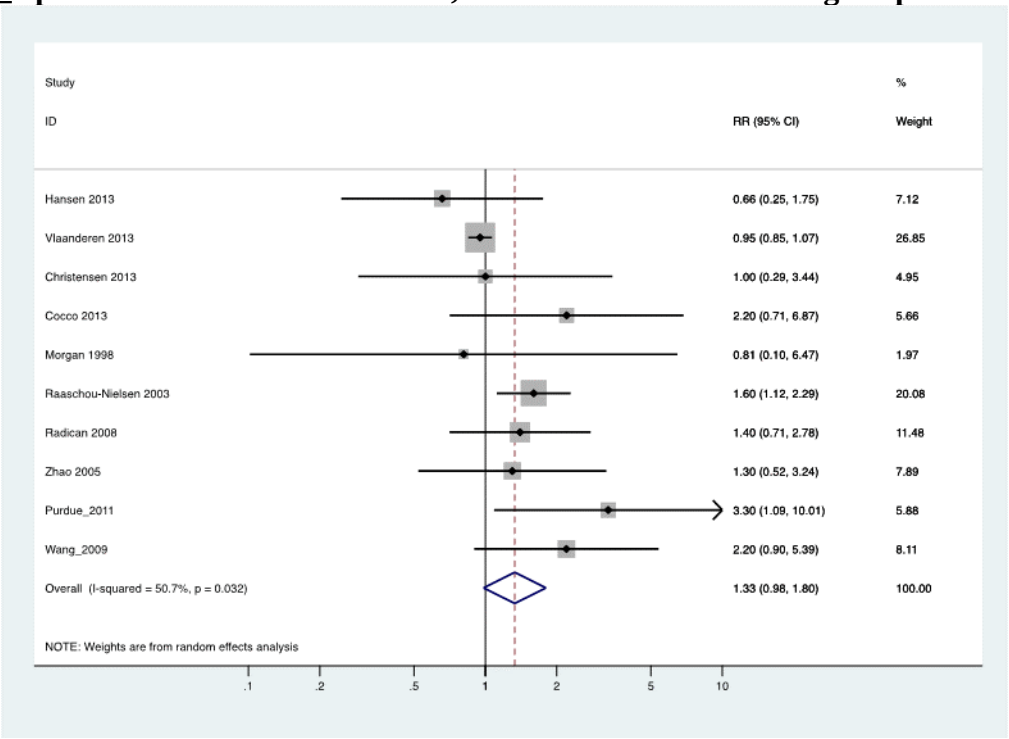
Figure_Apx H-2. Random-effects model, overall association of NHL and exposure to TCE.



Figure_Apx H-3. Fixed-effects model, association of NHL and high exposure to TCE.



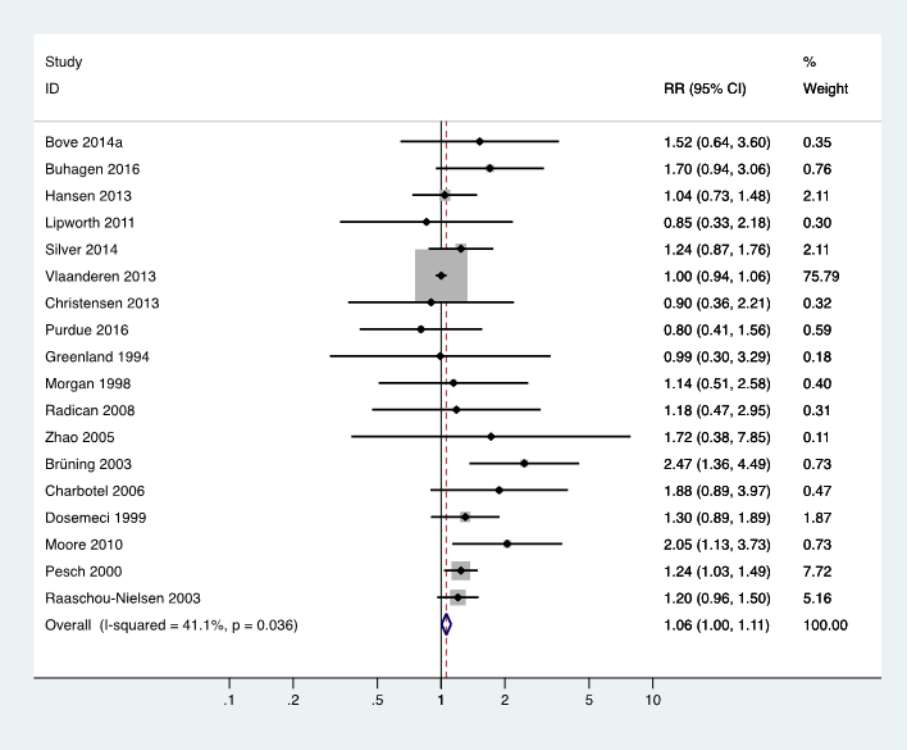
Figure_Apx H-4. Random-effects model, association of NHL and high exposure to TCE.



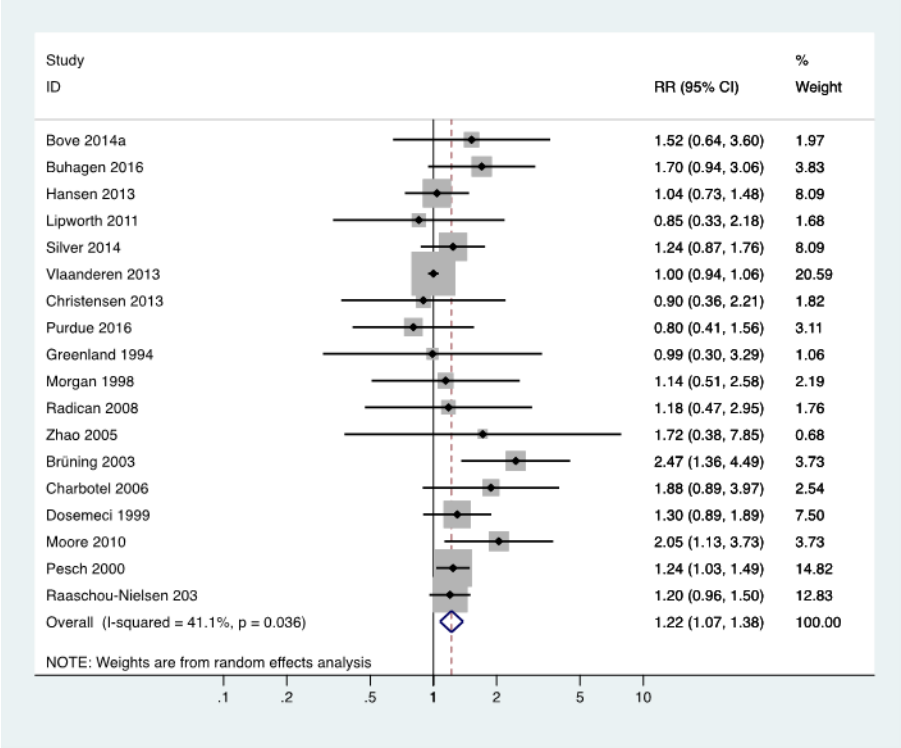
Kidney Cancer

For kidney cancer, the fixed effects model (Figure_Apx H-5) gave a meta-RR of 1.06 (95% CI 1.00-1.11) for overall exposure, with moderate, statistically-significant heterogeneity (I^2 41.1%, p 0.04). As for NHL, the study of (Vlaanderen et al., 2013) was heavily weighted. In the random-effects model (Figure_Apx H-6), the meta-RR was 1.22 (95% CI 1.07-1.38). Extracted RR estimates and confidence intervals from each kidney cancer study are presented in Table_Apx H-10 and Table_Apx H-11.

Figure_Apx H-5. Fixed-effects model, overall association of kidney cancer and exposure to TCE.



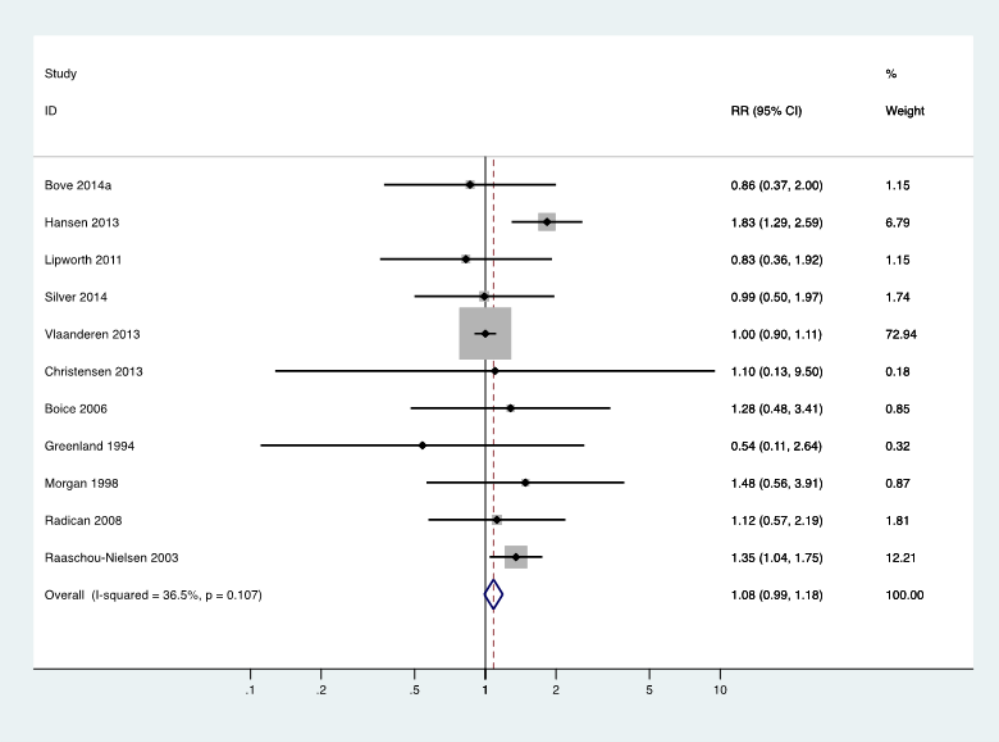
Figure_Apx H-6. Random-effects model, overall association of kidney cancer and exposure to TCE.



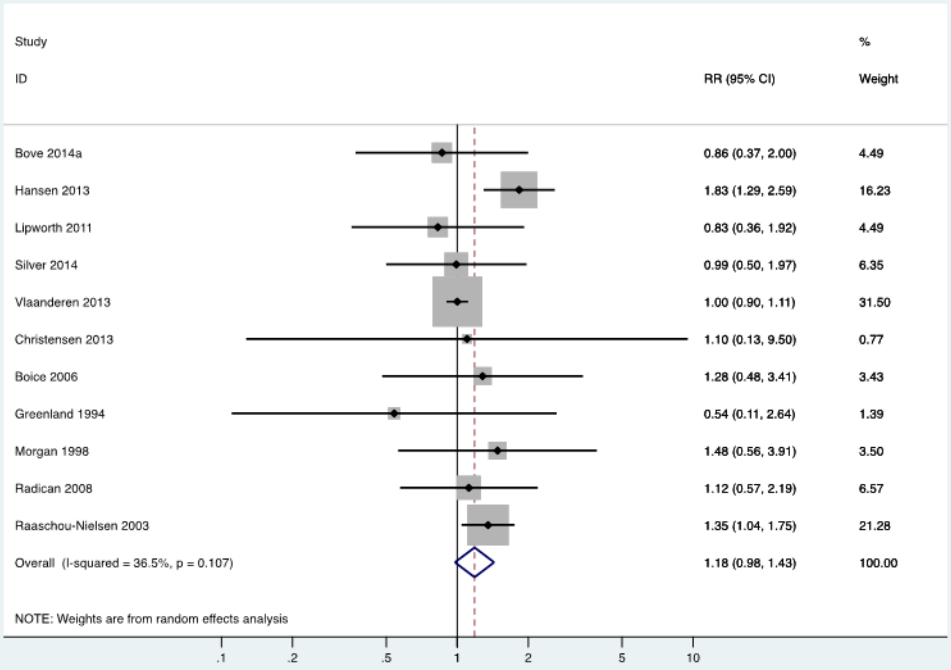
Liver cancer

Fixed- and random-effects models for liver cancer showed a similar pattern of results, with meta-RRs of 1.08 (95% CI 0.99-1.18) and 1.18 (95% CI 0.98-1.43), respectively (Figure_Apx H-7 and Figure_Apx H-8). Heterogeneity was moderate and not statistically significant (I^2 36.5%, p 0.107). Extracted RR estimates and confidence intervals from each liver cancer study are presented in Table_Apx H-12 and Table_Apx H-13.

Figure_Apx H-7. Fixed-effects model, overall association of liver cancer and exposure to TCE.



Figure_Apx H-8. Random-effects model, overall association of liver cancer and exposure to TCE.



H.2.2.2 Sensitivity analyses

Removal of Vlaanderen et al. (2013)

In analyses of influential observations, the study of (Vlaanderen et al., 2013) strongly influenced the meta-RRs for all three cancers (Table_Apx H-4, Table_Apx H-5, and Table_Apx H-6). No other single study had an appreciable impact on the overall association. Further meta-analyses were conducted to characterize the sensitivity of the results to the influence of that study.

Table_Apx H-4. Analysis of influential studies: NHL

Study omitted	Estimate	95% CI	
Bove et al. 2014a	1.02	0.97	1.08
Bove et al. 2014b	1.03	0.97	1.09
Hansen et al. 2013	1.02	0.96	1.08
Lipworth et al. 2011	1.02	0.97	1.09
Silver et al. 2014	1.03	0.97	1.09
Vlaanderen et al. 2013	1.20	1.07	1.34
Christensen et al. 2013	1.02	0.97	1.08
Cocco et al. 2013	1.02	0.96	1.08
Greenland et al. 1994	1.02	0.97	1.09
Morgan et al. 1998	1.02	0.97	1.09
Raaschou-Nielsen 2003	1.01	0.95	1.07
Radican et al. 2008	1.02	0.96	1.08
Zhao et al. 2005	1.02	0.96	1.08
Hardell et al. 1994	1.02	0.96	1.08
Nordstrom et al. 1998	1.02	0.96	1.08
Persson and Fredrikson 1999	1.02	0.97	1.08
Purdue et al. 2011	1.02	0.96	1.08
Wang et al. 2009	1.02	0.96	1.08

Table_Apx H-5. Analysis of influential studies: Kidney cancer

Study omitted	Estimate	95% CI	
Bove et al. 2014a	1.06	1.00	1.11
Buhagen et al. 2016	1.05	1.00	1.11
Hansen et al. 2013	1.06	1.00	1.11
Lipworth et al. 2011	1.06	1.01	1.11
Silver et al. 2014	1.05	1.00	1.11
Vlaanderen et al. 2013	1.26	1.14	1.40
Christensen et al. 2013	1.06	1.01	1.11
Purdue et al. 2016	1.06	1.01	1.12
Greenland et al. 1994	1.06	1.00	1.11
Morgan et al. 1998	1.06	1.00	1.11
Radican et al. 2008	1.06	1.00	1.11
Zhao et al. 2005	1.06	1.00	1.11
Brüning et al. 2003	1.05	1.00	1.11

Table_Apx H-5. Analysis of influential studies: Kidney cancer

Study omitted	Estimate	95% CI	
Charbotel et al. 2006	1.05	1.00	1.11
Dosemeci et al. 1999	1.05	1.00	1.11
Moore et al. 2010	1.05	1.00	1.11
Pesch et al. 2000	1.04	0.99	1.10
Raaschou-Nielsen et al. 2003	1.05	1.00	1.11

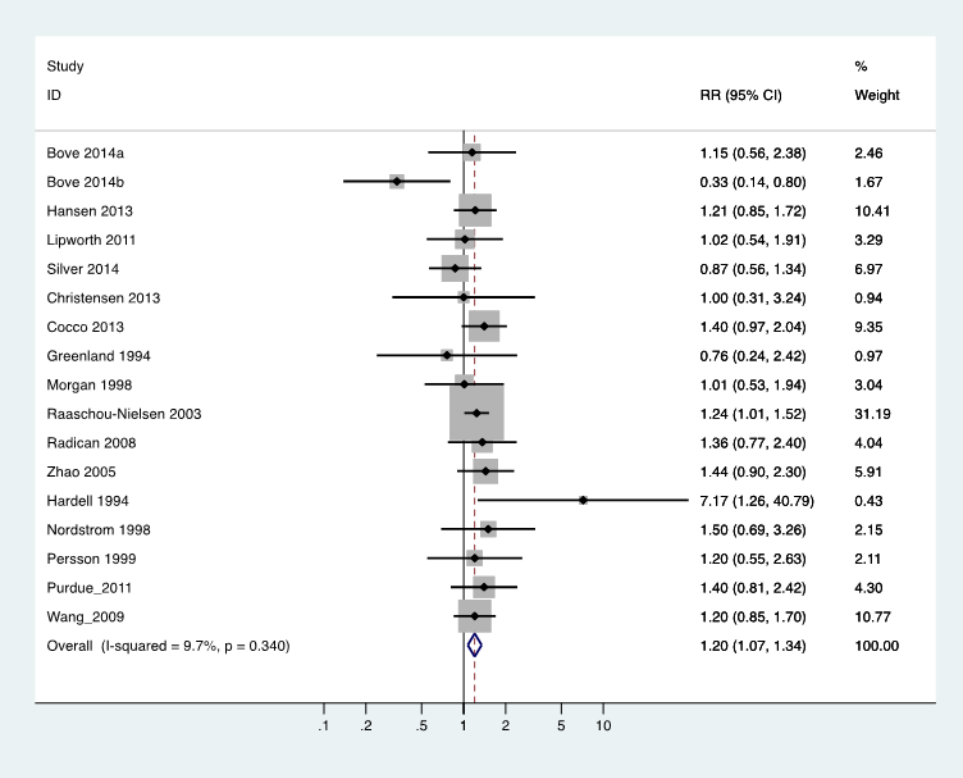
Table_Apx H-6. Analysis of influential studies: Liver cancer

Study omitted	Estimate	95% CI	
Bove et al. 2014a	1.09	0.99	1.19
Hansen et al. 2013	1.04	0.95	1.14
Lipworth et al. 2011	1.09	0.99	1.19
Silver et al. 2014	1.08	0.99	1.19
Vlaanderen et al. 2013	1.34	1.13	1.59
Christensen et al. 2013	1.08	0.99	1.18
Boice et al. 2006	1.08	0.99	1.18
Greenland et al. 1994	1.08	0.99	1.19
Morgan et al. 1998	1.08	0.99	1.18
Radican et al. 2008	1.08	0.99	1.19
Raaschou-Nielsen et al. 2003	1.05	0.95	1.16

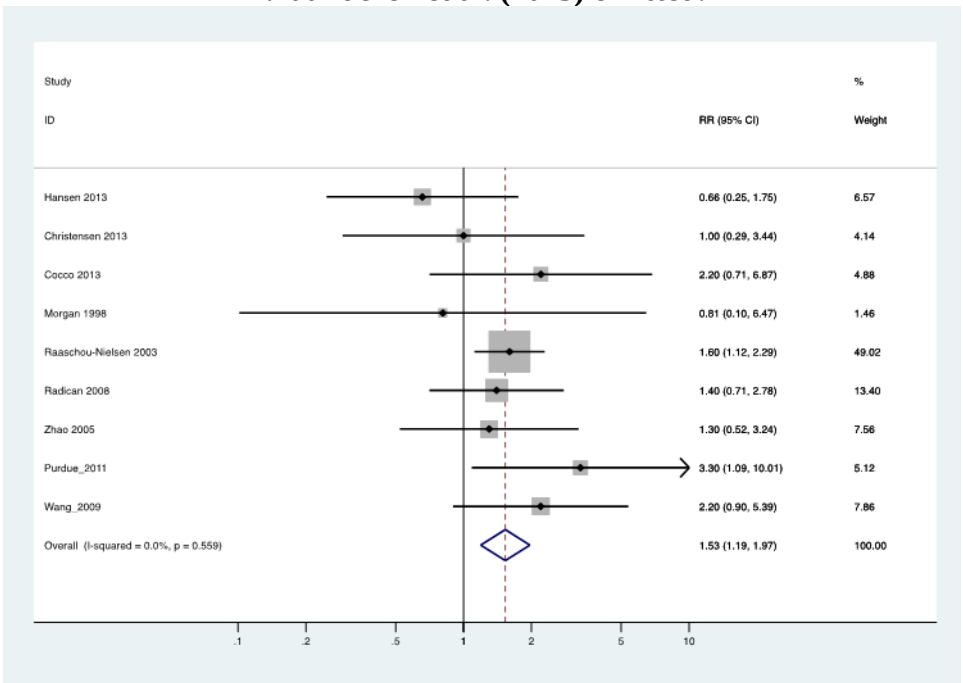
Meta-RRs for each cancer were re-estimated by omitting that study from the fixed-effects model. For NHL, omitting the study of (Vlaanderen et al., 2013) from the analysis of overall exposure to TCE (Figure_Apx H-9) substantially reduced between-study heterogeneity (I^2 9.7%, p 0.34) and yielded a meta-RR of 1.20 (95% CI 1.07-1.34). In the model for NHL using only the high exposure groups (Figure_Apx H-10), no heterogeneity remained when the (Vlaanderen et al., 2013) study was omitted (I^2 0.0%, p 0.56); the meta-RR for high exposure was 1.53 (95% CI 1.19-1.97). Omitting the study of (Vlaanderen et al., 2013) from the model for kidney cancer (Figure_Apx H-11), gave a meta-RR of 1.26 (95% CI 1.14-1.40) with no indication of heterogeneity (I^2 0.0%, p 0.57). Dropping that study from the analysis of liver cancer (

Figure_Apx **H-12**) similarly eliminated the heterogeneity among studies (I^2 0.0%, p 0.56) and gave a meta-RR of 1.34 (95% CI 1.13-1.59). Meta-RR values for all three tissues increased without the (Vlaanderen et al., 2013) study and achieved statistical significance.

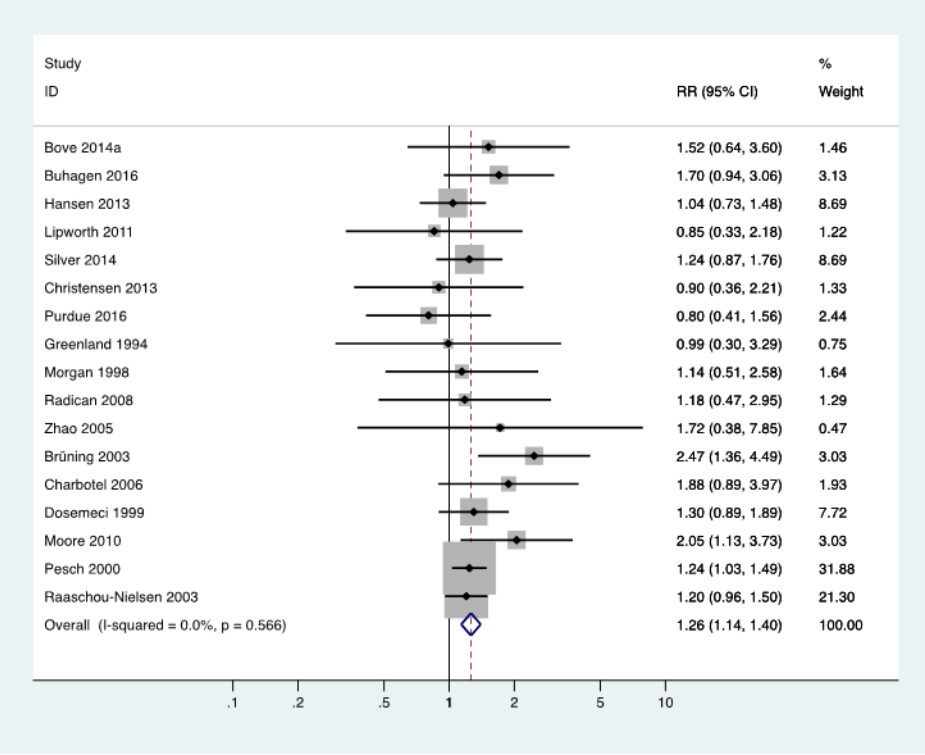
Figure_Apx H-9. Fixed-effects model, overall association of NHL and exposure to TCE, study of Vlaanderen et al. (2013) omitted.



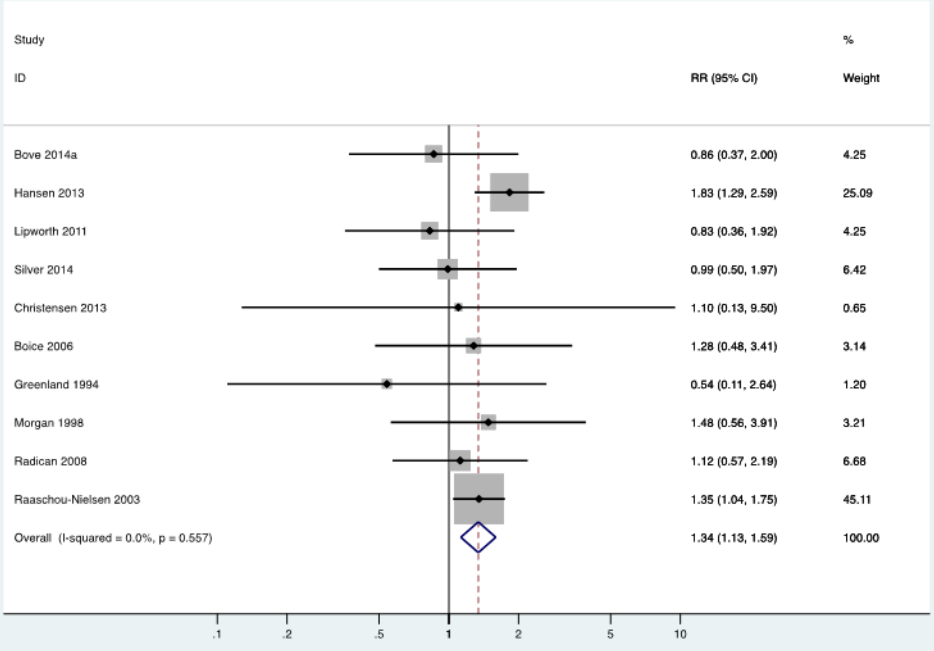
Figure_Apx H-10. Fixed-effects model, association of NHL and high exposure to TCE, study of Vlaanderen et al. (2013) omitted.



Figure_Apx H-11. Fixed-effects model, overall association of kidney cancer and exposure to TCE, study of Vlaanderen et al. (2013) omitted.



Figure_Apx H-12. Fixed-effects model, overall association of liver cancer and exposure to TCE, study of Vlaanderen et al. (2013) omitted.

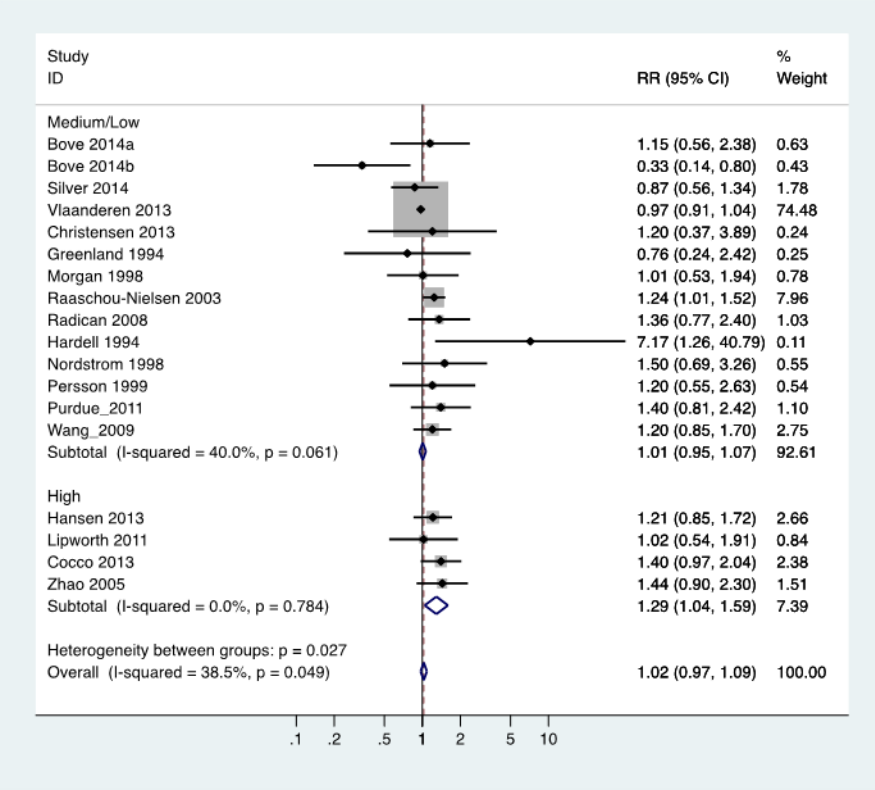


Stratification by Data Quality

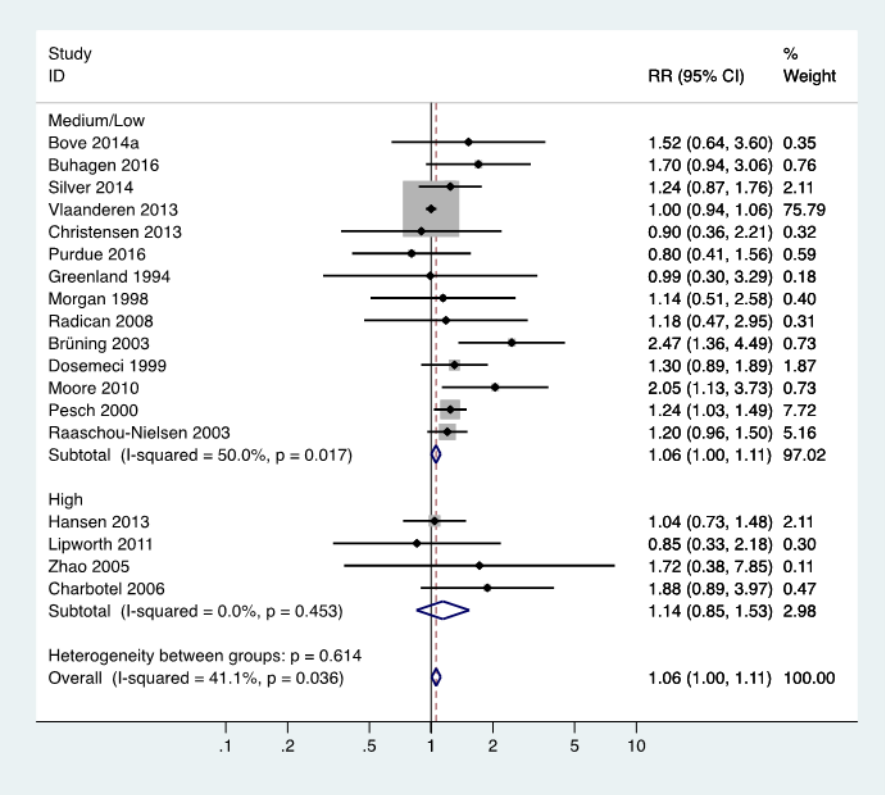
Fixed-effects meta-analyses for each cancer were also stratified by the study quality score assigned in EPA's review to assess whether the strength of association varied between highest- and lower-quality studies. In this manner, the meta-RR was compared among studies scoring High in data quality to those scoring Medium or Low. For NHL (Figure_Apx H-13), there was no heterogeneity among studies scored as high quality (I^2 0.0%, p 0.78) and the meta-RR was 1.29 (95% CI 1.04-1.59), while among studies scored medium or low the meta-RR was 1.01 (95% CI 0.95-1.07) with moderate heterogeneity (I^2 40.0%, p 0.06). Studies of kidney cancer (

Figure_**Apx H-14**) that scored high for data quality gave a meta-RR of 1.14 (95% CI 0.85-1.53) with no indicated heterogeneity (I^2 0.0% p 0.45), whereas lower-ranked studies gave a meta-RR of 1.06 (95% CI 1.00-1.11) with significant heterogeneity (I^2 50.0% p 0.02). In contrast, moderate, non-significant heterogeneity (I^2 36.0% p 0.21), remained among the three studies of liver cancer (Figure_Apx H-15) scored high for data quality; the meta-RR among those studies was 1.59 (95% CI 1.17-2.16). Lower scoring studies showed no heterogeneity (I^2 0.0% p 0.56) and a meta-RR of 1.04 (95% CI 0.95-1.15). Fitting a random-effects model reduced the meta-RR for highly scored studies to 1.42 (95% CI 0.88-2.30) but did not change the estimate for lower-scored studies. For all three tissues, the meta-RR was greater among the high quality studies compared to medium or low quality studies. Statistical significance was not always achieved due to the low number of studies scored High, however this stratification demonstrates stronger associations of cancer with TCE exposure among higher-quality data.

Figure_Apx H-13. Fixed-effects model, overall association of NHL and exposure to TCE stratified by study quality score.

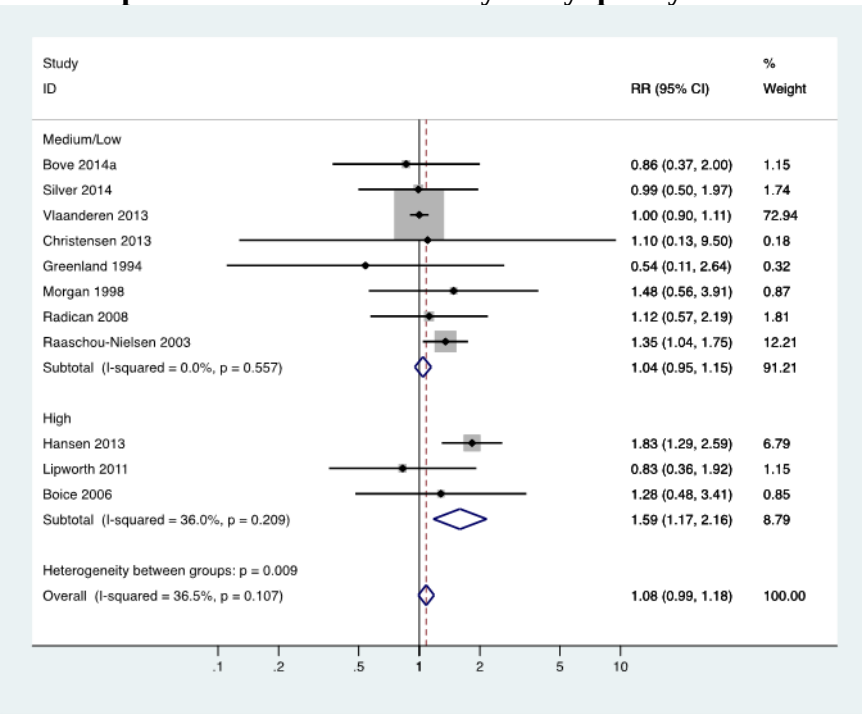


Figure_Apx H-14. Fixed-effects model, overall association of kidney cancer and exposure to TCE stratified by study quality score.



Figure_Apx H-15. Fixed-effects model, overall association of liver cancer and

exposure to TCE stratified by study quality score.



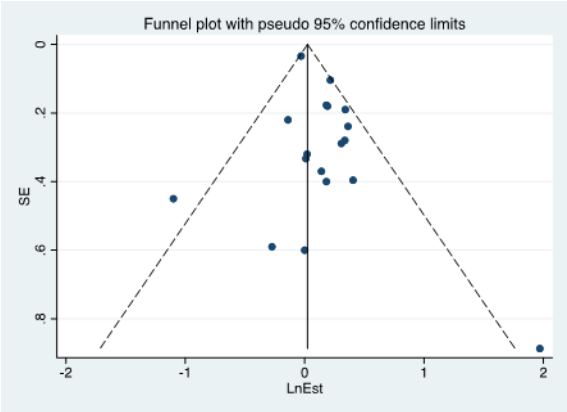
Assessment of Publication Bias

Funnel plots can be used to assess publication bias, a systematic error that occurs if statistically significant studies are more likely to be submitted and published than nonsignificant studies. One feature of publication bias is that smaller studies tend to have larger effect sizes than larger studies, since smaller studies need larger effect sizes in order to be statistically significant. To measure this, funnel plots plot standard error (SE) vs natural log of the RR (LnEst) to compare study size and effect size. If there is no relationship, the studies should be symmetrically distributed around the summary RR estimate (the vertical line), while publication bias is indicated by the points veering towards higher RR estimates with increasing SEs (i.e. toward the lower right).

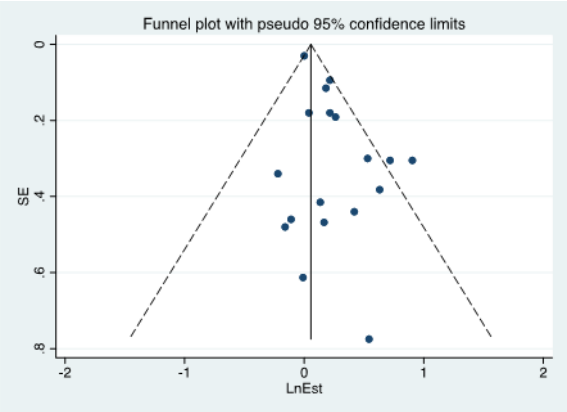
Funnel plots including all studies (Figure_Apx H-16, a-c) were consistent with modest publication bias, with a possible tendency toward omission of moderate-sized studies with weak or null associations. With the (Vlaanderen et al., 2013) study omitted, however, the plots became more symmetrical, consistent with an absence of publication bias among the remaining studies (Figure_Apx H-16, d-f).

Figure_Apx H-16. Funnel plots for publication bias.
All studies: a. NHL; b. kidney cancer; c. liver cancer;
Omitting Vlaanderen et al. (2013): d. NHL; e. kidney cancer; f. liver cancer.

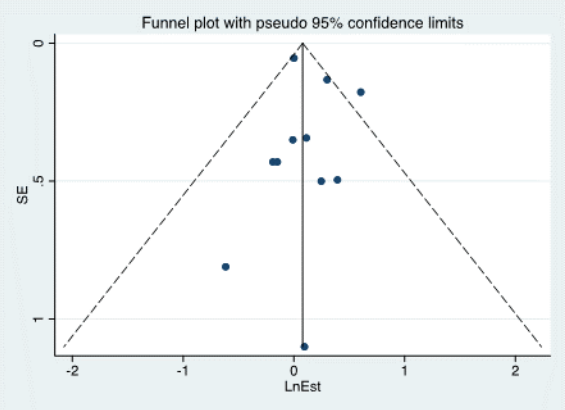
a.



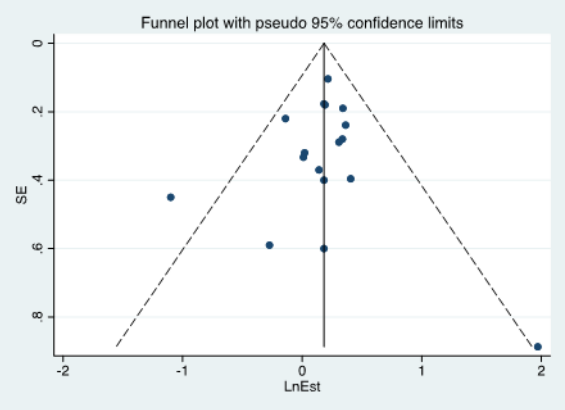
b.



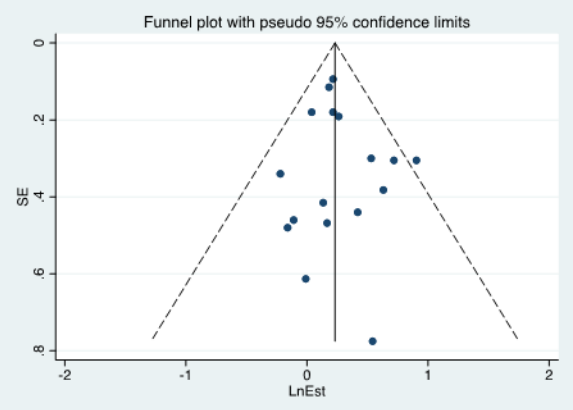
c.



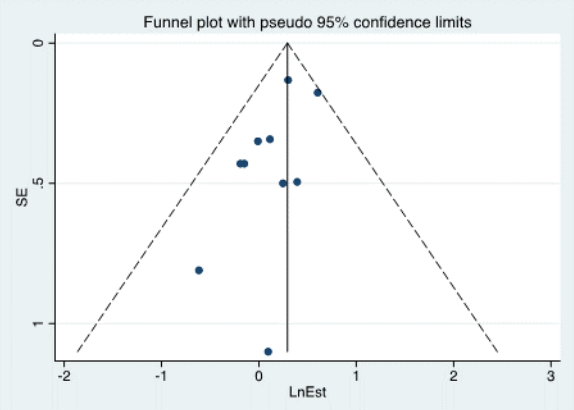
d.



e.



f.



H.2.3 Selected RR estimates and confidence intervals by study and cancer type

Table_Apx H-7. Selected RR estimates for NHL associated with TCE exposure (overall effect) from cohort studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Bove et al. (2014a) (2799547)	1.15	0.56	2.34	HR	0.140	0.37	None	Adjusted hazard ratio for males and females; cumulative exposure for high exposure in enlisted personnel; reference group had no exposure to TCE; 10-year lag time; specific ICD codes were not reported.
Bove et al. (2014b) (2800329)	0.32	0.05	2.10	HR	-1.1	0.45	None	Adjusted hazard ratio for males and females, Camp Lejeune cohort; cumulative exposure to TCE, >median vs <median (referent group); 10-year lag time; specific ICD codes not reported.
Hansen et al. (2013) (2128005)	1.21	0.83	1.71	SIR	0.191	0.18	1.11 (0.68-1.72) SIR for 20-year lag time; 1.26 (0.89-1.73) SIR for no lag	ICD-7 200 + 202; standard incidence ratio for males and females in three populations (Denmark, Sweden, and Finland); 10-year lag time; study also reports hazard rate ratios for NHL based on urinary TCE metabolite
Lipworth et al. (2011) (1235276)	1.02	0.55	1.90	RR	0.020	0.32	1.10 (0.59-2.04) RR for 1-4 yr exposure; 0.84 (0.48-1.47) RR for <1 yr exposure; 1.31 (0.97-1.73) SMR for routine and intermittent exposure for at least 1 yr (compared with general population)	ICD-9 200 + 202; relative risk for sex and race combined; ≥5 yr exposure in workers, routine and intermittent exposure; referent category was nonexposed factory workers
Silver et al (2014) (2799800)	0.87	0.57	1.35	HR	-0.14	0.22	None	Hazard ratio at 5 modified exposure years for males and females; cumulative exposure; adjusted for sex and paycode; 10-year lag time; specific ICD codes not reported.

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Vlaanderen et al (2013) 2128436	0.97	0.91	1.04	HR	-0.030	0.034	0.95 (0.84-1.06) HR for men and women; cumulative exposure for high exposure groups only (n=353 cases)	ICD-7 200 + 202; hazard ratio for men and women; third tertile of cumulative exposure (n=1211 cases); occupationally unexposed individuals were used as the reference group; unlagged exposure (up to 20 years of lag time had a negligible impact on HR)

Table_Apx H-8. Selected RR estimates for NHL associated with TCE exposure (overall effect) from case-control studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Christensen et al. (2013) (2127914)	1.2	0.5	2.9	0.18	0.45	1.0 (0.3–3.5) OR for substantial exposure	ICD-9 200 + 202; odds ratio for males and females; any exposure; adjusted by age, census tract median income, educational attainment (years), ethnicity, questionnaire respondent (self vs. proxy) and, smoking using population and cancer controls weighting proportionately
Cocco et al. (2013) (2129584)	1.4	0.9	2.1	0.34	0.22	1.0 (0.8-1.2); any vs no exposure in all subjects	Specific ICD codes not reported; odds ratio for males and females; all study subjects with high probability of exposure ; adjusted by age, gender, and contributing study (50 cases, 38 controls).

Table_Apx H-9. Selected RR estimates for NHL associated with TCE exposure (effect in the highest exposure group) studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	log RR	SE (log RR)	Alternate RR estimates (95% CI)	Comments
Cohort Studies							

Hansen et al. (2013) (2128005)	0.66	0.21	2.03	HRR	-0.42	0.50	None
Vlaanderen et al (2013) 2128436 Nested Case-control	0.95	0.84	1.06	HR	-0.051	0.059	0.96 (0.84-1.09) HR for men and women; intensity x prevalence for high exposure groups only (n=269 cases); occupationally unexposed individuals were used as the reference group; unlagged exposure
Case-Control Studies							
Christensen et al. (2013) (2127914)	1.0	0.3	3.5	0.00	0.63	NA	ICD-9 200 + 202; odds ratio for males and females; substantial exposure; adjusted by age, census tract median income, educational attainment (years), ethnicity, questionnaire respondent (self vs. proxy) and, smoking using population and cancer controls weighting proportionately.
Cocco et al. (2013) (2129584)	2.2	0.7	6.7	0.79	0.58	1.4 (1.0-2.1) OR for >150 ppm intensity level among all subjects.	Specific ICD codes were not reported; odds ratio for males and females; >75 ppm intensity level for study subjects with high probability of exposure (9 cases, 5 controls); adjusted by age, gender, and study.

Table_Apx H-10. Selected RR estimates for kidney cancer associated with TCE exposure (overall effect) from cohort studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Bove et al (2014a) (2799547)	1.52	0.64	3.61	HR	0.419	0.44	None	Adjusted hazard ratio for males and females; cumulative exposure for high exposure in enlisted personnel; reference group had no exposure to TCE; 10-year lag time
Buhagen et al (2016) 3502047	1.7	1.0	3.0	SIR	0.53	0.30	None	14 cases had confirmed occupational exposure to TCE.
Hansen et al. (2013) (2128005)	1.04	0.71	1.50	SIR	0.039	0.18	1.11 (0.67-1.73) SIR for 20-year lag time; 1.01 (0.70-1.42) SIR for no lag	Standard incidence ratio for males and females in three populations (Denmark, Sweden, and Finland); 10-year lag time; study also reports hazard rate ratios for kidney cancer based on urinary TCE metabolite

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Lipworth et al (2011) (1235276)	0.85	0.33	2.19	RR	-0.16	0.48	0.42 (0.13-1.42) RR for 1-4 yr exposure; 0.52 (0.21-1.30) RR for <1 yr exposure; 0.66 (0.38-1.07) SMR for routine and intermittent exposure for at least 1 yr (compared with general population)	Relative risk; sex and race combined; ≥5 yr exposure in workers, routine and intermittent exposure; referent category was nonexposed factory workers
Silver et al (2014) (2799800)	1.24	0.87	1.77	HR	0.215	0.18	None	Hazard ratio at 5 modified exposure years for males and females; cumulative exposure; adjusted for sex and paycode; 10-year lag time
Vlaanderen et al (2013) (2128436)	1.00	0.95	1.07	HR	0.00	0.030	0.86 (0.75-0.98) HR for men and women; cumulative exposure for high exposure groups only (n=251 cases)	Hazard ratio for males and females; third tertile of cumulative exposure (n=1372 cases); occupationally unexposed individuals were used as the reference group; unlagged exposure (up to 20 years of lag time had a negligible impact on HR)

Table_Apx H-11. Selected RR estimates for kidney cancer associated with TCE exposure (overall effect) from case-control studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	ln RR	SE (ln RR)	Alternate RR estimate (95% CI)	Comments
Christensen et al. (2013) (2127914)	0.9	0.4	2.4	-0.11	0.46	0.6 (0.1-2.8) OR for substantial exposure	Odds ratio for males and females; any exposure, adjusted by age, census tract median income, educational attainment (years), ethnicity, questionnaire respondent (self vs. proxy), smoking, and coffee, beer, wine, and spirit intake using population and cancer controls weighting proportionately

Purdue et al. (2016) (3482059)	0.8	0.4	1.5	-0.22	0.34	OR 0.9 (0.5 – 1.9) for third tertile of cumulative hours exposed, any exposure intensity (23 cases, 19 controls).	Odds ratio for kidney cancer in group with highest probability of exposure (≥90%; 32 cases, 32 controls); adjusted for age, sex, race, study center, education level, smoking status, BMI and history of hypertension

Table_Apx H-12. Selected RR estimates for liver cancer associated with TCE exposure (overall effect) from cohort studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Bove et al (2014a) (2799547)	0.86	0.37	1.97	HR	-0.15	0.43	None	Adjusted hazard ratio for males and females; cumulative exposure for high exposure in enlisted personnel; reference group had no exposure to TCE; 10-year lag time
Hansen et al. (2013) (2128005)	1.83	1.24	2.56	SIR	0.604	0.177	2.09 (1.34-3.11) SIR for 20-year lag time; 1.77 (1.24-2.45) SIR for no lag	Liver and biliary passages; standard incidence ratio for males and females in three populations (Denmark, Sweden, and Finland); 10-year lag time; study also reports hazard rate ratios for liver and biliary passages cancer based on urinary TCE metabolite
Lipworth et al (2011) (1235276)	0.83	0.36	1.91	RR	-0.19	0.43	0.69 (0.28-1.71) RR for 1-4 yr exposure; 0.67 (0.32-1.42) RR for <1 yr exposure 0.89 (0.57-1.33) SMR for routine and intermittent exposure for at least 1 yr (compared with general population)	Liver and biliary passages; relative risk; sex and race combined; ≥5 yr exposure in workers, routine and intermittent exposure; referent category was nonexposed factory workers
Silver et al (2014) (2799800)	0.99	0.50	1.95	HR	-0.010	0.35	None	Liver, biliary passages, and gallbladder; hazard ratio at 5 modified exposure years for males and females; cumulative exposure; adjusted for sex and paycode; 10-year lag time

Study	RR	95% LCL	95% UCL	RR type	ln RR	SE (ln RR)	Alternate RR estimates (95% CI)	Comments
Vlaanderen et al (2013) 2128436	1.00	0.90	1.11	HR	0.00	0.054	1.02 (0.82-1.25) HR for men and women; cumulative exposure for high exposure groups only (n=106 cases)	Hazard ratio for males and females; third tertile of cumulative exposure (n=422 cases); occupationally unexposed individuals were used as the reference group; unlagged exposure (up to 20 years of lag time had a negligible impact on HR)

Table_Apx H-13. Selected RR estimates for liver cancer associated with TCE exposure (overall effect) from case-control studies published after U.S. EPA (2011)

Study	RR	95% LCL	95% UCL	ln RR	SE (ln RR)	Alternate RR estimate (95% CI)	Comments
Christensen et al. (2013) (2127914)	1.1	0.1	8.5	0.095	1.1	2.1 (0.2-18) OR for substantial exposure	Odds ratio for males and females; any exposure, adjusted by age, census tract median income, educational attainment (years), ethnicity, questionnaire respondent (self vs. proxy), smoking, and beer, wine, and spirit intake using population and cancer controls weighting proportionately

H.2.4 Sample Stata commands for meta-analysis

Notes: the variables LnEst and SE are the natural log(RR) and its estimated standard error, respectively; Author_date labels studies on forest plots.

Basic fixed-effects analysis with axis labels:

```
metan LnEst SE, eform label(namevar=Author_date) effect(RR) xlabel(0.1, 0.2, 0.5, 1.0,
2.0,5.0,10)
```

Basic random-effects analysis with axis labels:

```
metan LnEst SE random, eform label(namevar=Author_date) effect(RR) xlabel(0.1, 0.2, 0.5, 1.0,
2.0,5.0,10)
```

Basic fixed-effects model omitting one study (indicated by NAME):

```
metan LnEst SE if Author!="NAME", eform label(namevar=Author_date) effect(RR) xlabel(0.1,
0.2, 0.5, 1.0, 2.0,5.0,10)
```

Fixed-effects model stratifying by quality score (HiQ):

```
metan LnEst SE, eform label(namevar=Author_date) effect(RR) xlabel(0.1, 0.2, 0.5, 1.0,
2.0,5.0,10) by(HiQ)
```

Basic “leave one out” analysis of influence:

```
metaninf LnEst SE, eform label(namevar=Author_date) effect(RR)
```

Basic funnel plot:

```
metafunnel LnEst SE
```

Appendix I APPROACH FOR ESTIMATING WATER RELEASES FROM MANUFACTURING SITES USING EFFLUENT GUIDELINES

This appendix presents a methodology for estimating water releases of TCE from manufacturing sites using effluent guidelines (EGs). This method uses the maximum daily and maximum average monthly concentrations allowed under the Organic Chemicals, Plastics and Synthetic Fibers (OCPSF) Effluent Guidelines and Standards (U.S. EPA). EGs are national regulatory standards set forth by EPA for wastewater discharges to surface water and municipal sewage treatment plants. The OCPSF EG applies to facilities classified under the following SIC codes:

- 2821—Plastic Materials, Synthetic Resins, and Nonvulcanizable Elastomers;
- 2823—Cellulosic Man-Made Fibers;
- 2865—Cyclic Crudes and Intermediates, Dyes, and Organic Pigments; and
- 2869—Industrial Organic Chemicals, Not Elsewhere Classified.

Manufacturers of TCE would typically be classified under SIC code 2869; therefore, the requirements of the OCPSF EG are assumed to apply to manufacturing sites. Subparts I, J, and K of the OCPSF EG set limits for the concentration of TCE in wastewater effluent for industrial facilities that are direct discharge point sources using end-of-pipe biological treatment, direct discharge point sources that do not use end-of-pipe biological treatment, and indirect discharge point sources, respectively (U.S. EPA, 2019c). Direct dischargers are facilities that discharge effluent directly to surface waters and indirect dischargers are facilities that discharge effluent to publicly-owned treatment works (POTW). The OCPSF limits for TCE in each of the Subparts are provided in Table_Apx I-1.

Table_Apx I-1. Summary of OCPSF Effluent Guidelines for Trichloroethylene

OCPSF Subpart	Maximum for Any One Day (µg/L)	Maximum for Any Monthly Average (µg/L)	Basis
Subpart I – Direct Discharge Point Sources That Use End-of-Pipe Biological Treatment	54	21	BAT effluent limitations and NSPS
Subpart J – Direct Discharge Point Sources That Do Not Use End-of-Pipe Biological Treatment	69	26	BAT effluent limitations and NSPS
Subpart K – Indirect Discharge Point Sources	69	26	Pretreatment Standards for Existing Sources (PSES) and Pretreatment Standards for New Sources (PSNS)

BAT = Best Available Technology Economically Achievable; NSPS = New Source Performance Standards; PSES = Pretreatment Standards for Existing Sources; PSNS = Pretreatment Standards for New Sources.
Source: (U.S. EPA)

To estimate daily releases from the EG, EPA used Equation I-1 to estimate daily releases and Equation D-2 to estimate annual releases using the parameters in Table_Apx I-2. The prevalence of end-of-pipe biological treatment is unknown; therefore, EPA used the discharge limits for direct discharge point sources that do not use end-of-pipe biological treatment (Subpart J) and indirect discharge point sources (Subpart K). EPA estimated a central tendency daily release using the limit for the maximum monthly average (26 µg/L) from Subparts J and K, a high-end daily release using the limit for the maximum for any one day (69 µg/L) from Subparts J and K, and an annual release using the maximum monthly average from Subparts J and K.

Equation I-1

$$DR = \frac{DL \times PW \times PV}{1{,}000{,}000{,}000 \times OD}$$

Equation I-2

$$AR = \frac{DL \times PW \times PV}{1{,}000{,}000{,}000}$$

Table_Apx I-2. Default Parameters for Estimating Water Releases of Trichloroethylene from Manufacturing Sites

Parameter	Parameter Description	Default Value	Unit
DR	Daily release rate	Calculated from equation	kg/site-day
DL	Discharge limit[a]	Max Daily: 69 Average Daily: 26 Annual: 26	µg/L
PW	Produced water[b]	10	L/kg
PV	Annual TCE production volume	Site-specific	kg/site-yr
OD	Operating Days[c]	350	days/yr
AR	Annual release rate	Calculated from equation	kg/site-yr

[a] Discharge limits are based on the maximum discharge limits allowed in the OCPSF EG, which correspond to the discharge limits for direct discharge point sources with no biological end-of-pipe treatment (Subpart J) and indirect discharge points sources (Subpart K) (citation for 40 C.F.R. 414). There is no "average" daily discharge limit set by the EGs; therefore, EPA assumed that the average daily discharge concentration would be equal to the maximum monthly average discharge limit.
[b] The amount of produced water per kilogram of TCE produced is based on the SpERC developed by the European Solvent Industry Group for the manufacture of a substance, which estimates 10 m^3 of wastewater generated per metric ton of substance produced and converted to 10 L/kg (European Solvents Industry Group (ESIG), 2012).
[c] Due to large throughput, manufacturing sites are assumed to operate seven days per week and 50 weeks per year with two weeks per year for shutdown activities.

EPA did not identify TCE-specific information on the amount of wastewater produced per day. The Specific Environmental Release Category (SpERC) developed by the European Solvent Industry Group for the manufacture of a substance estimates 10 m^3 of wastewater generated per metric ton of substance produced (equivalent to 10 L water/kg of substance produced) (European Solvents Industry Group (ESIG), 2012). In lieu of TCE-specific information, EPA estimated wastewater flow using the SpERC specified wastewater production volume and the annual TCE production rates for each facility. Table_Apx I-3 provides estimated daily production volume and wastewater flow for each facility that EPA used the EG to assess water releases.

Table_Apx I-3. Summary of Facility Trichloroethylene Production Volumes and Wastewater Flow Rates

Site	Annual Production Volume (kg/site-yr)	Annual Operating Days (days/yr)	Daily Production Volume (kg/site-day)	Daily Wastewater Flow (L/site-day)
Solvents & Chemicals, Pearland, TX[a]	20,382,094	350	58,234	582,345
Occidental Chemical Corp. Wichata, KS[a]	20,382,094	350	58,234	582,345

[a] The 2015 annual production volumes in the 2016 CDR for these sites was either claimed as CBI or withheld. EPA estimate the production volume by subtracting known site production volumes from the national production volume and averaging the result over all the sites with CBI or withheld production volumes and converting from pounds to kilograms.

[b] Annual production volume for this site is based on the 2015 production volume reported in the 2016 CDR and converting from pounds to kilograms.

EPA estimated both a maximum daily release and an average daily release using the OCPSF EG limits for TCE for maximum on any one day and maximum for any monthly average, respectively. Prevalence of end-of-pipe biological treatment at TCE manufacturing sites is unknown; therefore, EPA used limits for direct discharges with no end-of-pipe biological treatment and indirect dischargers as conservative. EPA estimated annual releases from the average daily release and assuming 350 days/yr of operation.

Example max daily, average daily, and annual water release calculations for TCE at manufacturing sites based on the estimated production volume for Solvents & Chemicals (44,934,862 lbs/yr or 20,382,094 kg/yr):[23]

$$Max\ DR = \frac{69\frac{\mu g}{L} \times 10\frac{L}{kg} \times 20{,}382{,}094\frac{kg}{yr}}{1{,}000{,}000{,}000\frac{\mu g}{kg} \times 350\frac{days}{yr}} = 0.04\frac{kg}{day}$$

[23] This estimated production volume is equal to the estimated production volume assessed for all manufacturing sites.

$$Average\ DR = \frac{26\frac{\mu g}{L} \times 10\frac{L}{kg} \times 20{,}382{,}094\frac{kg}{yr}}{1{,}000{,}000{,}000\frac{\mu g}{kg} \times 350\frac{days}{yr}} = 0.015\ \frac{kg}{day}$$

$$AR = \frac{26\frac{\mu g}{L} \times 10\frac{L}{kg} \times 20{,}382{,}094\frac{kg}{yr}}{1{,}000{,}000{,}000\frac{\mu g}{kg}} = 5.3\frac{kg}{yr}$$

Appendix J SAMPLE CALCULATIONS FOR CALCULATING ACUTE AND CHRONIC (NON-CANCER AND CANCER) INHALATION EXPOSURE

Sample calculations for high-end and central tendency acute and chronic exposure concentrations for one setting, Manufacturing, are demonstrated below. The explanation of the equations and parameters used is provided in [*Environmental Releases and Occupational Exposure Assessment. Docket: EPA-HQ-OPPT-2019-0500*]. The final values will have two significant figures since they are based on values from modeling.

J.1 Example High-End AC, ADC, and LADC

Calculate AC_{HE}:

$$AC_{HE} = \frac{C_{HE} \times ED}{AT_{acute}}$$

$$AC_{HE} = \frac{2.6\ ppm \times 8\ hr/day}{24\ hr/day} = 0.87\ ppm$$

Calculate ADC_{HE}:

$$\boldsymbol{ADC_{HE} = \frac{C_{HE} \times ED \times EF \times EWY}{AT}}$$

$$\boldsymbol{ADC_{HE} = \frac{2.6\ ppm \times 8\frac{hr}{day} \times 250\frac{days}{year} \times 40\ years}{\left(40\ years \times 365\frac{days}{year} \times 24\frac{hours}{day}\right)} = 0.59\ ppm}$$

Calculate $LADC_{HE}$:

$$\boldsymbol{LADC_{HE} = \frac{C_{HE} \times ED \times EF \times EWY}{AT_{LADC}}}$$

$$\boldsymbol{LADC_{HE} = \frac{2.6\ ppm \times 8\frac{hr}{day} \times 250\frac{days}{year} \times 40\ years}{\left(78\ years \times 365\frac{days}{year} \times 24\frac{hours}{day}\right)} = 0.30\ ppm}$$

J.2 Example Central Tendency AEC, ADC, and LADC

Calculate AC_{CT}:

$$AC_{CT} = \frac{C_{CT} \times ED}{AT_{acute}}$$

$$AC_{CT} = \frac{0.03\ ppm \times 8\ hr/day}{24\ hr/day} = 0.01\ ppm$$

Calculate ADC_{CT}:

$$ADC_{CT} = \frac{C_{CT} \times ED \times EF \times WY}{AT}$$

$$ADC_{CT} = \frac{0.03\ ppm \times 8\frac{hr}{day} \times 250\frac{days}{year} \times 31\ years}{31\ years \times 365\frac{days}{yr} \times 24\frac{hr}{day}} = 0.01\ ppm$$

Calculate $LADC_{CT}$:

$$LADC_{CT} = \frac{C_{CT} \times ED \times EF \times WY}{AT_c}$$

$$\boldsymbol{LADC_{CT} = \frac{0.03\ ppm \times 8\frac{hr}{day} \times 250\frac{days}{year} \times 31\ years}{78\ years \times 365\frac{days}{year} \times 24\ hr/day} = 2.8 \times 10^{-3}\ ppm}$$

Appendix K VAPOR DEGREASING AND COLD CLEANING NEAR-FIELD/FAR-FIELD INHALATION EXPOSURE MODELS APPROACH AND PARAMETERS

This appendix presents the modeling approach and model equations used in the following models:

- Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model;
- Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model;
- Web Degreasing Near-Field/Far-Field Inhalation Exposure Model; and
- Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model.

The models were developed through review of the literature and consideration of existing EPA exposure models. These models use a near-field/far-field approach (Nicas, 2009), where a vapor generation source located inside the near-field diffuses into the surrounding environment. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field.

The model uses the following parameters to estimate exposure concentrations in the near-field and far-field:

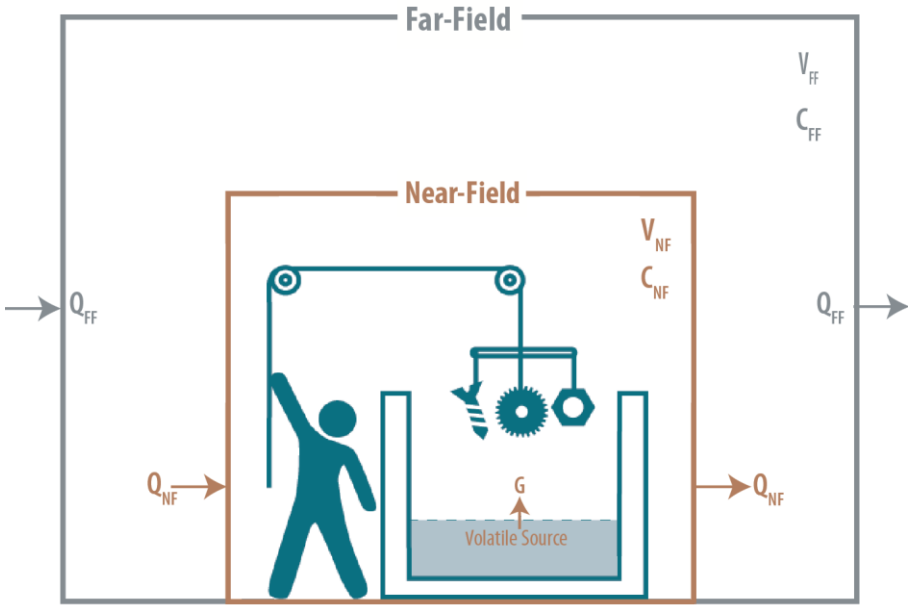
- Far-field size;
- Near-field size;
- Air exchange rate;
- Indoor air speed;
- Exposure duration;
- Vapor generation rate; and
- Operating hours per day.

An individual model input parameter could either have a discrete value or a distribution of values. EPA assigned statistical distributions based on reasonably available literature data. A Monte Carlo simulation (a type of stochastic simulation) was conducted to capture variability in the model input parameters. The simulation was conducted using the Latin hypercube sampling method in @Risk Industrial Edition, Version 7.0.0. The Latin hypercube sampling method is a statistical method for generating a sample of possible values from a multi-dimensional distribution. Latin hypercube sampling is a stratified method, meaning it guarantees that its generated samples are representative of the probability density function (variability) defined in the model. EPA performed the model at 100,000 iterations to capture the range of possible input values (i.e., including values with low probability of occurrence).

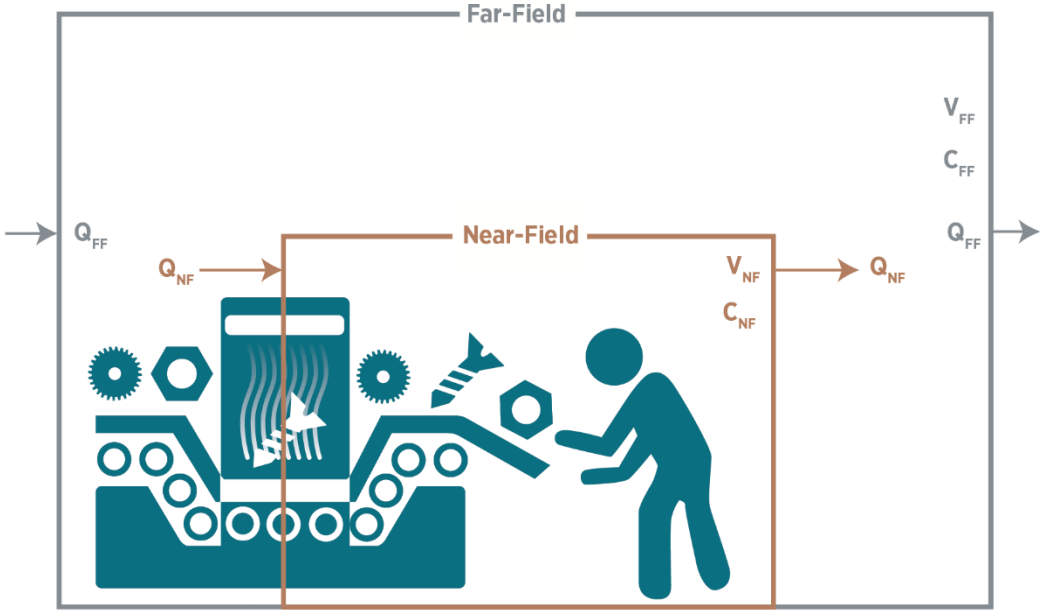
Model results from the Monte Carlo simulation are presented as 95^{th} and 50^{th} percentile values. The statistics were calculated directly in @Risk. The 95^{th} percentile value was selected to represent high-end exposure level, whereas the 50^{th} percentile value was selected to represent typical exposure level. The following subsections detail the model design equations and parameters for vapor degreasing and cold cleaning models.

K.1 Model Design Equations

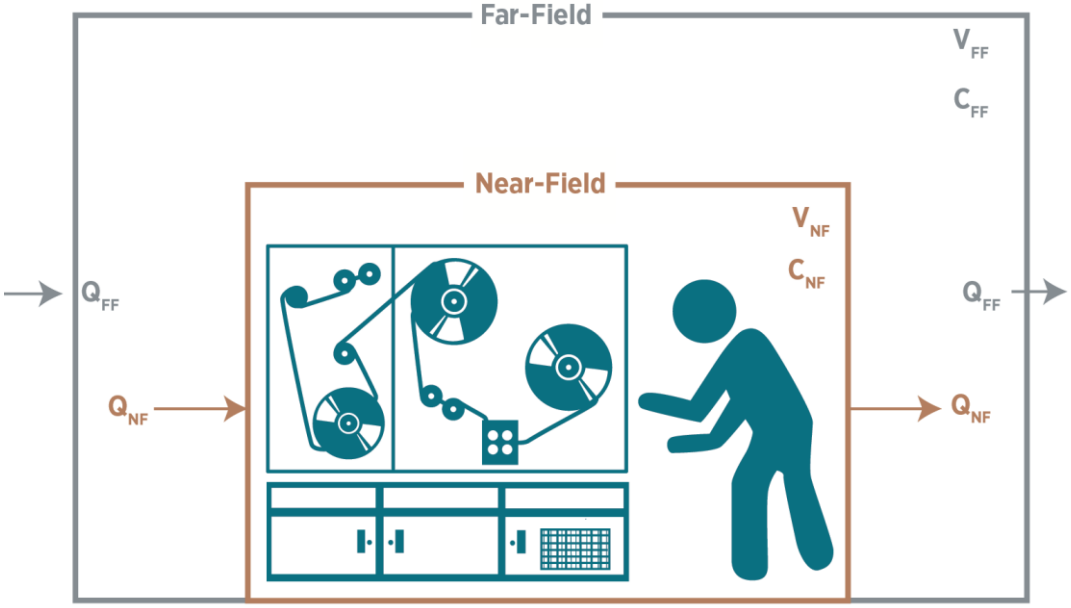
Figure_Apx K-1 through Figure_Apx K-3 illustrate the near-field/far-field modeling approach as it was applied by EPA to each vapor degreasing and cold cleaning model. As the figures show, volatile TCE vapors evaporate into the near-field, resulting in worker exposures at a TCE concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, (denoted by "G" in Figure 2-7), into the near-field, whose volume is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field, resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outside air.



Figure_Apx K-1. The Near-Field/Far-Field Model as Applied to the Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model and the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model



Figure_Apx K-2. The Near-Field/Far-Field Model as Applied to the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model



Figure_Apx K-3. The Near-Field/Far-Field Model as Applied to the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model

The model design equations are presented below in Equation K-1 through Equation K-18. Note the design equations are the same for each of the models discussed in this appendix.

Near-Field Mass Balance

Equation K-1

$$V_{NF}\frac{dC_{NF}}{dt} = C_{FF}Q_{NF} - C_{NF}Q_{NF} + G$$

Far-Field Mass Balance

Equation K-2

$$V_{FF}\frac{dC_{FF}}{dt} = C_{NF}Q_{NF} - C_{FF}Q_{NF} - C_{FF}Q_{FF}$$

Where:

V_{NF} = near-field volume;
V_{FF} = far-field volume;
Q_{NF} = near-field ventilation rate;
Q_{FF} = far-field ventilation rate;
C_{NF} = average near-field concentration;
C_{FF} = average far-field concentration;
G = average vapor generation rate; and
t = elapsed time.

Both of the previous equations can be solved for the time-varying concentrations in the near-field and far-field as follows (Nicas, 2009):

Equation K-3

$$C_{NF} = G\left(k_1 + k_2 e^{\lambda_1 t} - k_3 e^{\lambda_2 t}\right)$$

Equation K-4

$$C_{FF} = G\left(\frac{1}{Q_{FF}} + k_4 e^{\lambda_1 t} - k_5 e^{\lambda_2 t}\right)$$

Where:

Equation K-5

$$k_1 = \frac{1}{\left(\frac{Q_{NF}}{Q_{NF}+Q_{FF}}\right)Q_{FF}}$$

Equation K-6

$$k_2 = \frac{Q_{NF}Q_{FF} + \lambda_2 V_{NF}(Q_{NF}+Q_{FF})}{Q_{NF}Q_{FF}V_{NF}(\lambda_1 - \lambda_2)}$$

Equation K-7

$$k_3 = \frac{Q_{NF}Q_{FF} + \lambda_1 V_{NF}(Q_{NF}+Q_{FF})}{Q_{NF}Q_{FF}V_{NF}(\lambda_1 - \lambda_2)}$$

Equation K-8

$$k_4 = \left(\frac{\lambda_1 V_{NF} + Q_{NF}}{Q_{NF}}\right)k_2$$

Equation K-9

$$k_5 = \left(\frac{\lambda_2 V_{NF} + Q_{NF}}{Q_{NF}}\right)k_3$$

Equation K-10

$$\lambda_1 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) + \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

Equation K-11

$$\lambda_2 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) - \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

EPA calculated the hourly TWA concentrations in the near-field and far-field using Equation M-1221 and Equation M-13, respectively. Note that the numerator and denominator of Equation M-1221 and Equation M-132 use two different sets of time parameters. The numerator is based on operating times for the scenario (e.g., two or eight hours for OTVDs, 8 to 24 hours for conveyorized degreasers, 8 hours for web degreasers, and 3 to 8 hours for cold cleaning, see Appendix M.2) while the denominator is fixed to an average time span, t_avg, of eight hours (since EPA is interested in calculating 8-hr TWA exposures). Mathematically, the numerator and denominator must reflect the same amount of time. This is indeed the case since the numerator assumes exposures are zero for any hours not within the operating time. Therefore, mathematically speaking, both the numerator and the denominator reflect eight hours regardless of the values selected for t_1 and t_2.

Equation K-12

$$C_{NF,TWA} = \frac{\int_{t_1}^{t_2} C_{NF}dt}{\int_0^{t_{avg}} dt} = \frac{\int_{t_1}^{t_2} G\left(k_1 + k_2 e^{\lambda_1 t} - k_3 e^{\lambda_2 t}\right)dt}{t_{avg}} =$$

$$\frac{G\left(k_1 t_2 + \frac{k_2 e^{\lambda_1 t_2}}{\lambda_1} - \frac{k_3 e^{\lambda_2 t_2}}{\lambda_2}\right) - G\left(k_1 t_1 + \frac{k_2 e^{\lambda_1 t_1}}{\lambda_1} - \frac{k_3 e^{\lambda_2 t_1}}{\lambda_2}\right)}{t_{avg}}$$

Equation K-13

$$C_{FF,TWA} = \frac{\int_{t_1}^{t_2} C_{FF}dt}{\int_0^{t_{avg}} dt} = \frac{\int_{t_1}^{t_2} G\left(\frac{1}{Q_{FF}} + k_4 e^{\lambda_1 t} - k_5 e^{\lambda_2 t}\right)dt}{t_{avg}} =$$

$$\frac{G\left(\frac{t_2}{Q_{FF}} + \frac{k_4 e^{\lambda_1 t_2}}{\lambda_1} - \frac{k_5 e^{\lambda_2 t_2}}{\lambda_2}\right) - G\left(\frac{t_1}{Q_{FF}} + \frac{k_4 e^{\lambda_1 t_1}}{\lambda_1} - \frac{k_5 e^{\lambda_2 t_1}}{\lambda_2}\right)}{t_{avg}}$$

To calculate the mass transfer to and from the near-field, the free surface area, FSA, is defined to be the surface area through which mass transfer can occur. Note that the FSA is not equal to the surface area of the entire near-field. EPA defined the near-field zone to be a rectangular box resting on the floor; therefore, no mass transfer can occur through the near-field box's floor. FSA is calculated in Equation M-23, below:

Equation K-14

$$FSA = 2(L_{NF}H_{NF}) + 2(W_{NF}H_{NF}) + (L_{NF}W_{NF})$$

Where: L_{NF}, W_{NF}, and H_{NF} are the length, width, and height of the near-field, respectively. The near-field ventilation rate, Q_{NF}, is calculated in Equation M-154 from the near-field indoor wind speed, v_{NF}, and FSA, assuming half of FSA is available for mass transfer into the near-field and half of FSA is available for mass transfer out of the near-field:

Equation K-15

$$Q_{NF} = \frac{1}{2} v_{NF} FSA$$

The far-field volume, V_{FF}, and the air exchange rate, AER, is used to calculate the far-field ventilation rate, Q_{FF}, as given by Equation M-25:

Equation K-16

$$Q_{FF} = V_{FF} AER$$

Using the model inputs described in Appendix E.2, EPA estimated TCE inhalation exposures for workers in the near-field and for occupational non-users in the far-field. EPA then conducted the Monte Carlo simulations using @Risk (Version 7.0.0). The simulations applied 100,000 iterations and the Latin Hypercube sampling method for each model.

K.2 Model Parameters

Table_Apx K-1 through Table_Apx K-4 summarize the model parameters and their values for each of the models discussed in this Appendix. Each parameter is discussed in detail in the following subsections.

Table_Apx K-1. Summary of Parameter Values and Distributions Used in the Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Deterministic Values		Uncertainty Analysis Distribution Parameters				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Far-field volume	V_{FF}	ft^3	10,594	Midpoint	10,594	70,629	17,657	Triangular	See Section K.2.1
Air exchange rate	AER	hr^{-1}	2	Mode	2	20	3.5	Triangular	See Section K.2.2
Near-field indoor wind speed	v_{NF}	ft/hr	1,181	50th percentile	154	23,882	—	—	See Section K.2.3
		cm/s	10	50th percentile	1.3	202.2	—	—	
Near-field length	L_{NF}	ft	10	—	—	—	—	Constant Value	See Section K.2.4
Near-field width	W_{NF}	ft	10	—	—	—	—	Constant Value	
Near-field height	H_{NF}	ft	6	—	—	—	—	Constant Value	
Starting time	t_1	hr	0	—	—	—	—	Constant Value	Constant.
Exposure Duration	t_2	hr	8	—	2	8	—	--	See Section K.2.5
Averaging Time	t_{avg}	hr	8	—	—	—	—	Constant Value	See Section K.2.6
Vapor generation rate	G	mg/hr	2.34E+07	Average	4.54E+02	4.67E+07	—	Discrete	See Section K.2.7
		lb/hr	51.50	Average	0.001	103.00	—	Discrete	
Operating hours per day	OH	hr/day	8	—			—	Discrete	See Section E.2.8

Table_Apx K-2. Summary of Parameter Values and Distributions Used in the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Deterministic Values		Uncertainty Analysis Distribution Parameters				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Far-field volume	V_{FF}	ft^3	10,594	Midpoint	10,594	70,629	17,657	Triangular	See Section K.2.1
Air exchange rate	AER	hr^{-1}	2	Mode	2	20	3.5	Triangular	See Section K.2.2
Near-field indoor wind speed	v_{NF}	ft/hr	1,181	50th percentile	154	23,882	—	—	See Section K.2.3
		cm/s	10	50th percentile	1.3	202.2	—	—	
Near-field length	L_{NF}	ft	10	—	—	—	—	Constant Value	See Section K.2.4
Near-field width	W_{NF}	ft	10	—	—	—	—	Constant Value	
Near-field height	H_{NF}	ft	6	—	—	—	—	Constant Value	
Starting time	t_1	hr	0	—	—	—	—	Constant Value	Constant.
Exposure Duration	t_2	hr	24	—	24	8	—	Constant Value	See Section K.2.5
Averaging Time	t_{avg}	hr	8	—	—	—	—	Constant Value	See Section K.2.6
Vapor generation rate	G	mg/hr	1.6E+07	Average	3.63E+05	3.29E+07	—	Discrete	See Section K.2.7
		lb/hr	36.6	Average	0.80	72.5	—	Discrete	
Operating hours per day	OH	hr/day	24	—	—	—	—	Constant	See Section E.2.8

Table_Apx K-3. Summary of Parameter Values and Distributions Used in the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Deterministic Values		Uncertainty Analysis Distribution Parameters				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Far-field volume	V_{FF}	ft^3	10,594	Midpoint	10,594	70,629	17,657	Triangular	See Section K.2.1
Air exchange rate	AER	hr^{-1}	2	Mode	2	20	3.5	Triangular	See Section K.2.2
Near-field indoor wind speed	v_{NF}	ft/hr	1,181	50th percentile	154	23,882	—	—	See Section K.2.3
		cm/s	10	50th percentile	1.3	202.2	—	—	
Near-field length	L_{NF}	ft	10	—	—	—	—	Constant Value	See Section K.2.4
Near-field width	W_{NF}	ft	10	—	—	—	—	Constant Value	
Near-field height	H_{NF}	ft	6	—	—	—	—	Constant Value	
Starting time	t_1	hr	0	—	—	—	—	Constant Value	Constant.
Exposure Duration	t_2	hr	8	—	8	8	—	Constant Value	See Section K.2.5
Averaging Time	t_{avg}	hr	8	—	—	—	—	Constant Value	See Section K.2.6
Vapor generation rate	G	mg/hr	—	—	1.12E+05	1.12E+05	—	Discrete	See Section K.2.7; Single Data Point
Operating hours per day	OH	hr/day	24	—	—	—	—	Constant	See Section M.2.8

Table_Apx K-4. Summary of Parameter Values and Distributions Used in the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Deterministic Values		Uncertainty Analysis Distribution Parameters				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Far-field volume	V_{FF}	ft^3	10,594	Midpoint	10,594	70,629	17,657	Triangular	See Section K.2.1
Air exchange rate	AER	hr^{-1}	2	Mode	2	20	3.5	Triangular	See Section K.2.2
Near-field indoor wind speed	v_{NF}	ft/hr	1,181	50th percentile	154	23,882	—	—	See Section K.2.3
		cm/s	10	50th percentile	1.3	202.2	—	—	
Near-field length	L_{NF}	ft	10	—	—	—	—	Constant Value	See Section K.2.4
Near-field width	W_{NF}	ft	10	—	—	—	—	Constant Value	
Near-field height	H_{NF}	ft	6	—	—	—	—	Constant Value	
Starting time	t_1	hr	0	—	—	—	—	Constant Value	Constant.
Exposure Duration	t_2	hr	—	—	3	8	—	Discrete	See Section K.2.5
Averaging Time	t_{avg}	hr	8	—	—	—	—	Constant Value	See Section K.2.6
Vapor generation rate	G	mg/hr	5.14E+05	Average	6.28E+02	1.02E+06	—	Discrete	See Section K.2.7
		lb/hr	1.13	Average	0.001	2.26	—	Discrete	
Operating hours per day	OH	hr/day	—	—	—	—	—	—	See Section M.2.8

K.2.1 Far-Field Volume

EPA used the same far-field volume distribution for each of the models discussed. The far-field volume is based on information obtained from (Von Grote et al., 2003) that indicated volumes at German metal degreasing facilities can vary from 300 to several thousand cubic meters. They noted that smaller volumes are more typical and assumed 400 and 600 m^3 (14,126 and 21,189 ft^3) in their exposure models (Von Grote et al., 2003). These are the highest and lowest values EPA identified in the literature; therefore, EPA assumes a triangular distribution bound from 300 m^3 (10,594 ft^3) to 2,000 m^3 (70,629 ft^3) with a mode of 500 m^3 (the midpoint of 400 and 600 m^3) (17,657 ft^3).

K.2.2 Air Exchange Rate

EPA used the same air exchange rate distribution for each of the models discussed. The air exchange rate is based on data from (Hellweg et al., 2009) and information received from a peer reviewer during the development of the 2014 *TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses* (U.S. EPA, 2013a). (Hellweg et al., 2009) reported that average air exchange rates for occupational settings using mechanical ventilation systems vary from 3 to 20 hr^{-1}. The risk assessment peer reviewer comments indicated that values around 2 to 5 hr^{-1} are likely (U.S. EPA, 2013a), in agreement with the low end reported by (Hellweg et al., 2009). Therefore, EPA used a triangular distribution with the mode equal to 3.5 hr^{-1}, the midpoint of the range provided by the risk assessment peer reviewer (3.5 is the midpoint of the range 2 to 5 hr^{-1}), with a minimum of 2 hr^{-1}, per the risk assessment peer reviewer (U.S. EPA, 2013a) and a maximum of 20 hr^{-1} per (Hellweg et al., 2009).

K.2.3 Near-Field Indoor Air Speed

(Baldwin and Maynard, 1998a) measured indoor air speeds across a variety of occupational settings in the United Kingdom. Fifty-five work areas were surveyed across a variety of workplaces.

EPA analyzed the air speed data from (Baldwin and Maynard, 1998a) and categorized the air speed surveys into settings representative of industrial facilities and representative of commercial facilities. EPA fit separate distributions for these industrial and commercial settings and used the industrial distribution for facilities performing vapor degreasing and/or cold cleaning.

EPA fit a lognormal distribution for both data sets as consistent with the authors observations that the air speed measurements within a surveyed location were lognormally distributed and the population of the mean air speeds among all surveys were lognormally distributed. Since lognormal distributions are bound by zero and positive infinity, EPA truncated the distribution at the largest observed value among all of the survey mean air speeds from (Baldwin and Maynard, 1998a) (1998).

EPA fit the air speed surveys representative of industrial facilities to a lognormal distribution with the following parameter values: mean of 22.414 cm/s and standard deviation of 19.958 cm/s. In the model, the lognormal distribution is truncated at a maximum allowed value of 202.2 cm/s (largest surveyed mean air speed observed in (Baldwin and Maynard, 1998a) (1998)) to prevent the model from sampling values that approach infinity or are otherwise unrealistically large.

(Baldwin and Maynard, 1998a) only presented the mean air speed of each survey. The authors did not present the individual measurements within each survey. Therefore, these distributions represent a distribution of mean air speeds and not a distribution of spatially variable air speeds within a single workplace setting. However, a mean air speed (averaged over a work area) is the required input for the model.

K.2.4 Near-Field Volume

EPA assumed a near-field of constant dimensions of 10 ft x 10 ft x 6 ft resulting in a total volume of 600 ft^3.

K.2.5 Exposure Duration

EPA assumed the maximum exposure duration for each model is equal to the entire work-shift (eight hours). Therefore, if the degreaser/cold cleaning machine operating time was greater than eight hours, then exposure duration was set equal to eight hours. If the operating time was less than eight hours, then exposure duration was set equal to the degreaser/cold cleaning machine operating time (see Appendix E.2.8 for discussion of operating hours).

K.2.6 Averaging Time

EPA was interested in estimating 8-hr TWAs for use in risk calculations; therefore, a constant averaging time of eight hours was used for each of the models.

K.2.7 Vapor Generation Rate

For the vapor generation rate from each machine type (OTVD, conveyorized and cold), EPA used a discrete distribution based on the annual unit emission rates reported in the (U.S. EPA, 2018a). No web degreasers were reported in the 2014 NEI, therefore, (U.S. EPA, 2011a) data was used for web degreasers. Annual unit emission rates were converted to hourly unit emission rates by dividing the annual reported emissions by the reported annual operating hours (see Appendix E.2.8). Reported annual emissions in NEI without accompanying reported annual operating hours were not included in the analysis. Emission rates reported as zero were also excluded as it is unclear if this is before or after vapor controls used by the site and if the vapor controls used would control emissions into the work area (thus reducing exposure) or only control emissions to the environment (which would not affect worker exposures). Table_Apx K-5 summarizes the data available in the 2014 NEI.

Table_Apx K-5. Summary of Trichloroethylene Vapor Degreasing and Cold Cleaning Data from the 2014 NEI

Unit Type	Total Units	Units with Zero Emissions	Units without Accompanying Operating Hours	Units Used in Analysis[a]
Open-Top Vapor Degreasers	149	29	62	76
Conveyorized Degreasers	8	0	5	3
Web Degreasers[b]	1	0	0	1
Cold Cleaning Machines	17	1	6	10

a – Some units with zero emissions also did not include accompanying operating hours; therefore, subtracting the units with zero emissions and the units without operating hours from the total units does not equal the units in the analysis due to double counting.

b – No web degreasers reported in the 2014 NEI. One web degreaser reported in the (U.S. EPA, 2011a) was used in this analysis.

Source: (U.S. EPA, 2018a); (U.S. EPA, 2011a)

Table_Apx K-6 through Table_Apx K-9 summarize the distribution of hourly unit emissions for each machine type calculated from the annual emission in the 2014 NEI.

Table_Apx K-6. Distribution of Trichloroethylene Open-Top Vapor Degreasing Unit Emissions

Count of Units	Unit Emissions (lb/unit-hr)	Fractional Probability
1	103.00	0.0132
1	63.95	0.0132
1	19.04	0.0132
1	13.20	0.0132
1	12.18	0.0132
1	9.47	0.0132
1	9.21	0.0132
1	8.14	0.0132
1	7.30	0.0132
1	6.93	0.0132
1	6.64	0.0132
1	6.61	0.0132
1	6.44	0.0132
1	6.40	0.0132
1	6.32	0.0132
1	5.10	0.0132
1	5.06	0.0132
1	4.89	0.0132
1	4.85	0.0132
1	4.14	0.0132
1	3.96	0.0132
1	3.82	0.0132
1	3.77	0.0132
1	3.68	0.0132
2	3.66	0.0263
1	3.64	0.0132
1	3.43	0.0132
1	3.40	0.0132
1	2.88	0.0132
1	2.79	0.0132
1	2.64	0.0132
1	2.61	0.0132
1	2.48	0.0132
1	2.37	0.0132
1	2.20	0.0132
1	1.97	0.0132
1	1.96	0.0132
1	1.73	0.0132
1	1.62	0.0132

Count of Units	Unit Emissions (lb/unit-hr)	Fractional Probability
1	1.59	0.0132
1	1.44	0.0132
1	1.33	0.0132
1	1.22	0.0132
1	1.09	0.0132
2	0.93	0.0263
1	0.90	0.0132
2	0.84	0.0263
1	0.83	0.0132
1	0.79	0.0132
3	0.79	0.0395
1	0.70	0.0132
1	0.62	0.0132
1	0.60	0.0132
1	0.43	0.0132
1	0.42	0.0132
1	0.39	0.0132
1	0.38	0.0132
1	0.38	0.0132
1	0.35	0.0132
1	0.23	0.0132
1	0.18	0.0132
1	0.15	0.0132
1	0.15	0.0132
1	0.14	0.0132
1	0.11	0.0132
1	0.10	0.0132
2	0.10	0.0263
1	0.07	0.0132
1	0.03	0.0132
1	0.001	0.0132

Table_Apx K-7. Distribution of Trichloroethylene Conveyorized Degreasing Unit Emissions

Count of Units	Unit Emissions (lb/unit-hr)	Fractional Probability
1	72.48	0.3333
1	1.51	0.3333
1	0.80	0.3333

Table_Apx K-8. Distribution of Trichloroethylene Web Degreasing Unit Emissions

Count of Units	Unit Emissions (lb/unit-hr)	Fractional Probability
—	0.247	1.00

Table_Apx K-9. Distribution of Trichloroethylene Cold Cleaning Unit Emissions

Count of Units	Unit Emissions (lb/unit-hr)	Fractional Probability
1.00	2.26	0.1000
1.00	0.83	0.1000
1.00	0.83	0.1000
1.00	0.83	0.1000
1.00	0.83	0.1000
1.00	0.05	0.1000
1.00	0.01	0.1000
1.00	0.01	0.1000
1.00	0.01	0.1000
1.00	0.00	0.1000

K.2.8 Operating Hours

For the operating hours of each machine type (OTVD, conveyorized, web, and cold), EPA used a discrete distribution based on the daily operating hours reported in the 2014 NEI. It should be noted that not all units had an accompanying reported daily operating hours; therefore, the distribution for the operating hours per day is based on a subset of the reported units. Table_Apx K-10 through Table_Apx K-13 summarize the distribution of operating hours per day for each machine type.

Table_Apx K-10. Distribution of Trichloroethylene Open-Top Vapor Degreasing Operating Hours

Count of Occurrences	Operating Hours (hr/day)	Fractional Probability
—	24	0.4048
—	16	0.0952
—	8	0.2381
—	6	0.0476
—	4	0.0714
—	2	0.1429

Table_Apx K-11. Distribution of Trichloroethylene Conveyorized Degreasing Operating Hours

Count of Occurrences	Operating Hours (hr/day)	Fractional Probability
—	24	1.0000

Table_Apx K-12. Distribution of Trichloroethylene Web Degreasing Operating Hours

Count of Occurrences	Operating Hours (hr/day)	Fractional Probability
—	24	1.0000

Table_Apx K-13. Distribution of Trichloroethylene Cold Cleaning Operating Hours

Count of Occurrences	Operating Hours (hr/day)	Fractional Probability
—	24	0.4000
—	8	0.5000
—	3	0.1000

Appendix L BRAKE SERVICING NEAR-FIELD/FAR-FIELD INHALATION EXPOSURE MODEL APPROACH AND PARAMETERS

This appendix presents the modeling approach and model equations used in the Brake Servicing Near-Field/Far-Field Inhalation Exposure Model. The model was developed through review of the literature and consideration of existing EPA exposure models. This model uses a near-field/far-field approach (Nicas, 2009), where an aerosol application located inside the near-field generates a mist of droplets, and indoor air movements lead to the convection of the droplets between the near-field and far-field. Workers are assumed to be exposed to TCE droplet concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field.

The model uses the following parameters to estimate exposure concentrations in the near-field and far-field:

- Far-field size;
- Near-field size;
- Air exchange rate;
- Indoor air speed;
- Concentration of TCE in the aerosol formulation;
- Amount of degreaser used per brake job;
- Number of degreaser applications per brake job;
- Time duration of brake job;
- Operating hours per week; and
- Number of jobs per work shift.

An individual model input parameter could either have a discrete value or a distribution of values. EPA assigned statistical distributions based on reasonably available literature data. A Monte Carlo simulation (a type of stochastic simulation) was conducted to capture variability in the model input parameters. The simulation was conducted using the Latin hypercube sampling method in @Risk Industrial Edition, Version 7.0.0. The Latin hypercube sampling method is a statistical method for generating a sample of possible values from a multi-dimensional distribution. Latin hypercube sampling is a stratified method, meaning it guarantees that its generated samples are representative of the probability density function (variability) defined in the model. EPA performed the model at 100,000 iterations to capture the range of possible input values (i.e., including values with low probability of occurrence).

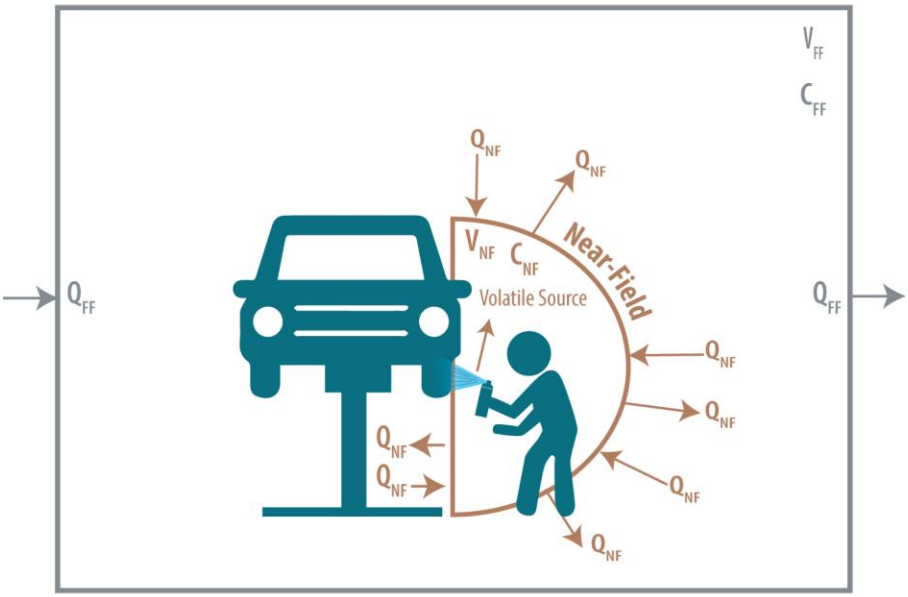
Model results from the Monte Carlo simulation are presented as 95th and 50th percentile values. The statistics were calculated directly in @Risk. The 95th percentile value was selected to represent high-end exposure level, whereas the 50th percentile value was selected to represent central tendency exposure level. The following subsections detail the model design equations and parameters for the brake servicing model.

L.1 Model Design Equations

In brake servicing, the vehicle is raised on an automobile lift to a comfortable working height to allow the worker (mechanic) to remove the wheel and access the brake system. Brake servicing can include

inspections, adjustments, brake pad replacements, and rotor resurfacing. These service types often involve disassembly, replacement or repair, and reassembly of the brake system. Automotive brake cleaners are used to remove oil, grease, brake fluid, brake pad dust, or dirt. Mechanics may occasionally use brake cleaners, engine degreasers, carburetor cleaners, and general purpose degreasers interchangeably (CARB, 2000). Automotive brake cleaners can come in aerosol or liquid form (CARB, 2000): this model estimates exposures from aerosol brake cleaners (degreasers).

Figure_Apx L-1 illustrates the near-field/far-field modeling approach as it was applied by EPA to brake servicing using an aerosol degreaser. The application of the aerosol degreaser immediately generates a mist of droplets in the near-field, resulting in worker exposures at a TCE concentration C_{NF}. The concentration is directly proportional to the amount of aerosol degreaser applied by the worker, who is standing in the near-field-zone (i.e., the working zone). The volume of this zone is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outside air.



Figure_Apx L-1. The Near-Field/Far-Field Model as Applied to the Brake Servicing Near-Field/Far-Field Inhalation Exposure Model

In brake servicing using an aerosol degreaser, aerosol degreaser droplets enter the near-field in non-steady "bursts," where each burst results in a sudden rise in the near-field concentration. The near-field and far-field concentrations then decay with time until the next burst causes a new rise in near-field concentration. Based on site data from automotive maintenance and repair shops obtained by CARB (CARB, 2000) for brake cleaning activities and as explained in Sections L.2.5 and L.2.9 below, the model assumes a worker will perform an average of 11 applications of the degreaser product per brake job with five minutes between each application and that a worker may perform one to four brake jobs per day each taking one hour to complete. EPA modeled two scenarios: one where the brake jobs occurred back-to-back and one where brake jobs occurred one hour apart. In both scenarios, EPA

assumed the worker does not perform a brake job, and does not use the aerosol degreaser, during the first hour of the day.

EPA denoted the top of each five-minute period for each hour of the day (e.g., 8:00 am, 8:05 am, 8:10 am, etc.) as $t_{m,n}$. Here, m has the values of 0, 1, 2, 3, 4, 5, 6, and 7 to indicate the top of each hour of the day (e.g., 8 am, 9 am, etc.) and n has the values of 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 11 to indicate the top of each five-minute period within the hour. No aerosol degreaser is used, and no exposures occur, during the first hour of the day, $t_{0,0}$ to $t_{0,11}$ (e.g., 8 am to 9 am). Then, in both scenarios, the worker begins the first brake job during the second hour, $t_{1,0}$ (e.g., 9 am to 10 am). The worker applies the aerosol degreaser at the top of the second 5-minute period and each subsequent 5-minute period during the hour-long brake job (e.g., 9:05 am, 9:10 am,…9:55 am). In the first scenario, the brake jobs are performed back-to-back, if performing more than one brake job on the given day. Therefore, the second brake job begins at the top of the third hour (e.g., 10 am), and the worker applies the aerosol degreaser at the top of the second 5-minute period and each subsequent 5-minute period (e.g., 10:05 am, 10:10 am,…10:55 am). In the second scenario, the brake jobs are performed every other hour, if performing more than one brake job on the given day. Therefore, the second brake job begins at the top of the fourth hour (e.g., 11 am), and the worker applies the aerosol degreaser at the top of the second 5-minute period and each subsequent 5-minute period (e.g., 11:05 am, 11:10 am,…11:55 am).

In the first scenario, after the worker performs the last brake job, the workers and occupational non-users (ONUs) continue to be exposed as the airborne concentrations decay during the final three to six hours until the end of the day (e.g., 4 pm). In the second scenario, after the worker performs each brake job, the workers and ONUs continue to be exposed as the airborne concentrations decay during the time in which no brake jobs are occurring and then again when the next brake job is initiated. In both scenarios, the workers and ONUs are no longer exposed once they leave work.

Based on data from CARB (CARB, 2000), EPA assumes each brake job requires one 14.4-oz can of aerosol brake cleaner as described in further detail below. The model determines the application rate of TCE using the weight fraction of TCE in the aerosol product. EPA uses a uniform distribution of weight fractions for TCE based on facility data for the aerosol products in use (CARB, 2000).

The model design equations are presented below.

Near-Field Mass Balance

Equation L-1

$$V_{NF}\frac{dC_{NF}}{dt} = C_{FF}Q_{NF} - C_{NF}Q_{NF}$$

Far-Field Mass Balance

Equation L-2

$$V_{FF}\frac{dC_{FF}}{dt} = C_{NF}Q_{NF} - C_{FF}Q_{NF} - C_{FF}Q_{FF}$$

Where:

V_{NF} = near-field volume;
V_{FF} = far-field volume;
Q_{NF} = near-field ventilation rate;
Q_{FF} = far-field ventilation rate;
C_{NF} = average near-field concentration;
C_{FF} = average far-field concentration; and

t = elapsed time.

Solving the above equations in terms of the time-varying concentrations in the near-field and far-field yields Equation L-3 and Equation L-4, which EPA applied to each of the 12 five-minute increments during each hour of the day. For each five-minute increment, EPA calculated the initial near-field concentration at the top of the period ($t_{m,n}$), accounting for both the burst of TCE from the degreaser application (if the five-minute increment is during a brake job) and the residual near-field concentration remaining after the previous five-minute increment ($t_{m,n-1}$; except during the first hour and $t_{m,0}$ of the first brake job, in which case there would be no residual TCE from a previous application). The initial far-field concentration is equal to the residual far-field concentration remaining after the previous five-minute increment. EPA then calculated the decayed concentration in the near-field and far-field at the end of the five-minute period, just before the degreaser application at the top of the next period ($t_{m,n+1}$). EPA then calculated a 5-minute TWA exposure for the near-field and far-field, representative of the worker's and ONUs' exposures to the airborne concentrations during each five-minute increment using Equation L-13 and Equation L-14. The *k* coefficients (Equation L-5 through Equation L-8) are a function of the initial near-field and far-field concentrations, and therefore are re-calculated at the top of each five-minute period. In the equations below, where the subscript "m, n-1" is used, if the value of n-1 is less than zero, the value at "m-1, 11" is used and where the subscript "m, n+1" is used, if the value of n+1 is greater than 11, the value at "m+1, 0" is used.

Equation L-3

$$C_{NF,t_{m,n+1}} = \left(k_{1,t_{m,n}} e^{\lambda_1 t} + k_{2,t_{m,n}} e^{\lambda_2 t}\right)$$

Equation L-4

$$C_{FF,t_{m,n+1}} = \left(k_{3,t_{m,n}} e^{\lambda_1 t} - k_{4,t_{m,n}} e^{\lambda_2 t}\right)$$

Where:

Equation L-5

$$k_{1,t_{m,n}} = \frac{Q_{NF}\left(C_{FF,0}(t_{m,n}) - C_{NF,0}(t_{m,n})\right) - \lambda_2 V_{NF} C_{NF,0}(t_{m,n})}{V_{NF}(\lambda_1 - \lambda_2)}$$

Equation L-6

$$k_{2,t_{m,n}} = \frac{Q_{NF}\left(C_{NF,0}(t_{m,n}) - C_{FF,0}(t_{m,n})\right) + \lambda_1 V_{NF} C_{NF,0}(t_{m,n})}{V_{NF}(\lambda_1 - \lambda_2)}$$

Equation L-7

$$k_{3,t_{m,n}} = \frac{(Q_{NF} + \lambda_1 V_{NF})(Q_{NF}\left(C_{FF,0}(t_{m,n}) - C_{NF,0}(t_{m,n})\right) - \lambda_2 V_{NF} C_{NF,0}(t_{m,n}))}{Q_{NF} V_{NF}(\lambda_1 - \lambda_2)}$$

Equation L-8

$$k_{4,t_{m,n}} = \frac{(Q_{NF} + \lambda_2 V_{NF})(Q_{NF}\left(C_{NF,0}(t_{m,n}) - C_{FF,0}(t_{m,n})\right) + \lambda_1 V_{NF} C_{NF,0}(t_{m,n}))}{Q_{NF} V_{NF}(\lambda_1 - \lambda_2)}$$

Equation L-9

$$\lambda_1 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) + \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

Equation L-10

$$\lambda_2 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) - \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

Equation L-11

$$C_{NF,o}(t_{m,n}) = \begin{cases} 0, & m = 0 \\ \frac{Amt}{V_{NF}}\left(1{,}000\,\frac{mg}{g}\right) + C_{NF}(t_{m,n-1}), & n > 0 \text{ for all } m \text{ where brake job occurs} \end{cases}$$

Equation L-12

$$C_{FF,o}(t_{m,n}) = \begin{cases} 0, & m = 0 \\ C_{FF}(t_{m,n-1}), & \text{for all } n \text{ where } m > 0 \end{cases}$$

Equation L-13

$$C_{NF,\text{ 5-min TWA, }t_{m,n}} = \frac{\left(\frac{k_{1,t_{m,n-1}}}{\lambda_1}e^{\lambda_1 t_2} + \frac{k_{2,t_{m,n-1}}}{\lambda_2}e^{\lambda_2 t_2}\right) - \left(\frac{k_{1,t_{m,n-1}}}{\lambda_1}e^{\lambda_1 t_1} + \frac{k_{2,t_{m,n-1}}}{\lambda_2}e^{\lambda_2 t_1}\right)}{t_2 - t_1}$$

Equation L-14

$$C_{FF,\text{ 5-min TWA, }t_{m,n}} = \frac{\left(\frac{k_{3,t_{m,n-1}}}{\lambda_1}e^{\lambda_1 t_2} + \frac{k_{4,t_{m,n-1}}}{\lambda_2}e^{\lambda_2 t_2}\right) - \left(\frac{k_{3,t_{m,n-1}}}{\lambda_1}e^{\lambda_1 t_1} + \frac{k_{4,t_{m,n-1}}}{\lambda_2}e^{\lambda_2 t_1}\right)}{t_2 - t_1}$$

After calculating all near-field/far-field 5-minute TWA exposures (i.e., $C_{NF,\text{ 5-min TWA, }t_{m,n}}$ and $C_{FF,\text{ 5-min TWA, }t_{m,n}}$) for each five-minute period of the work day, EPA calculated the near-field/far-field 8-hour TWA concentration and 1-hour TWA concentrations following the equations below:

Equation L-15

$$C_{NF,\text{ 8-hr }TWA} = \frac{\sum_{m=0}^{7}\sum_{n=0}^{11}\left[C_{NF,\text{5-min }TWA,t_{m,n}} \times 0.0833\ hr\right]}{8\ hr}$$

Equation L-16

$$C_{NF,\text{ 8-hr }TWA} = \frac{\sum_{m=0}^{7}\sum_{n=0}^{11}\left[C_{FF,\text{5-min }TWA,t_{m,n}} \times 0.0833\ hr\right]}{8\ hr}$$

Equation L-17

$$C_{NF,\text{1-hr } TWA} = \frac{\sum_{n=0}^{11}\left[C_{NF,\text{5-min } TWA,t_{m,n}} \times 0.0833\ hr\right]}{1\ hr}$$

Equation L-18

$$C_{FF,\text{1-hr } TWA} = \frac{\sum_{n=0}^{11}\left[C_{FF,\text{5-min } TWA,t_{m,n}} \times 0.0833\ hr\right]}{1\ hr}$$

EPA calculated rolling 1-hour TWA's throughout the workday and the model reports the maximum calculated 1-hour TWA.

To calculate the mass transfer to and from the near-field, the free surface area (FSA) is defined to be the surface area through which mass transfer can occur. The FSA is not equal to the surface area of the entire near-field. EPA defined the near-field zone to be a hemisphere with its major axis oriented vertically, against the vehicle, and aligned through the center of the wheel (see Figure_Apx L-1). The top half of the circular cross-section rests against, and is blocked by, the vehicle and is not available for mass transfer. The FSA is calculated as the entire surface area of the hemisphere's curved surface and half of the hemisphere's circular surface per Equation L-19, below:

Equation L-19

$$FSA = \left(\frac{1}{2} \times 4\pi R_{NF}^2\right) + \left(\frac{1}{2} \times \pi R_{NF}^2\right)$$

Where: R_{NF} is the radius of the near-field

The near-field ventilation rate, Q_{NF}, is calculated in Equation M-1520 from the indoor wind speed, v_{NF}, and FSA, assuming half of the FSA is available for mass transfer into the near-field and half of the FSA is available for mass transfer out of the near-field:

Equation L-20

$$Q_{NF} = \frac{1}{2} v_{NF} FSA$$

The far-field volume, V_{FF}, and the air exchange rate, AER, is used to calculate the far-field ventilation rate, Q_{FF}, as given by Equation M-21:

Equation L-21

$$Q_{FF} = V_{FF} AER$$

Using the model inputs described in Appendix F.2, EPA estimated TCE inhalation exposures for workers in the near-field and for occupational non-users in the far-field. EPA then conducted the Monte Carlo simulations using @Risk (Version 7.0.0). The simulations applied 100,000 iterations and the Latin Hypercube sampling method.

L.2 Model Parameters

Table_Apx L-1 summarizes the model parameters and their values for the Brake Servicing Near-Field/ Far-Field Inhalation Exposure Model. Each parameter is discussed in detail in the following subsections.

Table_Apx L-1. Summary of Parameter Values and Distributions Used in the Brake Servicing Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Constant Model Parameter Values		Variable Model Parameter Values				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Far-field volume	V_{FF}	m^3	—	—	206	70,679	3,769	Triangular	Distribution based on data collected by CARB (CARB, 2000).
Air exchange rate	AER	hr^{-1}	—	—	1	20	3.5	Triangular	(Demou et al., 2009) identifies typical AERs of 1 hr^{-1} and 3 to 20 hr^{-1} for occupational settings without and with mechanical ventilation systems, respectively. (Hellweg et al., 2009) identifies average AERs for occupational settings utilizing mechanical ventilation systems to be between 3 and 20 hr^{-1}. (Golsteijn et al., 2014) indicates a characteristic AER of 4 hr^{-1}. Peer reviewers of EPA's 2013 TCE draft risk assessment commented that values around 2 to 5 hr^{-1} may be more likely (U.S. EPA, 2013a), in agreement with (Golsteijn et al., 2014). A triangular distribution is used with the mode equal to the midpoint of the range provided by the peer reviewer (3.5 is the midpoint of the range 2 to 5 hr^{-1}).
Near-field indoor wind speed	v_{NF}	ft/hr	—	—	0	23,882	—	Lognormal	Lognormal distribution fit to commercial-type workplace data from (Baldwin and Maynard, 1998a).
		cm/s	—	—	0	202.2	—	Lognormal	
Near-field radius	R_{NF}	m	1.5	—	—	—	—	Constant Value	Constant.
Starting time for each application period	t_1	hr	0	—	—	—	—	Constant Value	Constant.

Input Parameter	Symbol	Unit	Constant Model Parameter Values		Variable Model Parameter Values				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
End time for each application period	t_2	hr	0.0833	—	—	—	—	Constant Value	Assumes aerosol degreaser is applied in 5-minute increments during brake job.
Averaging Time	t_{avg}	hr	8	—	—	—	—	Constant Value	Constant.
TCE weight fraction	wtfrac	wt frac	—	—	0.40	1.00	—	Discrete	Discrete distribution of TCE-based aerosol product formulations based on products identified in EPA's Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal for TCE (U.S. EPA, 2017c). Where the weight fraction of TCE in the formulation was given as a range, EPA assumed a uniform distribution within the reported range for the TCE concentration in the product.
Degreaser Used per Brake Job	W_d	oz/ job	14.4	—	—	—	—	Constant Value	Based on data from CARB (CARB, 2000).
Number of Applications per Job	N_A	Applications/ job	11	—	—	—	—	Constant Value	Calculated from the average of the number of applications per brake and number of brakes per job.
Amount Used per Application	Amt	g TCE/ application	—	—	14.8	37.1	—	Calculated	Calculated from wtfrac, W_d, and N_A.
Operating hours per week	OHpW	hr/week	—	—	40	122.5	—	Lognormal	Lognormal distribution fit to the operating hours per week observed in CARB (CARB, 2000) site visits.
Number of Brake Jobs per Work Shift	N_J	jobs/site-shift	—	—	1	4	—	—	Calculated from the average number of brake jobs per site per year, OHpW, and assuming 52 operating weeks per year and 8 hours per work shift.

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L.2.1 Far-Field Volume

The far-field volume is based on information obtained from (CARB, 2000) from site visits of 137 automotive maintenance and repair shops in California. (CARB, 2000) indicated that shop volumes at the visited sites ranged from 200 to 70,679 m^3 with an average shop volume of 3,769 m^3. Based on this data EPA assumed a triangular distribution bound from 200 m^3 to 70,679 m^3 with a mode of 3,769 m^3 (the average of the data from (CARB, 2000)).

CARB measured the physical dimensions of the portion of the facility where brake service work was performed at the visited facilities. CARB did not consider other areas of the facility, such as customer waiting areas and adjacent storage rooms, if they were separated by a normally closed door. If the door was normally open, then CARB did consider those areas as part of the measured portion where brake servicing emissions could occur (CARB, 2000). CARB's methodology for measuring the physical dimensions of the visited facilities provides the appropriate physical dimensions needed to represent the far-field volume in EPA's model. Therefore, CARB's reported facility volume data are appropriate for EPA's modeling purposes.

L.2.2 Air Exchange Rate

The air exchange rate (AER) is based on data from (Demou et al., 2009), (Hellweg et al., 2009), (Golsteijn et al., 2014), and information received from a peer reviewer during the development of the 2014 *TSCA Work Plan Chemical Risk Assessment Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses* (U.S. EPA, 2013a). (Demou et al., 2009) identifies typical AERs of 1 hr^{-1} and 3 to 20 hr^{-1} for occupational settings without and with mechanical ventilation systems, respectively. Similarly, (Hellweg et al., 2009) identifies average AERs for occupational settings using mechanical ventilation systems to vary from 3 to 20 hr^{-1}. (Golsteijn et al., 2014) indicates a characteristic AER of 4 hr^{-1}. The risk assessment peer reviewer comments indicated that values around 2 to 5 hr^{-1} are likely (U.S. EPA, 2013a), in agreement with (Golsteijn et al., 2014) and the low end reported by (Demou et al., 2009) and (Hellweg et al., 2009). Therefore, EPA used a triangular distribution with the mode equal to 3.5 hr^{-1}, the midpoint of the range provided by the risk assessment peer reviewer (3.5 is the midpoint of the range 2 to 5 hr^{-1}), with a minimum of 1 hr^{-1}, per (Demou et al., 2009) and a maximum of 20 hr^{-1} per (Demou et al., 2009) and (Hellweg et al., 2009)).

L.2.3 Near-Field Indoor Air Speed

(Baldwin and Maynard, 1998a) measured indoor air speeds across a variety of occupational settings in the United Kingdom. Fifty-five work areas were surveyed across a variety of workplaces.

EPA analyzed the air speed data from (Baldwin and Maynard, 1998a) and categorized the air speed surveys into settings representative of industrial facilities and representative of commercial facilities. EPA fit separate distributions for these industrial and commercial settings and used the commercial distribution for facilities performing aerosol degreasing or other aerosol applications.

EPA fit a lognormal distribution for both data sets as consistent with the authors observations that the air speed measurements within a surveyed location were lognormally distributed and the population of the mean air speeds among all surveys were lognormally distributed. Since lognormal distributions are bound by zero and positive infinity, EPA truncated the distribution at the largest observed value among all of the survey mean air speeds from (Baldwin and Maynard, 1998a).

EPA fit the air speed surveys representative of commercial facilities to a lognormal distribution with the following parameter values: mean of 10.853 cm/s and standard deviation of 7.883 cm/s. In the model,

the lognormal distribution is truncated at a maximum allowed value of 202.2 cm/s (largest surveyed mean air speed observed in (Baldwin and Maynard, 1998a) to prevent the model from sampling values that approach infinity or are otherwise unrealistically large.

(Baldwin and Maynard, 1998a) only presented the mean air speed of each survey. The authors did not present the individual measurements within each survey. Therefore, these distributions represent a distribution of mean air speeds and not a distribution of spatially-variable air speeds within a single workplace setting. However, a mean air speed (averaged over a work area) is the required input for the model.

L.2.4 Near-Field Volume

EPA defined the near-field zone to be a hemisphere with its major axis oriented vertically, against the vehicle, and aligned through the center of the wheel (see Figure_Apx L-1). The near-field volume is calculated per Equation L-22. EPA defined a near-field radius (R_{NF}) of 1.5 meters, approximately 4.9 feet, as an estimate of the working height of the wheel, as measured from the floor to the center of the wheel.

Equation L-22

$$V_{NF} = \frac{1}{2} \times \frac{4}{3} \pi R_{NF}^3$$

L.2.5 Application Time

EPA assumed an average of 11 brake cleaner applications per brake job (see Section F.2.9). CARB observed, from their site visits, that the visited facilities did not perform more than one brake job in any given hour (CARB, 2000). Therefore, EPA assumed a brake job takes one hour to perform. Using an assumed average of 11 brake cleaner applications per brake job and one hour to perform a brake job, EPA calculates an average brake cleaner application frequency of once every five minutes (0.0833 hr). EPA models an average brake job of having no brake cleaner application during its first five minutes and then one brake cleaner application per each subsequent 5-minute period during the one-hour brake job.

L.2.6 Averaging Time

EPA was interested in estimating 8-hr TWAs for use in risk calculations; therefore, a constant averaging time of eight hours was used.

L.2.7 Trichloroethylene Weight Fraction

EPA reviewed the *Preliminary Information on Manufacturing, Processing, Distribution, Use, and Disposal: Trichloroethylene* report (U.S. EPA, 2017c) for aerosol degreasers that contain TCE. EPA (2017) identifies 16 aerosol degreaser products that overall range in TCE content from 40 to 100 weight percent. The identified aerosol degreasers include a brake cleaner as well as general purpose degreasers, machine cleaners, electronic/electrical parts cleaners, and a mold cleaner. EPA includes all of these aerosol degreasers in the estimation of TCE content as: 1) automotive maintenance and repair facilities may use different degreaser products interchangeably as observed by (CARB, 2000); and 2) EPA uses this brake servicing model as an exposure scenario representative of all commercial-type aerosol degreaser applications.

EPA used a discrete distribution to model the TCE weight fraction based on the number of occurrences of each product type. In some instances, the concentration of TCE was reported as a range. For these product types, EPA used a uniform distribution to model the TCE weight fraction within the product

type. Table_Apx L-2 provides a summary of the reported TCE content reported in the safety data sheets identified in (U.S. EPA, 2017c), the number of occurrences of each product type, and the fractional probability of each product type.

Table_Apx L-2. Summary of Trichloroethylene-Based Aerosol Degreaser Formulations

Name of Aerosol Degreaser Product Identified in (U.S. EPA, 2017c)	Trichloroethylene Weight Percent	Number of Occurrences	Fractional Probability
C-60 Solvent Degreaser	90-100%	1	0.063
Fusing Machine Cleaner	40-60%	1	0.063
Solvent Degreaser	> 90%	1	0.063
Electro Blast	90-100%	1	0.063
Electro Solv	90-100%	1	0.063
Pro Tools NF Solvent Degreaser	60-100%	1	0.063
Aerosolve II	>90%	1	0.063
Power Solv II	90-100%	1	0.063
Zep 45	40-50%	1	0.063
Super Solv	90-100%	1	0.063
Parts Cleaner	45-55%	1	0.063
Electronic Contact Cleaner & Protectant - Aerosol	97%	1	0.063
Flash Free Electrical Degreaser	98%	1	0.063
Chlorinated Brake & Parts Cleaner – Aerosol	98%	1	0.063
MR 351 - Mold Cleaner	69%	1	0.063
C-60 Solvent [TCE Cleaner] Degreaser	90-100%	1	0.063
	Total	**16**	**1.000**

L.2.8 Volume of Degreaser Used per Brake Job

(CARB, 2000) assumed that brake jobs require 14.4 oz of aerosol product. EPA did not identify other information to estimate the volume of aerosol product per job; therefore, EPA used a constant volume of 14.4 oz per brake job based on (CARB, 2000).

L.2.9 Number of Applications per Brake Job

Workers typically apply the brake cleaner before, during, and after brake disassembly. Workers may also apply the brake cleaner after brake reassembly as a final cleaning process (CARB, 2000). Therefore, EPA assumed a worker applies a brake cleaner three or four times per wheel. Since a brake job can be performed on either one axle or two axles (CARB, 2000), EPA assumed a brake job may involve either two or four wheels. Therefore, the number of brake cleaner (aerosol degreaser) applications per brake job can range from six (3 applications/brake x 2 brakes) to 16 (4 applications/brake x 4 brakes). EPA assumed a constant number of applications per brake job based on the midpoint of this range of 11 applications per brake job.

L.2.10 Amount of Trichloroethylene Used per Application

EPA calculated the amount of Trichloroethylene used per application using Equation L-23. The calculated mass of Trichloroethylene used per application ranges from 14.8 to 37.1 grams.

Equation L-23

$$Amt = \frac{W_d \times wtfrac \times 28.3495 \frac{g}{oz}}{N_A}$$

Where:

Amt	=	Amount of TCE used per application (g/application);
W_d	=	Weight of degreaser used per brake job (oz/job);
Wtfrac	=	Weight fraction of TCE in aerosol degreaser (unitless); and
N_A	=	Number of degreaser applications per brake job (applications/job).

L.2.11 Operating Hours per Week

(CARB, 2000) collected weekly operating hour data for 54 automotive maintenance and repair facilities. The surveyed facilities included service stations (fuel retail stations), general automotive shops, car dealerships, brake repair shops, and vehicle fleet maintenance facilities. The weekly operating hours of the surveyed facilities ranged from 40 to 122.5 hr/week. EPA fit a lognormal distribution to the surveyed weekly operating hour data. The resulting lognormal distribution has a mean of 16.943 and standard deviation of 13.813, which set the shape of the lognormal distribution. EPA shifted the distribution to the right such that its minimum value is 40 hr/week and set a truncation of 122.5 hr/week (the truncation is set as 82.5 hr/week relative to the left shift of 40 hr/week).

L.2.12 Number of Brake Jobs per Work Shift

(CARB, 2000) visited 137 automotive maintenance and repair shops and collected data on the number of brake jobs performed annually at each facility. CARB calculated an average of 936 brake jobs performed per facility per year. EPA calculated the number of brake jobs per work shift using the average number of jobs per site per year, the operating hours per week, and assuming 52 weeks of operation per year and eight hours per work shift using Equation L-24 and rounding to the nearest integer. The calculated number of brake jobs per work shift ranges from one to four.

Equation L-24

$$N_J = \frac{936 \frac{jobs}{site\text{-}year} \times 8 \frac{hours}{shift}}{52 \frac{weeks}{yr} \times OHpW}$$

Where:

N_J	=	Number of brake jobs per work shift (jobs/site-shift); and
OHpW	=	Operating hours per week (hr/week).

Appendix M SPOT CLEANING NEAR-FIELD/FAR-FIELD INHALATION EXPOSURE MODEL APPROACH AND PARAMETERS

This appendix presents the modeling approach and model equations used in the Spot Cleaning Near-Field/Far-Field Inhalation Exposure Model. The model was developed through review of relevant literature and consideration of existing EPA exposure models. The model uses a near-field/far-field approach (AIHA, 2009), where a vapor generation source located inside the near-field leads to the evaporation of vapors into the near-field, and indoor air movements lead to the convection of vapors between the near-field and far-field. Workers are assumed to be exposed to TCE vapor concentrations in the near-field, while occupational non-users are exposed at concentrations in the far-field.

The model uses the following parameters to estimate exposure concentrations in the near-field and far-field:

- Far-field size;
- Near-field size;
- Air exchange rate;
- Indoor air speed;
- Spot cleaner use rate;
- Vapor generation rate;
- Weight fraction of TCE in the spot cleaner; and
- Operating hours per day.

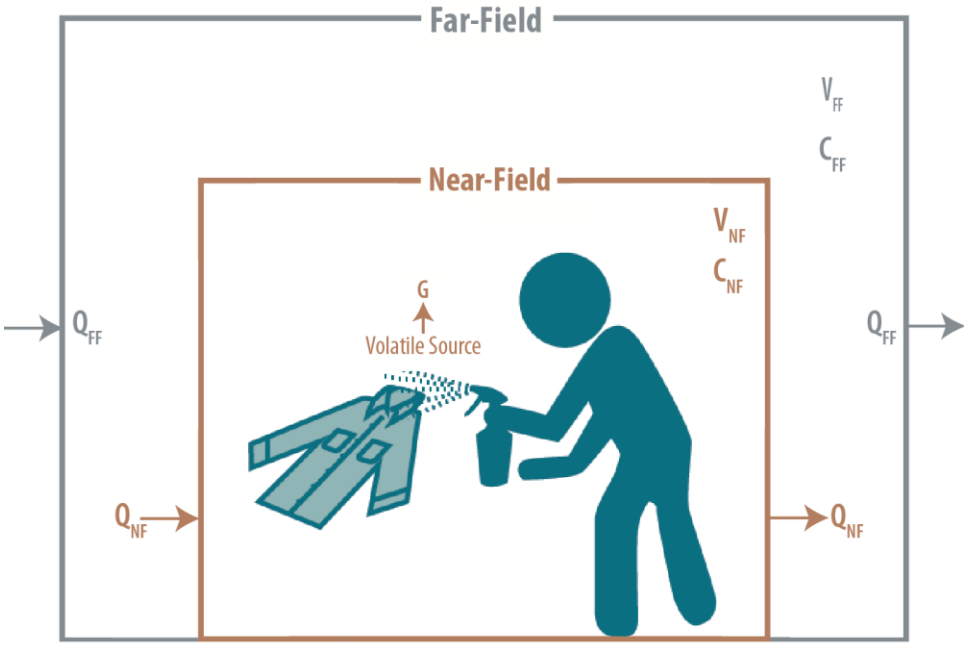
An individual model input parameter could either have a discrete value or a distribution of values. EPA assigned statistical distributions based on reasonably available literature data. A Monte Carlo simulation (a type of stochastic simulation) was conducted to capture variability in the model input parameters. The simulation was conducted using the Latin hypercube sampling method in @Risk Industrial Edition, Version 7.0.0. The Latin hypercube sampling method is a statistical method for generating a sample of possible values from a multi-dimensional distribution. Latin hypercube sampling is a stratified method, meaning it guarantees that its generated samples are representative of the probability density function (variability) defined in the model. EPA performed the model at 100,000 iterations to capture the range of possible input values (i.e., including values with low probability of occurrence).

Model results from the Monte Carlo simulation are presented as 95th and 50th percentile values. The statistics were calculated directly in @Risk. The 95th percentile value was selected to represent a high-end exposure, whereas the 50th percentile value was selected to represent a central tendency exposure level. The following subsections detail the model design equations and parameters for the spot cleaning model.

M.1 Model Design Equations

Figure_Apx M-1 illustrates the near-field/far-field modeling approach as it was applied by EPA to spot cleaning facilities. As the figure shows, TCE vapors evaporate into the near-field (at evaporation rate G), resulting in near-field exposures to workers at a concentration C_{NF}. The concentration is directly proportional to the amount of spot cleaner applied by the worker, who is standing in the near-field-zone (i.e., the working zone). The volume of this zone is denoted by V_{NF}. The ventilation rate for the near-

field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outdoor air.



Figure_Apx M-1. The Near-Field/Far-Field Model as Applied to the Spot Cleaning Near-Field/Far-Field Inhalation Exposure Model

The model design equations are presented below in Equation M-1 through Equation M-16.

Near-Field Mass Balance

Equation M-1

$$V_{NF}\frac{dC_{NF}}{dt} = C_{FF}Q_{NF} - C_{NF}Q_{NF} + G$$

Far-Field Mass Balance

Equation M-2

$$V_{FF}\frac{dC_{FF}}{dt} = C_{NF}Q_{NF} - C_{FF}Q_{NF} - C_{FF}Q_{FF}$$

Where:

V_{NF}	=	near-field volume;
V_{FF}	=	far-field volume;
Q_{NF}	=	near-field ventilation rate;
Q_{FF}	=	far-field ventilation rate;
C_{NF}	=	average near-field concentration;
C_{FF}	=	average far-field concentration;
G	=	average vapor generation rate; and
t	=	elapsed time.

Both of the previous equations can be solved for the time-varying concentrations in the near-field and far-field as follows (AIHA, 2009):

Equation M-3

$$C_{NF} = G\left(k_1 + k_2 e^{\lambda_1 t} - k_3 e^{\lambda_2 t}\right)$$

Equation M-4

$$C_{FF} = G\left(\frac{1}{Q_{FF}} + k_4 e^{\lambda_1 t} - k_5 e^{\lambda_2 t}\right)$$

Where:

Equation M-5

$$k_1 = \frac{1}{\left(\frac{Q_{NF}}{Q_{NF} + Q_{FF}}\right) Q_{FF}}$$

Equation M-6

$$k_2 = \frac{Q_{NF}Q_{FF} + \lambda_2 V_{NF}(Q_{NF} + Q_{FF})}{Q_{NF}Q_{FF}V_{NF}(\lambda_1 - \lambda_2)}$$

Equation M-7

$$k_3 = \frac{Q_{NF}Q_{FF} + \lambda_1 V_{NF}(Q_{NF} + Q_{FF})}{Q_{NF}Q_{FF}V_{NF}(\lambda_1 - \lambda_2)}$$

Equation M-8

$$k_4 = \left(\frac{\lambda_1 V_{NF} + Q_{NF}}{Q_{NF}}\right) k_2$$

Equation M-9

$$k_5 = \left(\frac{\lambda_2 V_{NF} + Q_{NF}}{Q_{NF}}\right) k_3$$

Equation M-10

$$\lambda_1 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) + \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

Equation M-11

$$\lambda_2 = 0.5\left[-\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right) - \sqrt{\left(\frac{Q_{NF}V_{FF} + V_{NF}(Q_{NF} + Q_{FF})}{V_{NF}V_{FF}}\right)^2 - 4\left(\frac{Q_{NF}Q_{FF}}{V_{NF}V_{FF}}\right)}\right]$$

EPA calculated the hourly TWA concentrations in the near-field and far-field using the following equations. Note that the numerator and denominator of Equation M-12 and Equation M-1313, use two

different sets of time parameters. The numerator is based on the operating hours for the scenario while the denominator is fixed to an averaging time span, t_avg, of 8 hours (since EPA is interested in calculating 8-hr TWA exposures). Mathematically, the numerator and denominator must reflect the same amount of time. This is indeed the case: although the spot cleaning operating hours ranges from two to five hours (as discussed in Section A.2.8), EPA assumes exposures are equal to zero outside of the operating hours, such that the integral over the balance of the eight hours (three to six hours) is equal to zero in the numerator. Therefore, the numerator inherently includes an integral over the balance of the eight hours equal to zero that is summed to the integral from t_1 to t_2.

Equation M-12

$$C_{NF,TWA} = \frac{\int_{t_1}^{t_2} C_{NF}\,dt}{\int_0^{t_{avg}} dt} = \frac{\int_{t_1}^{t_2} G\left(k_1 + k_2 e^{\lambda_1 t} - k_3 e^{\lambda_2 t}\right)dt}{t_{avg}} =$$

$$\frac{G\left(k_1 t_2 + \frac{k_2 e^{\lambda_1 t_2}}{\lambda_1} - \frac{k_3 e^{\lambda_2 t_2}}{\lambda_2}\right) - G\left(k_1 t_1 + \frac{k_2 e^{\lambda_1 t_1}}{\lambda_1} - \frac{k_3 e^{\lambda_2 t_1}}{\lambda_2}\right)}{t_{avg}}$$

Equation M-13

$$C_{FF,TWA} = \frac{\int_{t_1}^{t_2} C_{FF}\,dt}{\int_0^{t_{avg}} dt} = \frac{\int_{t_1}^{t_2} G\left(\frac{1}{Q_{FF}} + k_4 e^{\lambda_1 t} - k_5 e^{\lambda_2 t}\right)dt}{t_{avg}} =$$

$$\frac{G\left(\frac{t_2}{Q_{FF}} + \frac{k_4 e^{\lambda_1 t_2}}{\lambda_1} - \frac{k_5 e^{\lambda_2 t_2}}{\lambda_2}\right) - G\left(\frac{t_1}{Q_{FF}} + \frac{k_4 e^{\lambda_1 t_1}}{\lambda_1} - \frac{k_5 e^{\lambda_2 t_1}}{\lambda_2}\right)}{t_{avg}}$$

To calculate the mass transfer to and from the near-field, the Free Surface Area, FSA, is defined to be the surface area through which mass transfer can occur. Note that the FSA is not equal to the surface area of the entire near-field. EPA defined the near-field zone to be a rectangular box resting on the floor; therefore, no mass transfer can occur through the near-field box's floor. FSA is calculated in Equation M-14, below:

Equation M-14

$$FSA = 2(L_{NF}H_{NF}) + 2(W_{NF}H_{NF}) + (L_{NF}W_{NF})$$

Where: L_{NF}, W_{NF}, and H_{NF} are the length, width, and height of the near-field, respectively. The near-field ventilation rate, Q_{NF}, is calculated in Equation M-15 from the near-field indoor wind speed, v_{NF}, and FSA, assuming half of FSA is available for mass transfer into the near-field and half of FSA is available for mass transfer out of the near-field:

Equation M-15

$$Q_{NF} = \frac{1}{2} v_{NF} FSA$$

The far-field volume, V_{FF}, and the air exchange rate, AER, is used to calculate the far-field ventilation rate, Q_{FF}, as given by Equation M-:

Equation M-16

$$Q_{FF} = V_{FF}AER$$

Using the model inputs in Table H-1, EPA estimated TCE inhalation exposures for workers in the near-field and for occupational non-user in the far-field. EPA then conducted the Monte Carlo simulations using @Risk (Version 7.0.0). The simulations applied 100,000 iterations and the Latin hypercube sampling method.

M.2 Model Parameters

Table_Apx M-1 summarizes the model parameters and their values for the Spot Cleaning Near-Field/Far-Field Exposure Model. Each parameter is discussed in detail in the following subsections.

Table_Apx M-1. Summary of Parameter Values and Distributions Used in the Spot Cleaning Near-Field/Far-Field Inhalation Exposure Model

Input Parameter	Symbol	Unit	Constant Model Parameter Values		Variable Model Parameter Values				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Floor Area	A	ft^2	—	—	500	20,000	—	Beta	Facility floor area is based on data from the (CARB, 2006) and King County (Whittaker and Johanson, 2011) study. ERG fit a beta function to this distribution with parameters: α_1 = 6.655, α_2 = 108.22, min = 500 ft^2, max = 20,000 ft^2.
Far-field volume	V_{FF}	ft^3	—	—	6,000	240,000	—	—	Floor area multiplied by height. Facility height is 12 ft (median value per (CARB, 2006) study).
Near-field length	L_{NF}	ft	10	—	—	—	—	—	EPA assumed a constant near-field volume.
Near-field width	W_{NF}	ft	10	—	—	—	—	—	
Near-field height	H_{NF}	ft	6	—	—	—	—	—	
Air exchange rate	AER	hr^{-1}	—	—	1	19	3.5	Triangular	Values based on (von Grote et al., 2006), and (U.S. EPA, 2013a). The mode represents the midpoint of the range reported in (U.S. EPA, 2013a).
Near-field indoor wind speed	v_{NF}	cm/s	—	—	0	202.2	—	Lognormal	Lognormal distribution fit to the data presented in (Baldwin and Maynard, 1998a).
		ft/hr	—	—	0	23,882	—	Lognormal	
Starting time	t_1	hr	0	—	—	—	—	—	Constant value.
Exposure Duration	t_2	hr	—	—	2	5	—	Uniform	Equal to operating hours per day.
Averaging time	t_{avg}	hr	8	—	—	—	—	—	Constant value.

Input Parameter	Symbol	Unit	Constant Model Parameter Values		Variable Model Parameter Values				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Use rate	UR	gal/yr	8.4	—	—	—	—	—	(IRTA, 2007) used estimates of the amount of TCE-based spot cleaner sold in California and the number of textile cleaning facilities in California to calculate a use rate value.
Vapor generation rate	G	mg/hr	—	—	2.97E+03	9.32E+04	—	Calculated	G is calculated based on UR and assumes 100% volatilization and accounts for the weight fraction of TCE.
		g/min	—	—	0.05	1.55	—	Calculated	
TCE weight fraction	wtfrac	wt frac	—	—	0.1	1	—	Uniform	(IRTA, 2007) observed TCE-based spotting agents contain 10% to 100% TCE.
Operating hours per day	OH	hr/day	—	—	2	5	—	Uniform	Determined from a California survey performed by (Morris and Wolf, 2005) and an analysis of two model plants constructed by the researchers
Operating days per year	OD	days/yr	—	—	249	313	300	Triangular	Operating days/yr distribution assumed as triangular distribution with min of 250, max of 312, and mode of 300.

Input Parameter	Symbol	Unit	Constant Model Parameter Values		Variable Model Parameter Values				Comments
			Value	Basis	Lower Bound	Upper Bound	Mode	Distribution Type	
Fractional number of operating days that a worker works	*f*	Dimensionless	1	—	0.8	1.0	—	Uniform	In BLS/Census data, the weighted average worked hours per year and per worker in the dry cleaning sector is approximately 1,600 (i.e., 200 day/yr at 8 hr/day). The BLS/Census data weighted average of 200 day/yr falls outside the triangular distribution of operating days and to account for lower exposure frequencies and part-time workers, EPA defines *f* as a uniform distribution ranging from 0.8 to 1.0. The 0.8 value was derived from the observation that the weighted average of 200 day/yr worked (from BLS/Census) is 80% of the standard assumption that a full-time worker works 250 day/yr. The maximum of 1.0 is appropriate as dry cleaners may be family owned and operated and some workers may work as much as every operating day.

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M.2.1 Far-Field Volume

EPA calculated the far-field volume by setting a distribution for the facility floor area and multiplying the floor area by a facility height of 12 ft (median value per (CARB, 2006) study) as discussed in more detail below.

The 2006 CARB *California Dry Cleaning Industry Technical Assessment Report* (CARB, 2006) and the Local Hazardous Waste Management Program in King County *A Profile of the Dry Cleaning Industry in King County, Washington* (Whittaker and Johanson, 2011) provide survey data on dry cleaning facility floor area. The CARB (2006) study also provides survey data on facility height. Using survey results from both studies, EPA composed the following distribution of floor area. To calculate facility volume, EPA used the median facility height from the CARB (2006) study. The facility height distribution in the CARB (2006) study has a low level of variability, so the median height value of 12 ft presents a simple but reasonable approach to calculate facility volume combined with the floor area distribution. Results are provided in Table_Apx M-2

Table_Apx M-2. Composite Distribution of Dry Cleaning Facility Floor Areas

Floor Area Value (ft^2)	Percentile (as fraction)	Source
20,000	1	King County
3,000	0.96	King County
2,000	0.84	King County
1,600	0.5	CARB 2006
1,100	0.1	CARB 2006
500	0	CARB 2006

EPA fit a beta function to this distribution with parameters: $\alpha_1 = 6.655$, $\alpha_2 = 108.22$, min = 500 ft^2, max = 20,000 ft^2.

M.2.2 Near-Field Volume

EPA assumed a near-field of constant dimensions of 10 ft wide by 10 ft long by 6 ft high resulting in a total volume of 600 ft^3.

M.2.3 Air Exchange Rate

(von Grote et al., 2006) indicated typical air exchange rates (AERs) of 5 to 19 hr^{-1} for dry cleaning facilities in Germany. (Klein and Kurz, 1994a) indicated AERs of 1 to 19 hr^{-1}, with a mean of 8 hr^{-1} for dry cleaning facilities in Germany. During the 2013 peer review of EPA's 2013 draft risk assessment of TCE, a peer reviewer indicated that air exchange rate values around 2 to 5 hr^{-1} are likely (U.S. EPA, 2013a), in agreement with the low end of the ranges reported by von Grote et al. and (Klein and Kurz, 1994a). A triangular distribution is used with the mode equal to the midpoint of the range provided by the peer reviewer (3.5 is the midpoint of the range 2 to 5 hr^{-1}).

M.2.4 Near-Field Indoor Wind Speed

(Baldwin and Maynard, 1998a) measured indoor air speeds across a variety of occupational settings in the United Kingdom. Fifty-five work areas were surveyed across a variety of workplaces.

EPA analyzed the air speed data from (Baldwin and Maynard, 1998a) and categorizing the air speed surveys into settings representative of industrial facilities and representative of commercial facilities.

EPA fit separate distributions for these industrial and commercial settings and used the commercial distribution for dry cleaners (including other textile cleaning facilities that conduct spot cleaning).

EPA fit a lognormal distribution for both data sets as consistent with the authors observations that the air speed measurements within a surveyed location were lognormally distributed and the population of the mean air speeds among all surveys were lognormally distributed. Since lognormal distributions are bound by zero and positive infinity, EPA truncated the distribution at the largest observed value among all of the survey mean air speeds from (Baldwin and Maynard, 1998a).

The air speed surveys representative of commercial facilities were fit to a lognormal distribution with the following parameter values: mean of 10.853 cm/s and standard deviation of 7.883 cm/s. In the model, the lognormal distribution is truncated at a maximum allowed value of 202.2 cm/s (largest surveyed mean air speed observed in (Baldwin and Maynard, 1998a) to prevent the model from sampling values that approach infinity or are otherwise unrealistically large.

(Baldwin and Maynard, 1998a) only presented the mean air speed of each survey. The authors did not present the individual measurements within each survey. Therefore, these distributions represent a distribution of mean air speeds and not a distribution of spatially-variable air speeds within a single workplace setting. However, a mean air speed (averaged over a work area) is the required input for the model.

M.2.5 Averaging Time

EPA is interested in estimating 8-hr TWAs for use in risk calculations; therefore, a constant averaging time of eight hours was used.

M.2.6 Use Rate

EPA used a top-down approach to estimate use rate based on the volume of TCE-based spotting agent sold in California and the number of textile cleaning facilities in California.

(IRTA, 2007) estimated 42,000 gal of TCE-based spotting agents are sold in California annually and there are approximately 5,000 textile cleaning facilities in California. This results in an average use rate of 8.4 gal/site-year of TCE-based spotting agents.

The study authors' review of safety data sheets identified TCE-based spotting agents contain 10% to 100% TCE.

M.2.7 Vapor Generation Rate

EPA set the vapor generation rate for spot cleaning (G) equal to the use rate of TCE with appropriate unit conversions. EPA multiplied the spotting agent use rate by the weight fraction of TCE (which ranges from 0.1 to 1) and assumed all TCE applied to the garment evaporates. EPA used a density of 1.46 g/cm^3 (U.S. EPA, 2018d). To calculate an hourly vapor generation rate, EPA divided the annual use rate by the number of operating days and the number of operating hours selected from their respective distributions for each iteration.

M.2.8 Operating Hours

(Morris and Wolf, 2005) surveyed dry cleaners in California, including their spotting labor. The authors developed two model plants: a small PERC dry cleaner that cleans 40,000 lb of clothes annually; and a large PERC dry cleaner that cleans 100,000 lb of clothes annually. The authors modeled the small dry

cleaner with a spotting labor of 2.46 hr/day and the large dry cleaner with a spotting labor of 5 hr/day. EPA models a uniform distribution of spotting labor varying from 2 to 5 hr/day.

M.2.9 Operating Days

EPA modeled the operating days per year using a triangular distribution from 250 to 312 days per year with a mode of 300 days per year.[24] The low-end operating days per year is based on the assumption that at a minimum the dry cleaner operates five days per week and 50 weeks per year. The mode of 300 days per year is based on an assumption that most dry cleaners will operate six days per week and 50 weeks per year. The high-end value is based on the assumption that the dry cleaner would operate at most six days per week and 52 weeks per year, assuming the dry cleaner is open year-round.

M.2.10 Fractional Number of Operating Days that a Worker Works

To account for lower exposure frequencies and part-time workers, EPA defines a fractional days of exposure as a uniform distribution ranging from 0.8 to 1.0. EPA expects a worker's annual working days may be less than the operating days based on BLS/Census data that showed the weighted average worked hours per year and per worker in the dry cleaning sector is approximately 1,600 (i.e., 200 day/yr at 8 hr/day) which falls outside the range of operating days per year used in the model (250 to 312 day/yr with mode of 300 day/yr).

The low end of the range, 0.8, was derived from the observation that the weighted average of 200 day/yr worked (from BLS/Census) is 80% of the standard assumption that a full-time worker works 250 day/yr. The maximum of 1.0 is appropriate as dry cleaners may be family owned and operated and some workers may work as much as every operating day. EPA defines the exposure frequency as the number of operating days (250 to 312 day/yr) multiplied by the fractional days of exposure (0.8 to 1.0).

[24] For modeling purposes, the minimum value was set to 249 days per year and the maximum to 313 days per year; however, these values have a probability of zero; therefore, the true range is from 250 to 312 days per year.

Appendix N BENCHMARK DOSE MODELING UPDATE FOR NESTED FETAL DATA FROM (Johnson et al., 2003)

BMD modeling of the nested fetal data for cardiac defects from (Johnson et al., 2003) was done to verify the BMD modeling results reported in Appendix F.4.2.1 of the EPA 2011 IRIS Toxicological Review for TCE Appendices (U.S. EPA, 2011e).

1) BMD modeling was performed using the nested logistic model in BMDS (v3.1.1) with and without a litter specific covariate to account for intra-litter similarity (litter effects) based on pre-treatment condition and with and without modeling of intra-litter correlation to account for intra-litter similarity based on effects during treatment. IRIS also used the nested logistic model with and without litter specific covariate and intra-litter correlation. Previous modeling from (U.S. EPA, 2011e) was performed with and without the high dose group dropped, however the model based on dropping the highest dose was used in the assessment because it had smaller scaled residuals and predicted expected response values were closer to observed. Therefore, current modeling was performed without the high dose group. Modeling in (U.S. EPA, 2011e) was performed using applied dose and two alternative internal dose metrics based on PBPK modeling (avg amount of TCE metabolized by oxidation/$kg^{3/4}$-day and AUC for TCE in blood). The same 3 sets of doses were modeled for the current effort. BMRs used for both the IRIS and current modeling were 10%, 5% and 1% extra risk.

2) Total weight gain during pregnancy (TWtGn) was used as the litter specific covariate in the modeling performed for the IRIS assessment. The individual animal data reasonably available for the current effort included TWtGn for the treated groups, but not for the control group. Based on the data available, litter size was used as the covariate for the current modeling effort instead of TWtGn.

3) P-values reported by an older version of the BMDS software as presented in Table F-6 (U.S. EPA, 2011e) for the nested models are incorrect, apparently due to a problem with the software used at that time, suggesting that the models did not have adequate fit to the data. The exercise reported in Section F.4.2.1.2 of (U.S. EPA, 2011e) was performed to show that the p-values were much higher than indicated in the raw modeling results and that model fit was acceptable. Calculation of p-values for the nested models in the current version of BMDS follows a bootstrap methodology similar to that described in Section F.4.2.1.2. of the IRIS assessment. Because the original p-values in presented in (U.S. EPA, 2011e) were incorrect, comparisons of current modeling results to IRIS were only made for AIC, BMD and BMDL. The p-values from the updated BMD modeling runs are presented for context.

4) In the previous BMD modeling, the best fitting model as determined by lowest AIC was the model without litter-specific covariate but with intra-litter correlation. This was true for the current modeling as well.

5) Results from the models without litter-specific covariate, including the best-fitting model, closely matched the results from the IRIS assessment (see Table_Apx N-1).

6) Results for the models that included the litter-specific covariate differed from the IRIS results, because a different covariate was used (litter size rather than TWtGn, due to missing data).

7) Model fits (AICs) and BMD/BMDL values are identical (within rounding error) between the updated modeling results and those reported in (U.S. EPA, 2011e).

Table_Apx N-1. Results for Best-Fitting Model in Comparison to Results Reported in IRIS (U.S. EPA, 2011e) (Highlighted)

Model	Covariate	Intra-litter Correlation	Dose Metric	BMR	AIC	p-value[d]	BMD	BMDL
Nested Logistic	Not Used	Modeled	Applied Dose[a]	0.10	243.815	0.665	0.71114	0.227675
					243.815	NR	0.71114	0.227675
				0.05	243.815	0.641	0.336856	0.107846
					243.815	NR	0.336856	0.107846
				0.01	243.815	0.661	0.064649	0.020698
					243.815	NR	0.064649	0.020698
			TotOxMetabBW34[b]	0.10	243.816	0.642	0.489388	0.156646
					243.815	NR	0.489442	0.156698
				0.05	243.816	0.642	0.231816	0.074201
					ND	NR	ND	ND
				0.01	243.816	0.636	0.04449	0.014241
					243.815	NR	0.0444948	0.0142453
			AUCCBld[c]	0.10	243.816	0.656	0.022279	0.00713
					243.816	NR	0.0222789	0.00712997
				0.05	243.816	0.656	0.010553	0.003377
					ND	NR	ND	ND
				0.01	243.816	0.656	0.002025	0.000648
					243.816	NR	0.00202535	.000648179

[a]0, 0.00045, 0.048, 0.218 mg/kg-day
[b]Total oxidative metabolism scaled by body weight to the ¾-power: 0, 0.00031, 0.033, 0.15
[c]AUC of TCE in blood: 0, 0.0000141, 0.00150254, 0.00682727
[d] p-values from the 2011 IRIS Assessment are not reported because the original values were incorrect.
ND = no data
NR = not relevant; original p-values as calculated by BMDS software in 2011 were incorrect

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Appendix O CONSIDERATIONS FOR BMD MODELING AND APPLICATION OF UNCERTAINTY FACTORS

A set of dose-response models were applied to empirically model the dose-response relationship in the range of the observed data. The models in EPA's Benchmark Dose Software were applied. Consistent with EPA's *Benchmark Dose Technical Guidance Document* (U.S. EPA, 2012a), the benchmark dose (BMD) and 95% lower confidence limit on the BMD (BMDL) were estimated using a benchmark response (BMR) to represent a minimal, biologically significant level of change, when possible. The BMR is represented by a specified percentage change, or relative deviation (RD), for continuous data. The BMR for dichotomous data is represented by a specified incidence, or extra risk (ER). In the absence of information regarding the level of change that was considered biologically significant, a BMR of 1 standard deviation (SD) from the control mean for continuous data or a BMR of 10% ER for dichotomous data was used to estimate the BMD and BMDL, and to facilitate a consistent basis of comparison across endpoints, studies, and assessments. Endpoint-specific BMRs are described further below. Where modeling was feasible, the estimated BMDLs were used as points of departure (PODs). Further details, including the modeling output and graphical results for the model selected for each endpoint, can be found in the 2011 EPA IRIS Assessment (U.S. EPA, 2011e) and Appendix G (for (Selgrade and Gilmour, 2010)). A comparison of results from updated BMDL modeling runs with results from (U.S. EPA, 2011e) for (Johnson et al., 2003) are provided in Appendix N. Where dose-response modeling was not feasible, NOAELs or LOAELs were also identified and are summarized.

O.1 Selecting the BMD model to use for POD computation

The following approach is recommended for selecting the model(s) to use for computing the BMDL to serve as the POD for a specific dataset according to EPA Benchmark Dose Guidance (U.S. EPA, 2012a).

1) Assess goodness-of-fit, using a value of $\alpha = 0.1$ to determine a critical value (or $\alpha = 0.05$ or $\alpha = 0.01$) if there is reason to use a specific model(s) rather than fitting a suite of models.

2) Further reject models that apparently do not adequately describe the relevant low- dose portion of the dose-response relationship, examining residuals and graphs of models and data.

3) As the remaining models have met the recommended default statistical criteria for adequacy and visually fit the data, any of them theoretically could be used for determining the BMDL. The remaining criteria for selecting the BMDL are necessarily somewhat arbitrary and are suggested as defaults.

4) If the BMDL estimates from the remaining models are sufficiently close (given the needs of the assessment), reflecting no particular influence of the individual models, then the model with the lowest Akaike's Information Criteria (AIC)[25] may be used to calculate the BMDL for the POD. This criterion is intended to help arrive at a single BMDL value in an objective, reproducible manner. If two or more models share the lowest AIC, the simple average or geometric mean of the BMDLs with the lowest AIC may be used. Note that this is not the same as "model averaging", which involves weighing a fuller set of adequately fitting models. In addition, such an average has drawbacks, including the fact that it is not a 95% lower bound (on the average BMD); it is just the average of the particular BMDLs under consideration (i.e., the average loses the statistical properties of the individual estimates).

[25] Akaike's Information Criteria—a measure of information loss from a dose-response model that can be used to compare a set of models. Among a specified set of models, the model with the lowest AIC is considered the best. If two or more models share the lowest AIC, an average of the BMDLs could be used, but averaging was not used in this assessment because for the one occasion in which models shared the lowest AIC, a selection was made based on visual fit.

5) If the BMDL estimates from the remaining models are not sufficiently close, some model dependence of the estimate can be assumed. Expert statistical judgment may help at this point to judge whether model uncertainty is too great to rely on some or all of the results. If the range of results is judged to be reasonable, there is no clear remaining biological or statistical basis on which to choose among them, and the lowest BMDL may be selected as a reasonable conservative estimate. Additional analysis and discussion might include consideration of additional models, the examination of the parameter values for the models used, or an evaluation of the BMDs to determine if the same pattern exists as for the BMDLs. Discussion of the decision procedure should always be provided.

6) In some cases, modeling attempts may not yield useful results. When this occurs and the most biologically relevant effect is from a study considered adequate but not amenable to modeling, the NOAEL (or LOAEL) could be used as the POD. The modeling issues that arose should be discussed in the assessment, along with the impacts of any related data limitations on the results from the alternate NOAEL/LOAEL approach.

O.2 Uncertainty Factor Selection

After the PODs were determined for each study/endpoint, uncertainty factors (UFs) were used to derive acceptable benchmark margins of mxposure (MOEs). UFs are used to address differences between study conditions and conditions of human environmental exposure. These include:

(a) *Extrapolating from laboratory animals to humans (UF_A):*
If a POD is derived from experimental animal data, it is divided by an UF to reflect pharmacokinetic and pharmacodynamic differences that may make humans more sensitive than laboratory animals. For oral exposures, the standard value for the interspecies UF is 10, which breaks down (approximately) to a factor of 3 for pharmacokinetic differences (which is removed if the PBPK model is used) and a factor of 3 for pharmacodynamic differences. For inhalation exposures, ppm equivalence across species is generally assumed or other cross-species scaling is performed, in accordance with U.S. EPA inhalation dosimetry guidance (U.S. EPA, 1994b), in which case, residual pharmacokinetic differences are considered to be negligible. Therefore, the standard value used for the interspecies UF is 3, which is ascribed to pharmacodynamic differences. These standard values were used for all of the PODs based on laboratory animal data in this assessment.

(b) *Human (intraspecies) variability (UF_H):*
Sensitive humans could be adversely affected at lower exposures than a general study population; consequently, PODs from general-population studies are divided by an UF to address sensitive humans. Similarly, the animals used in most laboratory animal studies are considered to be typical or average responders, and the human (intraspecies) variability UF is also applied to PODs from such studies to address sensitive subgroups. The standard value for the human variability UF is 10, which breaks down (approximately) to a factor of 3 for pharmacokinetic variability (which is removed if the PBPK model is used) and a factor of 3 for pharmacodynamic variability. This standard value was used for all of the PODs in this assessment.

(c) *Uncertainty in extrapolating from subchronic to chronic exposures (UF_S):*[26]
Chronic risk estimates apply to long-term exposure over decades, but sometimes the best (or only) reasonably available data come from less-than-lifetime studies. Lifetime exposure can induce effects

[26] Chronic exposure covers > 10% of expected lifetime. Rodent studies exceeding 90 days of exposure are considered chronic, and rodent studies covering from 4 weeks to 90 days of exposure are considered subchronic. For human studies, chronic exposure exceeds 7-8years, on average (U.S. EPA, 1994b).

that may not be apparent or as large in magnitude in a shorter study; consequently, a dose that elicits a specific level of response from a lifetime exposure may be less than the dose eliciting the same level of response from a shorter exposure period. Thus, PODs based on subchronic exposure data are generally divided by a subchronic-to-chronic UF, which has a standard value of 10. If there is evidence suggesting that exposure for longer time periods does not increase the magnitude of an effect, a lower value of 3 or one might be used. For some reproductive and developmental effects, chronic exposure is that which covers a specific window of exposure that is relevant for eliciting the effect, and subchronic exposure would correspond to an exposure that is notably less than the full window of exposure.

(d) *Uncertainty in extrapolating from LOAELs to NOAELs (UF_L):*

PODs are intended to be estimates of exposure levels without appreciable risk under the study conditions so that, after the application of appropriate UFs for interspecies extrapolation, human variability, and/or duration extrapolation, the absence of appreciable risk is conveyed. Under the NOAEL/LOAEL approach to determining a POD, however, adverse effects are sometimes observed at all study doses. If the POD is a LOAEL, then it is divided by an UF to better estimate a NOAEL. The standard value for the LOAEL-to-NOAEL UF is 10, although a value of 3 is sometimes used if the effect is considered minimally adverse at the response level observed at the LOAEL or is an early marker for an adverse effect. For NOAEL or BMDL values, the UF_L is 1.

Appendix P OCCUPATIONAL INHALATION EXPOSURE AND WATER RELEASE ASSESSMENT

P.1 Manufacturing

P.1.1 Exposure Assessment

EPA assessed inhalation exposures during manufacturing using identified inhalation exposure monitoring data. Table_Apx P-1 summarizes 8-hr TWA samples obtained from data submitted by the Halogenated Solvents Industry Alliance (HSIA) via public comment for one company (Halogenated Solvents Industry Alliance, 2018 5176415) listed as "Company B". HSIA also provided "General 12-hr" full-shift exposure data from "Company A". However, "Company A" data points were listed as "Not detected ≤0.062 ppm. Two additional studies with monitoring data for manufacturing were identified; however, the data from these studies were not used as the data were from China and almost 30 years old and are unlikely to be representative of current conditions at U.S. manufacturing sites. No data was found to estimate ONU exposures during TCE manufacturing. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.

Table_Apx P-1. Summary of Worker Inhalation Exposure Monitoring Data from TCE Manufacturing

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	2.59	0.86	0.59	0.30	16	High
Central Tendency	0.38	0.13	0.09	0.03		

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
Source: (Halogenated Solvents Industry Alliance, 2018 5176415)

P.1.2 Water Release Assessment

In general, potential sources of water releases in the chemical industry may include the following: equipment cleaning operations, aqueous wastes from scrubbers/decanters, reaction water, process water from washing intermediate products, and trace water settled in storage tanks (OECD, 2019). Based on the process for manufacturing TCE, EPA expects the sources of water releases to be from aqueous wastes from decanters used to separate catalyst fines, caustic neutralizer column, and caustic scrubbers;

and water removed from the TCE product in drying columns (Most, 1989). Additional water releases may occur if a site uses water to clean process equipment; however, EPA does not expect this to be a primary source of water releases from manufacturing sites as equipment cleaning is not expected to occur daily and manufacturers would likely use an organic solvent to clean process equipment.

Of the five manufacturing sites assessed, three reported in the 2016 TRI (one of these three sites reported zero water releases to TRI). Additionally, one of these sites also reported to 2016 DMR. For the sites that reported water releases, EPA assessed water releases as reported in the 2016 TRI and 2016 DMR. For the remaining two sites, EPA assessed water releases at the maximum daily and maximum average monthly concentrations allowed under the Organic Chemicals, Plastics and Synthetic Fibers (OCPSF) Effluent Guidelines (EG) and Standards (40 C.F.R. Part 414) (U.S. EPA, 2019g). The OCPSF EG applies to facilities classified under the following SIC codes:

- 2821—Plastic Materials, Synthetic Resins, and Nonvulcanizable Elastomers;
- 2823—Cellulosic Man-Made Fibers;
- 2865—Cyclic Crudes and Intermediates, Dyes, and Organic Pigments; and
- 2869—Industrial Organic Chemicals, Not Elsewhere Classified.

Manufacturers of TCE would typically be classified under SIC code 2869; therefore, the requirements of the OCPSF EG apply to these sites. Subparts I, J, and K of the OCPSF EG set limits for the concentration of TCE in wastewater effluents for industrial facilities that are direct discharge point sources using end-of-pipe biological treatment, direct discharge point sources that do not use end-of-pipe biological treatment, and indirect discharge point sources, respectively 40 C.F.R. Part 414 (U.S. EPA, 2019g). Direct dischargers are facilities that discharge effluents directly to surface waters and indirect dischargers are facilities that discharge effluents to publicly-owned treatment works (POTW). The OCPSF limits for TCE are provided in Table_Apx P-2.

Table_Apx P-2. Summary of OCPSF Effluent Limitations for Trichloroethylene

OCPSF Subpart	Maximum for Any One Day (µg/L)	Maximum for Any Monthly Average (µg/L)	Basis
Subpart I – Direct Discharge Point Sources That Use End-of-Pipe Biological Treatment	54	21	BAT effluent limitations and NSPS
Subpart J – Direct Discharge Point Sources That Do Not Use End-of-Pipe Biological Treatment	69	26	BAT effluent limitations and NSPS
Subpart K – Indirect Discharge Point Sources	69	26	Pretreatment Standards for Existing Sources (PSES) and Pretreatment Standards for New Sources (PSNS)

BAT = Best Available Technology Economically Achievable; NSPS = New Source Performance Standards; PSES = Pretreatment Standards for Existing Sources; PSNS = Pretreatment Standards for New Sources.
Source: (U.S. EPA, 2019g)

EPA did not identify TCE-specific information on the amount of wastewater produced per day. The Specific Environmental Release Category (SpERC) developed by the European Solvent Industry Group for the manufacture of a substance estimates 10 m^3 of wastewater generated per metric ton of substance produced (ESIG, 2012). In lieu of TCE-specific information, EPA estimated water releases using the SpERC specified wastewater production volume and the annual TCE production rates from each facility.

EPA estimated both a maximum daily release and an average daily release using the OCPSF EG limitations for TCE for maximum on any one day, and maximum for any monthly average, respectively. Prevalence of end-of-pipe biological treatment at TCE manufacturing sites is unknown; therefore, EPA used limitations for direct discharges with no end-of-pipe biological treatment and indirect dischargers to address the uncertainty at these sites. EPA estimated annual releases from the average daily release and assuming 350 days/yr of operation.[27]

Table_Apx P-3 summarizes water releases from the manufacturing process for sites reporting to TRI and Table_Apx P-4 summarizes water releases from sites not reporting to TRI. The estimated total annual release across all sites is 60.5 – 453.6 kg/yr discharged to surface water or POTWs.

Table_Apx P-3. Reported Water Releases of Trichloroethylene from Manufacturing Sites Reporting to 2016 TRI

Site	Annual Release[a] (kg/site-yr)	Annual Release Days (days/yr)	Average Daily Release[a] (kg/site-day)	NPDES Code	Release Media
Olin Blue Cube, Freeport, TX	24	350	0.07	TX0059447	non-POTW WWT
Geon Oxy Vinyl Laporte Plant, Laporte, TX	0	N/A	0	TX0070416	N/A
Axiall Corporation dba Eagle US 2 LLC, Westlake, LA[b]	49.9-443[c]	350	0.14-1.27	LA0000761[d]	Surface Water

POTW = Publicly-Owned Treatment Works; WWT = Wastewater Treatment; N/A = Not applicable

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 300 days of operation per year.

[b] Axiall was purchased by Westlake Chemical in 2016. The site at 1300 PPG Drive Westlake, LA dba Eagle US 2 LLC.

[c] First value based on 2016 TRI, second value based on 2016 DMR data (U.S. EPA, 2016a).

[d] Based on Eagle US 2 LLC NPDES Permit provided in DMR Data (U.S. EPA, 2016a).

[27] Due to large throughput, manufacturing sites are assumed to operate seven days per week and 50 weeks per year with two weeks per year for shutdown activities.

Table_Apx P-4. Estimated Water Releases of Trichloroethylene from Manufacturing Sites Not Reporting to 2016 TRI

Site	Annual Operating Days (days/yr)	Daily Production Volume[a] (kg/site-day)	Daily Wastewater Flow[b] (L/site-day)	Maximum Daily Release[c] (kg/site-day)	Average Daily Release[d] (kg/site-day)	Average Annual Release[e] (kg/site-yr)	NPDES Code	Release Media
Solvents & Chemicals, Pearland, TX	350	58,234	582,345	0.04	0.02	5.3	Not available	Surface Water or POTW
Occidental Chemical Corp. Wichita, KS	350	58,234	582,345	0.04	0.02	5.3	Not available	Surface Water or POTW

POTW = Publicly-Owned Treatment Works

[a] Daily production volume calculated using the annual production volume and dividing by the annual operating days per year (300 days/yr).

[b] The estimated wastewater flow rate is calculated assuming 10 m^3 of wastewater is produced per metric ton of TCE produced (equivalent to 10 L wastewater/kg of TCE) based on the SpERC for the manufacture of a substance (ESIG, 2012).

[c] The maximum daily release is calculated using the maximum daily concentration from the OCPSF EG, 26 µg/L, and multiplying by the daily wastewater flow.

[d] The average daily release is calculated using the maximum monthly average concentration from the OCPSF EG, 69 µg/L, and multiplying by the daily wastewater flow.

[e] The average annual release is calculated as the maximum monthly average concentration multiplied by the daily wastewater production, and 350 operating days/year.

P.2 Processing as a Reactant

P.2.1 Exposure Assessment

EPA did not identify inhalation exposure monitoring data related processing TCE as a reactant. Therefore, EPA used monitoring data from the manufacture of TCE as surrogate. EPA believes the handling and TCE concentrations for both conditions of use to be similar. However, EPA is unsure of the representativeness of these surrogate data toward actual exposures to TCE at all sites covered by this condition of use.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

The surrogate data was obtained from (HSIA) via public comment (Halogenated Solvents Industry Alliance, 2018 5176415), presented in Table_Apx P-5 below. No data was found to estimate ONU exposures during use of TCE as a reactant. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

Table_Apx P-5. Summary of Worker Inhalation Exposure Surrogate Monitoring Data from TCE Use as a Reactant

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Associated Air Concentration Data
High-End	2.59	0.86	0.59	0.30	16	Medium
Central Tendency	0.38	0.13	0.09	0.03		

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

P.2.2 Water Release Assessment

In general, potential sources of water releases in the chemical industry may include the following: equipment cleaning operations, aqueous wastes from scrubbers/decanters, reaction water, process water from washing intermediate products, and trace water settled in storage tanks (OECD, 2019). Based on the use as a reactant, EPA expects minimal sources of TCE release to water.

Two of the three sites reporting to TRI did not report any water releases of TCE; the other TRI site reported 13 lb/yr (5.9 kg/yr) released to water. For the two sites found through DMR data, total water releases were calculated to be approximately 11 lb/yr (5 kg/yr). Based on the information for these 5 sites, an average annual release of approximately 2.2 kg/site-yr was calculated. Using this estimate, and assuming 440 sites as a high-end estimate, the total TCE water discharge from these 440 sites equal approximately 968 kg/yr. Table_Apx P-6 summarizes the low and high end water release estimates.

Table_Apx P-6. Water Release Estimates for Sites Using TCE as a Reactant

Number of Sites	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
Low End Number of Sites					
Arkema Inc., Calvert City, KY	5.9	350	0.02	KY0003603	Surface Water
Honeywell International - Geismar Complex, Geismar, LA	4.5	350	0.01	LA0006181	Surface Water
Praxair Technology Center, Tonawanda, NY	0.6	350	1.7E-03	NY0000281	Surface Water
High End Number of Sites					
440 unknown sites	2.2[a]	350	6.3E-03	N/A	Surface Water or POTW

[a] Calculated from the total yearly water releases of TCE from DMR and TRI data, and diving by the number of reporting sites (5 sites). Mexichem Fluor Inc. and Halocarbon Products Corp reported no water releases to TRI.

P.3 Formulation of Aerosol and Non-Aerosol Products

P.3.1 Exposure Assessment

EPA did not identify inhalation exposure monitoring data related using TCE when formulating aerosol and non-aerosol products. Therefore, EPA used monitoring data from repackaging as a surrogate, as EPA believes the handling and TCE concentrations for both conditions of use to be similar. However, EPA is unsure of the representativeness of these surrogate data toward actual exposures to TCE at all sites covered by this condition of use.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 33 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium.

Table_Apx P-7 summarizes the 8-hr TWA from monitoring data from unloading/loading TCE from bulk containers. The data were obtained from a Chemical Safety Report (DOW Deutschland, 2014b). No data was found to estimate ONU exposures during formulation of aerosol and non-aerosol products. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

Table_Apx P-7. Summary of Worker Inhalation Exposure Monitoring Data for Unloading TCE During Formulation of Aerosol and Non-Aerosol Products

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	1.1	0.4	0.3	0.1	33	Medium
Central Tendency	4.9E-4	1.6E-4	1.1E-4	4.5E-5		

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

P.3.2 Water Release Assessment

In general, potential sources of water releases in the chemical industry may include the following: equipment cleaning operations, aqueous wastes from scrubbers/decanters, reaction water, process water from washing intermediate products, and trace water settled in storage tanks (OECD, 2019). Based on the use in formulations and the amount of TCE used for this condition of use, EPA expects minimal sources of TCE release to water.

None of the sites reporting to TRI reported any water releases of TCE. All releases were to off-site land, incineration or recycling. Based on this information, EPA does not have enough information to estimate water releases of TCE for this condition of use.

P.4 Repackaging

P.4.1 Exposure Assessment

EPA identified inhalation exposure monitoring data related unloading/loading TCE into/from bulk transport containers. Table_Apx P-8 summarizes the 8-hr TWA from monitoring data from unloading/loading TCE from bulk containers. The data were obtained from a Chemical Safety Report (DOW Deutschland, 2014b). It should be noted that this study indicates that the filling system uses a "largely automated process" (DOW Deutschland, 2014b). Therefore, EPA is unsure of the representativeness of these data toward actual exposures to TCE for all sites covered by this condition of use.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 33 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.

No data was found to estimate ONU exposures during formulation of aerosol and non-aerosol products. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

Table_Apx P-8. Summary of Worker Inhalation Exposure Monitoring Data for Unloading/Loading TCE from Bulk Containers

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	1.1	0.4	0.26	0.1	33	Medium to High
Central Tendency	4.9E-4	1.6E-4	1.1E-4	4.5E-5		

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

P.4.2 Water Release Assessment

EPA expects the primary source of water releases from repackaging activities to be from the use of water or steam to clean bulk containers used to transport TCE or products containing TCE. EPA expects the use of water/steam for cleaning containers to be limited at repackaging sites as TCE is an organic substance and classified as a hazardous waste under RCRA. EPA expects the majority of sites to use organic cleaning solvents which would be disposed of as hazardous waste (incineration or landfill) over water or steam.

Water releases during repackaging were assessed using data reported in the 2016 DMR and 2016 TRI. One of the 20 sites reporting to TRI reported water releases of TCE to off-site wastewater treatment. All other sites reporting to TRI reported releases to off-site land or incineration. EPA assessed annual

releases as reported in the 2016 DMR and assessed daily releases by assuming 250 days of operation per year. A summary of the water releases reported to the 2016 DMR and TRI can be found in Table_Apx P-9.

Table_Apx P-9. Reported Water Releases of Trichloroethylene from Sites Repackaging TCE

Site Identity	Annual Release (kg/site-yr)[a]	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	NPDES Code	Release Media
Hubbard-Hall Inc, Waterbury, CT	277	250	1.1	Not available	Non-POTW WWT
St. Gabriel Terminal, Saint Gabriel, LA	1.4	250	5.5E-03	LA0052353	Surface Water
Vopak Terminal Westwego Inc, Westwego, LA	1.2	250	4.7E-03	LA0124583	Surface Water
Oiltanking Houston Inc, Houston, TX	0.8	250	3.3E-03	TX0091855	Surface Water
Research Solutions Group Inc, Pelham, AL	0.01	250	3.3E-05	AL0074276	Surface Water
Carlisle Engineered Products Inc, Middlefield, OH	1.7E-3	250	6.8E-06	OH0052370	Surface Water

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 250 days of operation per year.
Sources: (U.S. EPA, 2016a) and (U.S. EPA, 2017c)

P.5 Batch Open Top Vapor Degreasing

P.5.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from NIOSH investigations at twelve sites using TCE as a degreasing solvent in OTVDs. Due to the large variety in shop types that may use TCE as a vapor degreasing solvent, it is unclear how representative these data are of a "typical" shop. Therefore, EPA supplemented the identified monitoring data using the Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model. The following subsections detail the results of EPA's occupational exposure assessment for batch open-top vapor degreasing based on inhalation exposure monitoring data and modeling.

Table_Apx P-10 summarizes the 8-hr TWA monitoring data for the use of TCE in OTVDs. The data were obtained from NIOSH Health Hazard Evaluation reports (HHEs). NIOSH HHEs are conducted at the request of employees, employers, or union officials, and provide information on existing and potential hazards present in the workplaces evaluated (Daniels et al., 1988), (Ruhe et al., 1981), (Barsan, 1991), (Ruhe, 1982), (Rosensteel and Lucas, 1975), (Seitz and Driscoll, 1989), (Gorman et al., 1984), (Gilles et al., 1977), (Vandervort and Polakoff, 1973), and (Lewis, 1980).

Data from these sources cover exposures at several industries including metal tube production, valve manufacturing, jet and rocket engine manufacture, air conditioning prep and assembly, and AC motor parts (Ruhe et al., 1981), (Barsan, 1991), (Rosensteel and Lucas, 1975), (Gorman et al., 1984), (Vandervort and Polakoff, 1973), and (Lewis, 1980). Except for one site, sample times ranged from approximately five to eight hours (Ruhe et al., 1981), (Barsan, 1991), (Rosensteel and Lucas, 1975), (Gorman et al., 1984), and (Lewis, 1980). The majority of samples taken at the other site were taken for 2 hours or less (Vandervort and Polakoff, 1973). Where sample times were less than eight hours, EPA converted to an 8-hr TWA assuming exposure outside the sample time was zero. For sample times

greater than eight hours, EPA left the measured concentration as is. It should be noted that additional sources for degreasing were identified but were not used in EPA's analysis as they either: 1) did not specify the machine type in use; or 2) only provided a statistical summary of worker exposure monitoring.

Table_Apx P-10. Summary of Worker Inhalation Exposure Monitoring Data for Batch Open-Top Vapor Degreasing

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
Workers						
High-End	77.8	25.9	17.8	9.1	113	Medium
Central Tendency	13.8	4.6	3.2	1.3		
Occupational non-users						
High-End	9.1	3.0	2.1	1.1	10	Medium
Central Tendency	1.1	0.4	0.3	0.1		

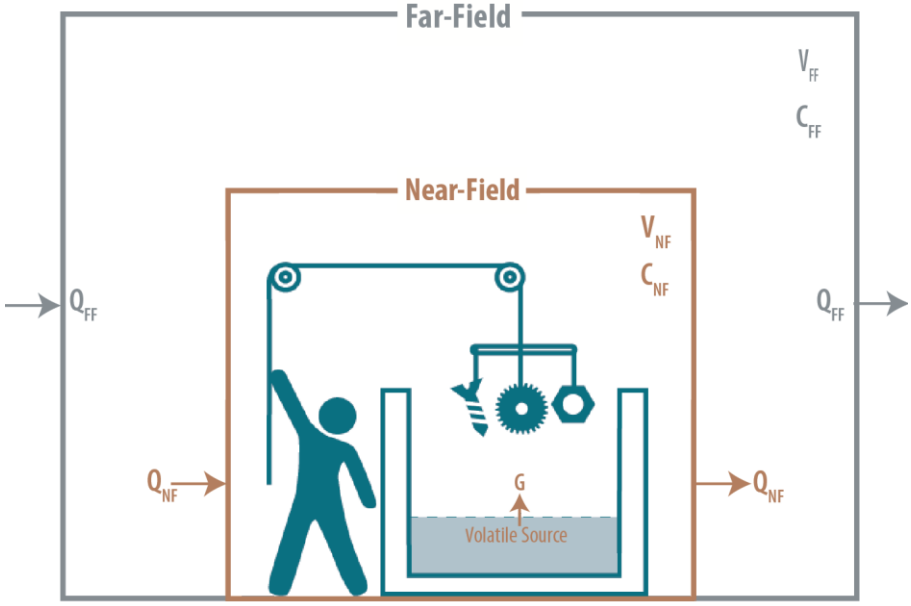
AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 123 data points from 16 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium.

EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that the underlying methodologies used to estimate these emissions in the 2014 NEI are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Figure_Apx P-1 illustrates the near-field/far-field model that can be applied to open-top vapor degreasing (AIHA, 2009). As the figure shows, volatile TCE vapors evaporate into the near-field, resulting in worker exposures at a concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, G, into the near-field, whose volume is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field, resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings,

denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outside air.



Figure_Apx P-1. Schematic of the Open-Top Vapor Degreasing Near-Field/Far-Field Inhalation Exposure Model

To estimate the TCE vapor generation rate, the model developed a distribution from the reported annual emission rates and annual operating times reported in the 2014 NEI. NEI records where the annual operating time was not reported were excluded from the distribution.

Batch degreasers are assumed to operate between two and 24 hours per day, based on NEI data on the reported operating hours for OTVD using TCE. EPA performed a Monte Carlo simulation with 100,000 iterations and the Latin Hypercube sampling method in @Risk to calculate 8-hour TWA near-field and far-field exposure concentrations. Near-field exposure represents exposure concentrations for workers who directly operate the vapor degreasing equipment, whereas far-field exposure represents exposure concentrations for occupational non-users (i.e., workers in the surrounding area who do not handle the degreasing equipment).

Table_Apx P-11 presents a statistical summary of the exposure modeling results. These exposure estimates represent modeled exposures for the workers and occupational non-users. For workers, the 50th percentile exposure is 34.8 ppm 8-hr TWA, with a 95th percentile of 388 ppm 8-hr TWA.

Both of these values are an order of magnitude higher than identified in 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. It is also uncertain of the underlying methodologies used to estimate emissions in the 2014 NEI data.

Table_Apx P-11. Summary of Exposure Modeling Results for TCE Degreasing in OTVDs

Percentile	8-hr TWA (ppm)	AC[a] (ppm)	ADC (ppm)	LADC (ppm)	Confidence Rating of Air Concentration Data
Workers (Near-field)					
High-End	388	129.3	88.5	35.3	N/A – Modeled Data
Central Tendency	34.8	79.0	8.0	3.0	
Occupational non-users (Far-Field)					
High-End	237	79.0	54.0	21.1	N/A – Modeled Data
Central Tendency	18.1	6.0	4.1	1.5	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] Acute exposures calculated as a 24-hr TWA.

P.5.2 Water Release Assessment

The primary source of water releases from OTVDs is wastewater from the water separator. Water in the OTVD may come from two sources: 1) Moisture in the atmosphere that condenses into the solvent when exposed to the condensation coils on the OTVD; and/or 2) steam used to regenerate carbon adsorbers used to control solvent emissions on OTVDs with enclosures (Durkee, 2014; Kanegsberg and Kanegsberg, 2011; NIOSH, 2002a, b, c, d). The water is removed in a gravity separator and sent for disposal (NIOSH, 2002a, b, c, d). The current disposal practices of the wastewater are unknown; however, a 1982 EPA (Gilbert et al., 1982) report estimated 20% of water releases from metal cleaning (including batch systems, conveyorized systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

Water releases for OTVDs were assessed using data reported in the 2016 TRI and 2016 DMR. Due to limited information in these reporting programs, these sites may in fact not operate OTVDs, but may operate other solvent cleaning machines or perform metalworking activities. They are included in the OTVD assessment as EPA expects OTVDs to be the most likely condition of use. EPA assessed annual releases as reported in the 2016 TRI or 2016 DMR and assessed daily releases by assuming 260 days of operation per year, as recommended in the 2017 ESD on Use of Vapor Degreasers, and averaging the annual releases over the operating days. A summary of the water releases reported to the 2016 TRI and DMR can be found in Table_Apx P-12.

Table_Apx P-12. Reported Water Releases of Trichloroethylene from Sites Using TCE in Open-Top Vapor Degreasing

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
US Nasa Michoud Assembly Facility, New Orleans, LA	509	260	1.96	LA0052256	Surface Water
GM Components Holdings LLC, Lockport, NY	34.2	260	0.13	NY0000558	Surface Water
Akebono Elizabethtown Plant, Elizabethtown, KY	17.9	260	0.07	KY0089672	Surface Water

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
Delphi Harrison Thermal Systems, Dayton, OH	9.3	260	0.04	OH0009431	Surface Water
Chemours Company Fc LLC, Washington, WV	6.7	260	0.03	WV0001279	Surface Water
Equistar Chemicals LP, La Porte, TX	4.4	260	0.02	TX0119792	Surface Water
GE Aviation, Lynn, MA	2.6	260	0.01	MA0003905	Surface Water
Certa Vandalia LLC, Vandalia, OH	2.1	260	0.01	OH0122751	Surface Water
GM Components Holdings LLC Kokomo Ops, Kokomo, IN	1.7	260	0.01	IN0001830	Surface Water
Amphenol Corp-Aerospace Operations, Sidney, NY	1.6	260	0.01	NY0003824	Surface Water
Emerson Power Trans Corp, Maysville, KY	1.6	260	0.01	KY0100196	Surface Water
Olean Advanced Products, Olean, NY	1.4	260	0.01	NY0073547	Surface Water
Texas Instruments, Inc., Attleboro, MA	1.3	260	5.18E-03	MA0001791	Surface Water
Hollingsworth Saco Lowell, Easley, SC	1.2	260	4.69E-03	SC0046396	Surface Water
Trelleborg YSH Incorporated Sandusky Plant, Sandusky, MI	0.9	260	3.60E-03	MI0028142	Surface Water
Timken Us Corp Honea Path, Honea Path, SC	0.9	260	3.55E-03	SC0047520	Surface Water
Johnson Controls Incorporated, Wichita, KS	0.6	260	2.28E-03	KS0000850	Surface Water
Accellent Inc/Collegeville Microcoax, Collegeville, PA	0.6	260	2.22E-03	PA0042617	Surface Water
National Railroad Passenger Corporation (Amtrak) Wilmington Maintenance Facility, Wilmington, DE	0.5	260	2.03E-03	DE0050962	Surface Water
Electrolux Home Products (Formerly Frigidaire), Greenville, MI	0.5	260	2.01E-03	MI0002135	Surface Water
Rex Heat Treat Lansdale Inc, Lansdale, PA	0.5	260	1.94E-03	PA0052965	Surface Water
Carrier Corporation, Syracuse, NY	0.5	260	1.77E-03	NY0001163	Surface Water
Globe Engineering Co Inc, Wichita, KS	0.5	260	1.74E-03	KS0086703	Surface Water
Cascade Corp (0812100207), Springfield, OH	0.3	260	1.17E-03	OH0085715	Surface Water
USAF-Wurtsmith AFB, Oscoda, MI	0.3	260	1.15E-03	MI0042285	Surface Water
AAR Mobility Systems, Cadillac, MI	0.3	260	1.12E-03	MI0002640	Surface Water
Eaton Mdh Company Inc, Kearney, NE	0.3	260	1.07E-03	NE0114405	Surface Water
Motor Components L C, Elmira, NY	0.3	260	9.64E-04	NY0004081	Surface Water
Salem Tube Mfg, Greenville, PA	0.233	260	8.97E-04	PA0221244	Surface Water

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
Ametek Inc. U.S. Gauge Div., Sellersville, PA	0.227	260	8.72E-04	PA0056014	Surface Water
GE (Greenville) Gas Turbines LLC, Greenville, SC	0.210	260	8.06E-04	SC0003484	Surface Water
Parker Hannifin Corporation, Waverly, OH	0.194	260	7.47E-04	OH0104132	Surface Water
Mahle Enginecomponents USA Inc, Muskegon, MI	0.193	260	7.42E-04	MI0004057	Surface Water
General Electric Company - Waynesboro, Waynesboro, VA	0.191	260	7.33E-04	VA0002402	Surface Water
Gayston Corp, Dayton, OH	0.167	260	6.43E-04	OH0127043	Surface Water
Styrolution America LLC, Channahon, IL	0.166	260	6.37E-04	IL0001619	Surface Water
Remington Arms Co Inc, Ilion, NY	0.159	260	6.12E-04	NY0005282	Surface Water
Lake Region Medical, Trappe, PA	0.1	260	5.06E-04	Not available	Surface Water
United Technologies Corporation, Pratt And Whitney Division, East Hartford, CT	0.1	260	4.80E-04	CT0001376	Surface Water
Atk-Allegany Ballistics Lab (Nirop), Keyser, WV	0.1	260	4.70E-04	WV0020371	Surface Water
Techalloy Co Inc, Union, IL	0.1	260	4.27E-04	IL0070408	Surface Water
Owt Industries, Pickens, SC	0.1	260	3.14E-04	SC0026492	Surface Water
Boler Company, Hillsdale, MI	0.1	260	2.69E-04	MI0053651	Surface Water
Mccanna Inc., Carpentersville, IL	0.1	260	2.68E-04	IL0071340	Surface Water
Cutler Hammer, Horseheads, NY	0.1	260	2.38E-04	NY0246174	Surface Water
Sperry & Rice Manufacturing Co LLC, Brookville, IN	8.54E-02	260	3.28E-04	IN0001473	Surface Water
US Air Force Offutt Afb Ne, Offutt A F B, NE	4.14E-02	260	1.59E-04	NE0121789	Surface Water
Troxel Company, Moscow, TN	3.49E-02	260	1.34E-04	TN0000451	Surface Water
Austin Tube Prod, Baldwin, MI	2.96E-02	260	1.14E-04	MI0054224	Surface Water
LS Starrett Precision Tools, Athol, MA	2.65E-02	260	1.02E-04	MA0001350	Surface Water
Avx Corp, Raleigh, NC	2.30E-02	260	8.83E-05	NC0089494	Surface Water
Handy & Harman Tube Co/East Norriton, Norristown, PA	1.61E-02	260	6.17E-05	PA0011436	Surface Water
Indian Head Division, Naval Surface Warfare Center, Indian Head, MD	1.08E-02	260	4.16E-05	MD0003158	Surface Water
General Dynamics Ordnance Tactical Systems, Red Lion, PA	6.34E-03	260	2.44E-05	PA0043672	Surface Water
Trane Residential Solutions - Fort Smith, Fort Smith, AR	3.46E-03	260	1.33E-05	AR0052477	Surface Water
Lexmark International Inc., Lexington, KY	3.23E-03	260	1.24E-05	KY0097624	Surface Water
Alliant Techsystems Operations LLC, Elkton, MD	3.02E-03	260	1.16E-05	MD0000078	Surface Water

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
Daikin Applied America, Inc. (Formally Mcquay International), Scottsboro, AL	2.15E-03	260	8.26E-06	AL0069701	Surface Water
Beechcraft Corporation, Wichita, KS	2.04E-03	260	7.86E-06	KS0000183	Surface Water
Federal-Mogul Corp, Scottsville, KY	1.50E-03	260	5.78E-06	KY0106585	Surface Water
Cessna Aircraft Co (Pawnee Facility), Wichita, KS	1.36E-03	260	5.24E-06	KS0000647	Surface Water
N.G.I, Parkersburg, WV	3.43E-04	260	1.32E-06	WV0003204	Surface Water
Hyster-Yale Group, Inc, Sulligent, AL	2.35E-04	260	9.03E-07	AL0069787	Surface Water
Hitachi Electronic Devices (USA), Inc., Greenville, SC	6.58E-05	260	2.53E-07	SC0048411	Surface Water

WWT = Wastewater Treatment

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 260 days of operation per year.

Sources: 2016 TRI (U.S. EPA, 2017c); 2016 DMR (U.S. EPA, 2016a)

Data from TRI and DMR may not represent the entirety of sites using TCE in OTVDs. EPA did not identify other data sources to estimate water releases from sites not reporting to TRI or DMR. However, sites operating degreasers are regulated by the following national ELGs:

- Electroplating Point Source Category Subparts A, B, D, E, F, G, and H (U.S. EPA, 2019d);[28]
- Iron and Steel Manufacturing Point Source Category Subpart J (U.S. EPA, 2019e);
- Metal Finishing Point Source Category Subpart A (U.S. EPA, 2019f);[29]
- Coil Coating Point Source Category Subpart D (U.S. EPA, 2019b);
- Aluminum Forming Point Source Category Subparts A, B, C, D, E, and F (U.S. EPA, 2019a); and
- Electrical and Electronic Components Point Source Category Subparts A and B (U.S. EPA, 2019c).

All above ELGs set discharges limits based on the total toxic organics (TTO) concentration in the wastewater stream and not a specific TCE limit. TTO is the summation of the concentrations for a specified list of pollutants which may be different for each promulgated ELG and includes TCE for the above referenced ELGs. Therefore, the concentration of TCE in the effluent is expected to be less than the TTO limit.

The operation of the water separator via gravity separation is such that the maximum concentration of TCE leaving the OTVD is equal to the solubility of TCE in water, 1,280 mg/L (Durkee, 2014). In cases where this concentration exceeds the limit set by the applicable ELGs, EPA expects sites will perform some form of wastewater treatment for the effluent stream leaving the OTVD to ensure compliance with

[28] The Electroplating ELG applies only to sites that discharge to POTW (indirect discharge) that were in operation before July 15, 1983. Processes that began operating after July 15, 1983 and direct dischargers are subject to the Metal Finishing ELG (40 C.F.R Part 433).

[29] The Metal Finishing ELG do not apply when wastewater discharges from metal finishing operations are already regulated by the Iron and Steel, Coil Coating, Aluminum Forming, or Electrical and Electronic Components ELGs.

the ELG prior to discharge. EPA did not identify information on the amount of wastewater generated from OTVDs to estimate releases from sites not reporting to TRI or DMR.

P.6 Batch Closed-Loop Vapor Degreasing

P.6.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from a European Chemical Safety report using TCE in closed degreasing operations. However, it is unclear how representative these data are of a "typical" batch closed-loop degreasing shop. Table_Apx P-13 summarizes the 8-hr TWA monitoring data for the use of TCE in vapor degreasers. The data were obtained from a Chemical Safety Report (DOW Deutschland, 2014a).

Data from these sources cover exposures at several industries where industrial parts cleaning occurred using vapor degreasing in closed systems. It should be noted that additional sources for degreasing were identified but were not used in EPA's analysis as they either: 1) did not specify the machine type in use; or 2) only provided a statistical summary of worker exposure monitoring.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 19 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to high.

Table_Apx P-13. Summary of Worker Inhalation Exposure Monitoring Data for Batch Closed-Loop Vapor Degreasing

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	1.4	0.5	0.3	0.2	19	High
Central Tendency	0.5	0.2	0.1	0.04		

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

P.6.2 Water Release Assessment

Similar to OTVDs, the primary source of water releases from closed-loop systems is wastewater from the water separator. However, unlike OTVDs, no water is expected to enter the system through condensation (Durkee, 2014). The reason for this is that enclosed systems flush the work chamber with water-free vapor (typically nitrogen gas) after the parts to be cleaned are added to the chamber and the chamber is sealed but before the solvent enters (Durkee, 2014). Multiple flushes can be performed to reduce the concentration of water to acceptable levels prior to solvent cleaning (Durkee, 2014). Therefore, the primary source of water in closed-loop systems is from steam used to regenerate carbon adsorbers (Durkee, 2014; Kanegsberg and Kanegsberg, 2011; NIOSH, 2002a, b, c, d). Similar to

OTVDs, the water is removed in a gravity separator and sent for disposal (NIOSH, 2002a, b, c, d). As indicated in the OTVD assessment, current disposal practices of the wastewater are unknown with the latest available data from a 1982 EPA (Gilbert et al., 1982) report estimating 20% of water releases were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

EPA assumes the TRI and DMR data cover all water discharges of TCE from closed-loop vapor degreasing. However, EPA cannot distinguish between degreaser types in TRI and DMR data; therefore, a single set of water release for all degreasing operations is used for OTVDs.

P.7 Conveyorized Vapor Degreasing

P.7.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from NIOSH investigations at two sites using TCE in conveyorized degreasing. Due to the large variety in shop types that may use TCE as a vapor degreasing solvent, it is unclear how representative these data are of a "typical" shop. Therefore, EPA supplemented the identified monitoring data using the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model. The following subsections detail the results of EPA's occupational exposure assessment for batch open-top vapor degreasing based on inhalation exposure monitoring data and modeling.

Table_Apx P-14 summarizes the 8-hr TWA monitoring data for the use of TCE in conveyorized degreasing. The data were obtained from two NIOSH Health Hazard Evaluation reports (HHEs) (Crandall and Albrecht, 1989), (Kinnes, 1998).

Table_Apx P-14. Summary of Worker Inhalation Exposure Monitoring Data for Conveyorized Vapor Degreasing

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	48.3	16.1	11.0	5.6	18	Medium
Central Tendency	32.4	10.8	7.4	2.9		

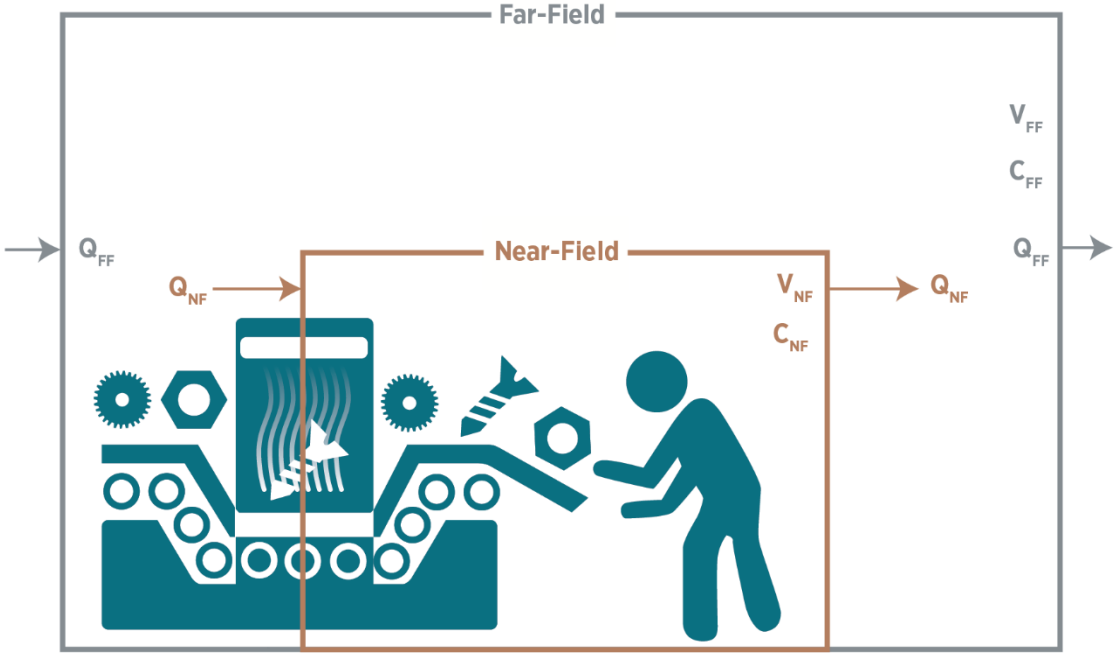
AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 18 data points from 2 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of

inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for three total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Figure_Apx P-2 illustrates the near-field/far-field model that can be applied to conveyorized vapor degreasing. As the figure shows, TCE vapors evaporate into the near-field (at evaporation rate G), resulting in near-field exposures to workers at a concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, G, into the near-field, whose volume is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outdoor air.



Figure_Apx P-2. Belt/Strip Conveyorized Vapor Degreasing Schematic of the Conveyorized Degreasing Near-Field/Far-Field Inhalation Exposure Model

To estimate the TCE vapor generation rate, the model uses the annual emission rate and annual operating time from the single conveyorized degreasing unit reported in the 2014 NEI. Because the vapor generation rate is based a limited data set, it is unknown how representative the model is of a "typical" conveyorized degreasing site.

EPA performed a Monte Carlo simulation with 100,000 iterations and the Latin Hypercube sampling method in @Risk to calculate 8-hour TWA near-field and far-field exposure concentrations. Near-field exposure represents exposure concentrations for workers who directly operate the vapor degreasing equipment, whereas far-field exposure represents exposure concentrations for occupational non-users (i.e., workers in the surrounding area who do not handle the degreasing equipment).

Table_Apx P-15 presents a statistical summary of the exposure modeling results. These exposure estimates represent modeled exposures for the workers and occupational non-users. For workers, the 50th percentile exposure is 40.8 ppm 8-hr TWA, with a 95th percentile of 3,043 ppm 8-hr TWA.

The high-end value is two orders of magnitude higher than identified in the monitoring data, but the central tendency is comparable to the monitoring data. This may be due to the limited number of sites from which the monitoring data were taken or that limited data for conveyorized degreaser were reported to the 2014 NEI data (data were only found for three total units). It is also uncertain of the underlying methodologies used to estimate emissions in the 2014 NEI data.

Table_Apx P-15. Summary of Exposure Modeling Results for TCE Degreasing in Conveyorized Degreasers

Scenario	8-hr TWA (ppm)	AC[a] (ppm)	ADC (ppm)	LADC (ppm)	Data Quality Rating of Associated Air Concentration Data
Workers (Near-field)					
High-End	3,043	1,014.4	694.8	275.2	N/A – Modeled Data
Central Tendency	40.8	13.6	9.3	5.3	
Occupational non-users (Far-Field)					
High-End	1,878	626	428.8	168.3	N/A – Modeled Data
Central Tendency	23.3	7.8	5.3	3.6	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] Acute exposures calculated as a 24-hr TWA.

P.7.2 Water Release Assessment

Similar to OTVDs, the primary source of water releases from conveyorized systems is expected to be from wastewater from the water separator with the primary sources of water being: 1) Moisture in the atmosphere that condenses into the solvent when exposed to the condensation coils on the system; and/or 2) steam used to regenerate carbon adsorbers used to control solvent emissions (Durkee, 2014; Kanegsberg and Kanegsberg, 2011; NIOSH, 2002a, b, c, d). The current disposal practices of the wastewater are unknown; however, a 1982 EPA (Gilbert et al., 1982) report estimated 20% of water releases from metal cleaning (including batch systems, conveyorized systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

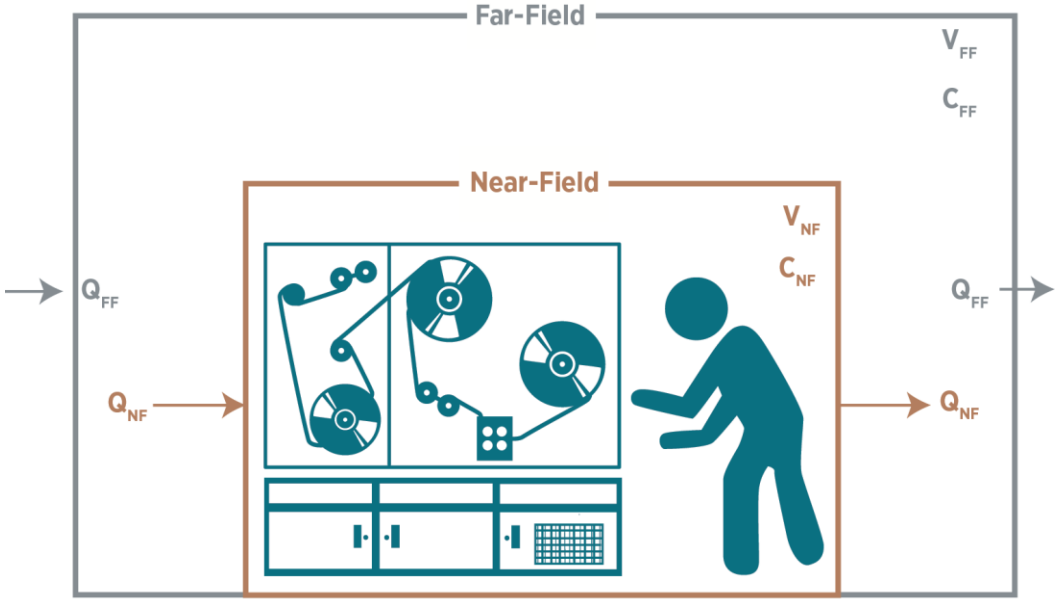
EPA assumes the TRI and DMR data cover all water discharges of TCE from conveyorized degreasing. However, EPA cannot distinguish between degreaser types in TRI and DMR data; therefore, a single set of water release for all degreasing operations is presented in Section P.5.2 for OTVDs.

P.8 Web Vapor Degreasing

P.8.1 Exposure Assessment

EPA did not identify inhalation exposure monitoring data related to the use of TCE in web degreasing. Therefore, EPA used the Near-Field/Far-Field Model to estimate exposures to workers and ONUs. The following details the results of EPA's occupational exposure assessment for use in web degreasers based on inhalation exposure modeling.

Figure_Apx P-3 illustrates the near-field/far-field model that can be applied to web degreasing. As the figure shows, TCE vapors evaporate into the near-field (at evaporation rate G), resulting in near-field exposures to workers at a concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, G, into the near-field, whose volume is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outdoor air.



Figure_Apx P-3. Schematic of the Web Degreasing Near-Field/Far-Field Inhalation Exposure Model

To estimate the TCE vapor generation rate, the model uses the annual emission rate and annual operating time from the single web degreasing unit reported in the (U.S. EPA, 2011). Because the vapor generation rate is based a limited data set, it is unknown how representative the model is of a "typical" web degreasing site.

EPA performed a Monte Carlo simulation with 100,000 iterations and the Latin Hypercube sampling method in @Risk to calculate 8-hour TWA near-field and far-field exposure concentrations. Near-field exposure represents exposure concentrations for workers who directly operate the vapor degreasing

equipment, whereas far-field exposure represents exposure concentrations for occupational non-users (i.e., workers in the surrounding area who do not handle the degreasing equipment).

Table_Apx P-16 presents a statistical summary of the exposure modeling results. These exposure estimates represent modeled exposures for the workers and occupational non-users. For workers, the 50th percentile exposure is 5.9 ppm 8-hr TWA, with a 95th percentile of 14.1 ppm 8-hr TWA.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2011 NEI were only found for one unit, and the underlying methodologies used to estimate the emission is unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Table_Apx P-16. Summary of Exposure Modeling Results for TCE Degreasing in Web Degreasers

Scenario	8-hr TWA (ppm)	AC[a] (ppm)	ADC (ppm)	LADC (ppm)	Confidence Rating of Air Concentration Data
Workers (Near-field)					
High-End	14.1	4.7	3.2	1.4	N/A – Modeled Data
Central Tendency	5.9	2.0	1.4	0.5	
Occupational non-users (Far-Field)					
High-End	9.6	3.2	2.2	0.9	N/A – Modeled Data
Central Tendency	3.1	1.0	0.7	0.3	

AC = Acute Concentration; ADC = Average Daily Concentration; and LADC = Lifetime Average Daily Concentration.
[a] Acute exposures calculated as a 24-hr TWA.

P.8.2 Water Release Assessment

Similar to OTVDs, the primary source of water releases from web systems is expected to be from wastewater from the water separator with the primary sources of water being: 1) Moisture in the atmosphere that condenses into the solvent when exposed to the condensation coils on the system; and/or 2) steam used to regenerate carbon adsorbers used to control solvent emissions (Durkee, 2014; Kanegsberg and Kanegsberg, 2011; NIOSH, 2002a, b, c, d). The current disposal practices of the wastewater are unknown; however, a 1982 EPA (Gilbert et al., 1982) report estimated 20% of water releases from metal cleaning (including batch systems, conveyorized systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

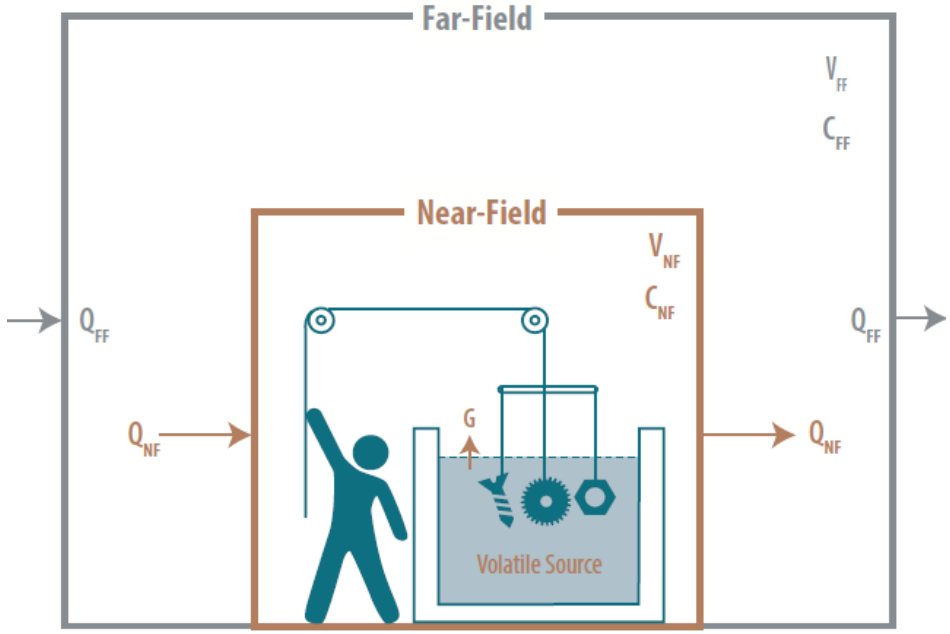
EPA assumes the TRI and DMR data cover all water discharges of TCE from web vapor degreasing. However, EPA cannot distinguish between degreaser types in TRI and DMR data; therefore, a single set of water release for all degreasing operations is used for OTVDs.

P.9 Cold Cleaning

P.9.1 Exposure Assessment

EPA did not identify inhalation exposure monitoring data for the Cold Cleaning condition of use. Therefore, EPA used the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model to estimate exposures to workers and ONUs. The following details the results of EPA's occupational exposure assessment for cold cleaning based on modeling.

Figure_Apx P-4 illustrates the near-field/far-field model that can be applied to cold cleaning. As the figure shows, TCE vapors evaporate into the near-field (at evaporation rate G), resulting in near-field exposures to workers at a concentration C_{NF}. The concentration is directly proportional to the evaporation rate of TCE, G, into the near-field, whose volume is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outdoor air.



Figure_Apx P-4. Schematic of the Cold Cleaning Near-Field/Far-Field Inhalation Exposure Model

To estimate the TCE vapor generation rate, the model developed a distribution from the reported annual emission rates and annual operating times reported in the 2014 NEI (U.S. EPA, 2018a). NEI records where the annual operating time was not reported were excluded from the distribution. Because the vapor generation rate is based a limited data set (ten total units), it is unknown how representative the model is of a "typical" cold cleaning site.

Cold cleaners are assumed to operate between 3 to 24 hours per day, based on NEI data on the reported operating hours for cold cleaners using TCE. EPA performed a Monte Carlo simulation with 100,000 iterations and the Latin Hypercube sampling method in @Risk to calculate 8-hour TWA near-field and far-field exposure concentrations. Near-field exposure represents exposure concentrations for workers who directly operate the vapor degreasing equipment, whereas far-field exposure represents exposure concentrations for occupational non-users (i.e., workers in the surrounding area who do not handle the cold cleaning equipment).

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Vapor generation rates were derived from TCE unit emissions and operating hours reported in the 2014 National Emissions Inventory. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that emissions data available in the 2014 NEI were only found for ten total units, and the underlying methodologies used to estimate these emissions are unknown. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Table_Apx P-17 presents a statistical summary of the exposure modeling results. Estimates of AC, ADC, and LADC for use in assessing risk were made using the approach and equations described in Appendix B. These exposure estimates represent modeled exposures for the workers and occupational non-users. For workers, the 50th percentile exposure is 3.33 ppm 8-hr TWA, with a 95th percentile of 57.2 ppm 8-hr TWA.

Table_Apx P-17. Summary of Exposure Modeling Results for Use of Trichloroethylene in Cold Cleaning

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Confidence Rating of Air Concentration Data
Workers (Near-field)					
High-End	57.2	19.1	13.1	5.2	N/A – Modeled Data
Central Tendency	3.33	1.11	0.8	0.3	
Occupational non-users (Far-Field)					
High-End	34.7	11.6	7.9	3.1	N/A – Modeled Data
Central Tendency	1.8	0.6	0.4	0.2	

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

P.9.2 Water Release Assessment

Similar to OTVDs, the primary source of water releases from cold cleaners is expected to be from wastewater from the water separator with the primary source of water expected to be from moisture in the atmosphere that condenses into the solvent. Water may also enter vapor degreasers via steam used to regenerate carbon adsorbers; however, it is unclear if carbon adsorbers would be used in conjunction with cold cleaning equipment. The current disposal practices of the wastewater are unknown; however, a 1982 EPA (Gilbert et al., 1982) report estimated 20% of water releases from metal cleaning (including batch systems, conveyorized systems, and vapor and cold systems) were direct discharges to surface water and 80% of water releases were discharged indirectly to a POTW.

EPA assesses water release using TRI and DMR data. However, EPA cannot distinguish between degreasers and cold cleaners in TRI and DMR data; therefore, a single set of water release for all degreasing and cold cleaning operations is used for OTVDs.

P.10 Aerosol Applications: Spray Degreasing/Cleaning, Automotive Brake and Parts Cleaners, Penetrating Lubricants, and Mold Releases

P.10.1 Exposure Assessment

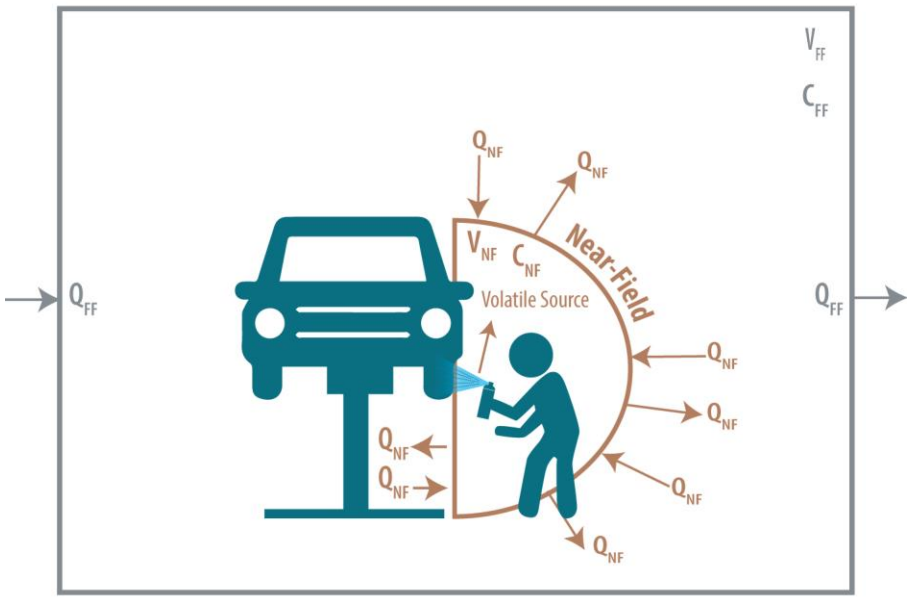
EPA did not identify inhalation exposure monitoring data related to the use of TCE in aerosol degreasers. Therefore, EPA estimated inhalation exposures using the Brake Servicing Near-field/Far-field Exposure Model. EPA used the brake servicing model as a representative scenario for this condition of use as there was ample data describing the brake servicing use and it is a significant use of TCE-based aerosol products. The following details the results of EPA's occupational exposure assessment for aerosol degreasing and aerosol lubricants based on modeling.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of modeling, in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input parameters. Various model parameters were derived from a CARB brake service study and TCE concentration data for 16 products representative of the condition of use. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium.

Figure_Apx P-5 illustrates the near-field/far-field for the aerosol degreasing scenario. As the figure shows, TCE in aerosolized droplets immediately volatilizes into the near-field, resulting in worker exposures at a concentration C_{NF}. The concentration is directly proportional to the amount of aerosol degreaser applied by the worker, who is standing in the near-field-zone (i.e., the working zone). The volume of this zone is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly TCE dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures to TCE at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the TCE dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly TCE dissipates out of the surrounding space and into the outside air.

In this scenario, TCE mists enter the near-field in non-steady "bursts," where each burst results in a sudden rise in the near-field concentration, followed by a more gradual rise in the far-field concentration. The near-field and far-field concentrations then decay with time until the next burst causes a new rise in near-field concentration.

Based on site data from maintenance and auto repair shops obtained by CARB (CARB, 2000) for brake cleaning activities, the model assumes a worker will perform 11 applications of the degreaser product per brake job with five minutes between each application and that a worker may perform one to four brake jobs per day each taking one hour to complete. EPA modeled two scenarios, one where the brake cleaning jobs occurred back-to-back and one where braking cleaning jobs occurred one hour apart. Based on data from CARB (CARB, 2000), EPA assumes each brake job requires 14.4 oz of aerosol brake cleaner. The model determines the application rate of TCE using the weight fraction of TCE in the aerosol product. EPA uses uniform distribution of weight fractions for TCE based on facility data for the aerosol products in use (CARB, 2000). It is uncertain whether the use rate and weight fractions for brake cleaning are representative of other aerosol degreasing and lubricant applications.



Figure_Apx P-5. Schematic of the Near-Field/Far-Field Model for Aerosol Degreasing

EPA performed a Monte Carlo simulation with 1,000,000 iterations and the Latin hypercube sampling method to model near-field and far-field exposure concentrations in the aerosol degreasing scenario. The model calculates both 8-hr TWA exposure concentrations and acute 24-hr TWA exposure concentrations. Table_Apx P-18 presents a statistical summary of the exposure modeling results.

For workers, the exposures are 7.63 ppm 8-hr TWA at the 50th percentile and 23.98 ppm 8-hr TWA at the 95th percentile. For occupational non-users, the model exposures are 0.14 ppm 8-hr TWA at the 50th percentile and 1.04 ppm 8-hr TWA at the 95th percentile.

Table_Apx P-18. Summary of Worker and Occupational Non-User Inhalation Exposure Modeling Results for Aerosol Degreasing

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Confidence Rating of Air Concentration Data
Workers (Near-field)					
High-End	24.0	8.0	5.5	2.2	N/A – Modeled Data
Central Tendency	7.6	2.5	1.7	0.6	
Occupational non-users (Far-Field)					
High-End	1.0	0.4	0.2	0.1	N/A – Modeled Data
Central Tendency	0.1	0.05	0.03	0.01	

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

P.10.2 Water Release Assessment

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

P.11 Metalworking Fluids

P.11.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from OSHA facility inspections (OSHA, 2017) at two sites using TCE in metalworking fluids. Due to small sample sizes, it is unclear how representative these data are of "typical" MWF use. Therefore, EPA supplemented the identified monitoring data with an assessment of inhalation exposures using the ESD on the Use of Metalworking Fluids (OECD, 2011b). The following subsections detail the results of EPA's occupational exposure assessment for TCE use in MWFs based on inhalation exposure monitoring data and modeling.

Table_Apx P-19 summarizes the 8-hr TWA monitoring data for the use of TCE in MWFs. No data was found to estimate ONU exposures from use in metalworking fluids. Data from this source covers exposures at a facility that produces various electrical resistors (Gilles and Philbin, 1976). The data were provided as full-shift TWAs.

Table_Apx P-19. Summary of Worker Inhalation Exposure Monitoring Data for TCE Use in Metalworking Fluids

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	75.4	25.1	17.2	8.8	3	High
Central Tendency	69.7	23.2	15.9	6.3		

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

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 3 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include limited dataset (3 data points from 1 site), and the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is low.

EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. Data from the 2011 Emission Scenario Document on the Use of Metalworking Fluids was used to estimate inhalation exposures. The primary limitations of the exposure outputs from this model include the uncertainty of the representativeness of these data toward the true distribution of inhalation for all TCE uses for the industries and sites covered by this scenario, and the difference between the modeling data and monitoring data. Added uncertainties include that the underlying TCE concentration used in the metalworking fluid was assumed from one metalworking fluid product. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium.

The ESD estimates typical and high-end exposures for different types of metalworking fluids. These estimates are provided in Table_Apx P-20 and are based on a NIOSH study of 79 small metalworking facilities (OECD, 2011b). The concentrations for these estimates are for the solvent-extractable portion and do not include water contributions (OECD, 2011b). The "typical" mist concentration is the geometric mean of the data and the "high-end" is the 90th percentile of the data (OECD, 2011b).

Table_Apx P-20. ESD Exposure Estimates for Metalworking Fluids Based on Monitoring Data

Type of Metalworking Fluid	Typical Mist Concentration (mg/m^3)[a]	High-End Mist Concentration (mg/m^3)[b]
Conventional Soluble	0.19	0.87
Semi-Synthetic	0.20	0.88
Synthetic	0.24	1.10
Straight Oil	0.39	1.42

[a] The typical mist concentration is the geometric mean of the data (OECD, 2011b)
[b] The high-end mist concentration is the 90th percentile of the data (OECD, 2011b)
Source: (OECD, 2011b)

The recommended use of the TCE-based metalworking fluid is an oil-based cutting and tapping fluid; therefore, EPA assesses exposure to the TCE-based metalworking fluids using the straight oil mist concentrations and the max concentration of TCE in the metalworking fluid. Straight oils are not diluted; therefore, the concentration of TCE specified in the SDS (98%) (U.S. EPA, 2017b) is equal to the concentration of TCE in the mist. Table_Apx P-21 presents the exposure estimates for the use of TCE-based metalworking fluids. The ESD estimates an exposure duration of eight hours per day; therefore, results are presented as 8-hr TWA exposure values. It should be noted that these estimates may underestimate exposures to TCE during use of metalworking fluids as they do not account for exposure to TCE that evaporates from the mist droplets into the air. This exposure is difficult to estimate and is not considered in this assessment.

Table_Apx P-21. Summary of Exposure Results for Use of TCE in Metalworking Fluids Based on ESD Estimates

Scenario	8-hr TWA (ppm)[a]	ADC (ppm)	LADC (ppm)	Data Quality Rating of Associated Air Concentration Data
High-End	0.3	0.1	0.03	N/A – Modeled Data
Central Tendency	0.1	0.02	6.0E-3	

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

[a] The TCE exposure concentrations are calculated by multiplying the straight oil mist concentrations in Table_Apx P-20 by 98% (the concentration of TCE in the metalworking fluid) and converting to ppm.

The monitoring data obtained is two orders of magnitude higher than the modeling data. It is uncertain if the limited monitoring data set (three sample points), or the age of the monitoring data (1976) is representative of exposures to TCE for all sites covered by this condition of use.

P.11.2 Water Release Assessment

The ESD states that water releases from use of straight oil metalworking fluids may come from disposal of container residue and dragout losses from cleaning the part after shaping (OECD, 2011b). Facilities typically treat wastewater onsite due to stringent discharge limits to POTWs (OECD, 2011b). Control technologies used in onsite wastewater treatment in the MP&M industry include ultrafiltration, oil/water separation, and chemical precipitation (OECD, 2011b). Facilities that do not treat wastewater onsite contract waste haulers to collect wastewater for off-site treatment (OECD, 2011b).

EPA assesses water release using TRI and DMR data. However, EPA cannot distinguish between sites using metalworking fluids and sites using TCE in degreasers in TRI and DMR data; therefore, a single set of water release for degreasing and metalworking fluid operations is used for OTVDs.

P.12 Adhesives, Sealants, Paints, and Coatings

P.12.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from a NIOSH a Health Hazard Evaluation report (HHE) (Chrostek, 1981) using TCE in coating applications and from OSHA facility inspections (OSHA, 2017) at three sites using TCE in adhesives and coatings. The following details the results of EPA's occupational exposure assessment for coating applications based on inhalation exposure monitoring data.

Table_Apx P-22 summarizes the 8-hr TWA monitoring data for the use of TCE in coatings. The data were obtained from a HHE (Chrostek, 1981) and from OSHA data (OSHA, 2017). The HHE data also provided two data points where the worker job description was "foreman." EPA assumed this data is applicable to ONU exposure. However, due to the limited data set and the various types of application methods that may be employed, EPA is unsure of the representativeness of these data toward actual exposures to TCE for all sites covered by this condition of use.

Table_Apx P-22. Summary of Worker Inhalation Exposure Monitoring Data for Adhesives/Paints/Coatings

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
Workers						
High-End	39.5	13.2	9.0	4.6	22	Medium
Central Tendency	4.6	1.6	1.1	0.4		
Occupational non-users						
High-End	1.0	0.3	0.2	0.1	2	Medium
Central Tendency	0.9	0.3	0.2	0.1		

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

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 22 data points from 2 sources, and the data quality ratings from systematic review for these data were medium to high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

For the ONU inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 2 data points from 1 source, and the data quality ratings from systematic review for the data point was high. The primary limitations of this data is the limited dataset (two data points from 1 site), and the uncertainty of the representativeness of this data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

EPA did not find data to provide inhalation exposure estimates for commercial adhesive, sealant, paint and coating applications. Therefore, EPA uses the industrial data discussed above as surrogate for

commercial coatings, as EPA believes the activities and exposures will be similar between industrial and commercial sites covered by this condition of use.

P.12.2 Water Release Assessment

In general, potential sources of water releases from adhesive, sealants, and paints/coatings use may include the following: equipment cleaning operations, and container cleaning wastes (OECD, 2011a).

Water releases for adhesives, sealants, paints and coating sites were assessed using data reported from three sites in the 2016 TRI and 2016 DMR. For the sites in the 2014 NEI (where release information is not provided), an average release per site was calculated from the total releases of the three aforementioned sites reporting water releases to DMR and TRI, and dividing the total release by the total number of sites in TRI and DMR (17 sites). This average release per site was used to estimate releases from the sites provided in the 2014 NEI. EPA assessed daily releases by assuming 250 days of operation per year, as recommended in the 2011 ESD on the Application of Radiation Curable Coatings, Inks, and Adhesives via Spray, Vacuum, Roll and Curtain Coating, and averaging the annual releases over the operating days (OECD, 2011a). A summary of the water releases can be found in Table_Apx P-23.

Table_Apx P-23. Reported Water Releases of Trichloroethylene from Sites Using TCE in Adhesives, Sealants, Paints and Coatings

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day) [a]	NPDES Code	Release Media
Able Electropolishing Co Inc, Chicago, IL	74.4	250	0.30	Not available	POTW
Garlock Sealing Technologies, Palmyra, NY	0.08	250	3.3E-04	NY0000078	Surface Water
Ls Starrett Co, Athol, MA	9.1E-04	250	3.6E-06	MAR05B615	Surface Water
Aerojet Rocketdyne, Inc., East Camden, AR	4.4	250	1.8E-02	Not available	Surface Water or POTW
Best One Tire & Service, Nashville, TN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Bridgestone Aircraft Tire (USA), Inc., Mayodan, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Clayton Homes Inc, Oxford, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Cmh Manufacturing, Inc. Dba Schult Homes - Plant 958, Richfield, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Delphi Thermal Systems, Lockport, NY	4.4	250	1.8E-02	Not available	Surface Water or POTW
Green Bay Packaging Inc - Coon Rapids, Coon Rapids, MN	4.4	250	1.8E-02	Not available	Surface Water or POTW

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day) [a]	NPDES Code	Release Media
Mastercraft Boat Company, Vonore, TN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Michelin Aircraft Tire Company, Norwood, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
M-Tek, Inc, Manchester, TN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Olin Corp, East Alton, IL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Parker Hannifin Corp - Paraflex Division, Manitowoc, WI	4.4	250	1.8E-02	Not available	Surface Water or POTW
Parrish Tire Company, Yadkinville, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Republic Doors And Frames, Mckenzie, TN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Ro-Lab Rubber Company Inc., Tracy, CA	4.4	250	1.8E-02	Not available	Surface Water or POTW
Royale Comfort Seating, Inc. - Plant No. 1, Taylorsville, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Snider Tire, Inc., Statesville, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Snyder Paper Corporation, Hickory, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Stellana Us, Lake Geneva, WI	4.4	250	1.8E-02	Not available	Surface Water or POTW
Thomas Built Buses - Courtesy Road, High Point, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Unicel Corp, Escondido, CA	4.4	250	1.8E-02	Not available	Surface Water or POTW
Acme Finishing Co Llc, Elk Grove Village, IL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Aerojet Rocketdyne, Inc., Rancho Cordova, CA	4.4	250	1.8E-02	Not available	Surface Water or POTW

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day) [a]	NPDES Code	Release Media
Allegheny Cnty Airport Auth/Pgh Intl Airport, Pittsburgh, PA	4.4	250	1.8E-02	Not available	Surface Water or POTW
Amphenol Corp - Aerospace Operations, Sidney, NY	4.4	250	1.8E-02	Not available	Surface Water or POTW
Aprotech Powertrain, Asheville, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Clayton Homes Inc, Oxford, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Coating & Converting Tech Corp/Adhesive Coatings, Philadelphia, PA	4.4	250	1.8E-02	Not available	Surface Water or POTW
Corpus Christi Army Depot, Corpus Christi, TX	4.4	250	1.8E-02	Not available	Surface Water or POTW
Electronic Data Systems Camp Pendleton, Camp Pendleton, CA	4.4	250	1.8E-02	Not available	Surface Water or POTW
Florida Production Engineering, Inc., Ormond Beach, FL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Goodrich Corporation, Jacksonville, FL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Kasai North America Inc, Madison Plant, Madison, MS	4.4	250	1.8E-02	Not available	Surface Water or POTW
Kirtland Air Force Base, Albuquerque, NM	4.4	250	1.8E-02	Not available	Surface Water or POTW
Marvin Windows & Doors, Warroad, MN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Mcneilus Truck & Manufacturing Inc, Dodge Center, MN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Metal Finishing Co. - Wichita (S Mclean Blvd), Wichita, KS	4.4	250	1.8E-02	Not available	Surface Water or POTW
Michelin Aircraft Tire Company, Norwood, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Murakami Manufacturing Usa Inc, Campbellsville, KY	4.4	250	1.8E-02	Not available	Surface Water or POTW

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day) [a]	NPDES Code	Release Media
Peterbilt Motors Denton Facility, Denton, TX	4.4	250	1.8E-02	Not available	Surface Water or POTW
Portsmouth Naval Shipyard, Kittery, ME	4.4	250	1.8E-02	Not available	Surface Water or POTW
R.D. Henry & Co., Wichita, KS	4.4	250	1.8E-02	Not available	Surface Water or POTW
Raytheon Company, Portsmouth, RI	4.4	250	1.8E-02	Not available	Surface Water or POTW
Rehau Inc, Cullman, AL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Rotochopper Inc, Saint Martin, MN	4.4	250	1.8E-02	Not available	Surface Water or POTW
Rubber Applications, Mulberry, FL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Sapa Precision Tubing Rockledge, Llc, Rockledge, FL	4.4	250	1.8E-02	Not available	Surface Water or POTW
Thomas & Betts, Albuquerque, NM	4.4	250	1.8E-02	Not available	Surface Water or POTW
Thomas Built Buses - Fairfield Road, High Point, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Timco, Dba Haeco Americas Airframe Services, Greensboro, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
Trelleborg Coated Systems Us, Inc - Grace Advanced Materials, Rutherfordton, NC	4.4	250	1.8E-02	Not available	Surface Water or POTW
U.S. Coast Guard Yard - Curtis Bay, Curtis Bay, MD	4.4	250	1.8E-02	Not available	Surface Water or POTW
Viracon Inc, Owatonna, MN	4.4	250	1.8E-02	Not available	Surface Water or POTW

3574 POTW = Publicly Owned Treatment Works
3575 Releases of 4.4 kg/site-yr for NEI sites estimated from total releases from TRI and DMR sites and divided by the 3 sites
3576 reporting water releases and the 14 sites reporting zero water releases in TRI).
3577 [a] Daily releases are back-calculated from the annual release rate and assuming 250 days of operation per year.
3578 Sources: (U.S. EPA, 2018a, 2017c, 2016a)
3579

P.13 Other Industrial Uses

P.13.1 Exposure Assessment

EPA did not identify inhalation exposure monitoring data related to using TCE for other industrial uses. Therefore, EPA used monitoring data from loading/unloading TCE during manufacturing as a surrogate. See section P.1.1 for additional information on the data used. EPA assumes the exposure sources, routes, and exposure levels are similar to those during loading at a TCE manufacturing facility. However, EPA is unsure of the representativeness of these surrogate data toward actual exposures to TCE at all sites covered by this condition of use.

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA inhalation air concentrations. The primary strengths include the assessment approach, which is the use of surrogate monitoring data, in the middle of the inhalation approach hierarchy. These monitoring data include 16 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these surrogate data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Table_Apx P-24 summarizes the 8-hr TWA from monitoring data from TCE manufacturing. The data were obtained from obtained from data submitted by the Halogenated Solvents Industry Alliance (HSIA) via public comment for one company (Halogenated Solvents Industry Alliance, 2018 5176415). No data was found to estimate ONU exposures during other industrial uses of TCE. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

Table_Apx P-24 Summary of Occupational Exposure Surrogate Monitoring Data for Unloading TCE During Other Industrial Uses

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	2.6	0.9	0.6	0.3	16	Medium
Central Tendency	0.4	0.1	0.1	0.03		

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

P.13.2 Water Release Assessment

Specifics of the processes and potential sources of release for other industrial uses are unknown. However, general potential sources of water releases in the chemical industry may include the following: equipment cleaning operations, aqueous wastes from scrubbers/decanters, reaction water, process water from washing intermediate products, and trace water settled in storage tanks (OECD, 2019).

EPA assessed water releases using the annual discharge values reported to the 2016 TRI and the 2016 DMR by the 49 sites using TCE in other industrial uses. In the 2016 TRI, all 28 reported zero discharge to water. In the 2016 DMR, twenty-one sites reported a direct discharge to surface water (indirect discharges not reported in DMR data).

To estimate the daily release, EPA assumed a default of 250 days/yr of operation and averaged the annual release over the operating days. Table_Apx P-25 summarizes the water releases from the 2016 TRI and DMR for sites with non-zero discharges.

Table_Apx P-25. Reported Water Releases of Trichloroethylene from Other Industrial Uses

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)[a]	Daily Release (kg/site-day)[a]	NPDES Code	Release Media
Eli Lilly And Company-Lilly Tech Ctr, Indianapolis, IN	388	250	1.6	IN0003310	Surface Water
Oxy Vinyls LP - Deer Park Pvc, Deer Park, TX	37	250	0.15	TX0007412	Surface Water
Solvay - Houston Plant, Houston, TX	8.3	250	0.03	TX0007072	Surface Water
Washington Penn Plastics, Frankfort, KY	8.0	250	0.03	KY0097497	Surface Water
Natrium Plant, New Martinsville, WV	5.5	250	2.2E-02	WV0004359	Surface Water
Leroy Quarry, Leroy, NY	4.8	250	1.9E-02	NY0247189	Surface Water
George C Marshall Space Flight Center, Huntsville, AL	2.6	250	1.0E-02	AL0000221	Surface Water
Whelan Energy Center Power Plant, Hastings, NE	2.4	250	9.4E-03	NE0113506	Surface Water
Akzo Nobel Surface Chemistry LLC, Morris, IL	0.1	250	4.6E-04	IL0026069	Surface Water
Solutia Nitro Site, Nitro, WV	0.1	250	4.4E-04	WV0116181	Surface Water
Amphenol Corporation - Columbia, Columbia, SC	0.1	250	2.8E-04	SC0046264	Surface Water
Army Cold Regions Research & Engineering Lab, Hanover, NH	0.1	250	2.3E-04	NH0001619	Surface Water
Corning - Canton Plant, Canton, NY	0.1	250	2.2E-04	NY0085006	Surface Water
Keeshan And Bost Chemical Co., Inc., Manvel, TX	0.03	250	1.3E-04	TX0072168	Surface Water
Ames Rubber Corp Plant #1, Hamburg Boro, NJ	0.03	250	1. 1E-04	NJG000141	Surface Water
Gorham, Providence, RI	0.02	250	9.2E-05	RIG85E004	Surface Water
Emerson Power Transmission, Ithaca, NY	0.02	250	6.9E-05	NY0002933	Surface Water
Chemtura North and South Plants, Morgantown, WV	8.3E-03	250	3.3E-05	WV0004740	Surface Water
Indorama Ventures Olefins, LLC, Sulphur, LA	5.1E-03	250	2.0E-05	LA0069850	Surface Water
William E. Warne Power Plant, Los Angeles County, CA	3.1E-03	250	1.2E-05	CA0059188	Surface Water
Raytheon Aircraft Co (Was Beech Aircraft), Boulder, CO	2.3E-03	250	9.2E-06	COG315176	Surface Water

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are calculated from the annual release rate and assuming 250 days of operation per year.

Sources: (U.S. EPA, 2017c, 2016a)

P.14 Spot Cleaning, Wipe Cleaning and Carpet Cleaning

P.14.1 Exposure Assessment

EPA identified minimal inhalation exposure monitoring data related to the spot cleaning using TCE. Therefore, EPA supplemented the identified monitoring data using the Near-field/Far-field Exposure Model. The following subsections detail the results of EPA's occupational exposure assessment for spot cleaning based on inhalation exposure monitoring data and modeling.

Table_Apx P-26 summarizes the 8-hr TWA monitoring data and acute TWAs from the monitoring data for the use of TCE in in spot cleaning. No data was found to estimate ONU exposures during spot cleaning. The data were obtained from NIOSH a Health Hazard Evaluation report (HHE) (Burton and Monesterskey, 1996), as well as a NIOSH Report on Control of Health and Safety Hazards on Commercial Drycleaners document (NIOSH, 1997). NIOSH HHEs are conducted at the request of employees, employers, or union officials, and provide information on existing and potential hazards present in the workplaces evaluated. NIOSH Health and Safety documents represents NIOSH research in collaboration with industry, labor and other government organizations to protect the health of workers in industry.

For full shift values, sample times ranged from approximately seven to nine hours (Burton and Monesterskey, 1996). Where sample times were less than eight hours, EPA converted to an 8-hr TWA assuming exposure outside the sample time was zero. For sample times greater than eight hours, EPA left the measured concentration as is. Because of the limited data set, EPA is unsure of the representativeness of these data toward actual exposures to TCE for all sites covered by this condition of use.

Table_Apx P-26. Summary of Worker Inhalation Exposure Monitoring Data for Spot Cleaning Using TCE

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of 8-hr TWA Data Points	Confidence Rating of Air Concentration Data
High-End	2.8	1.0	0.7	0.3	8	Medium
Central Tendency	0.4	0.1	0.1	0.04		

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

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 8 data points from 2 sources, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

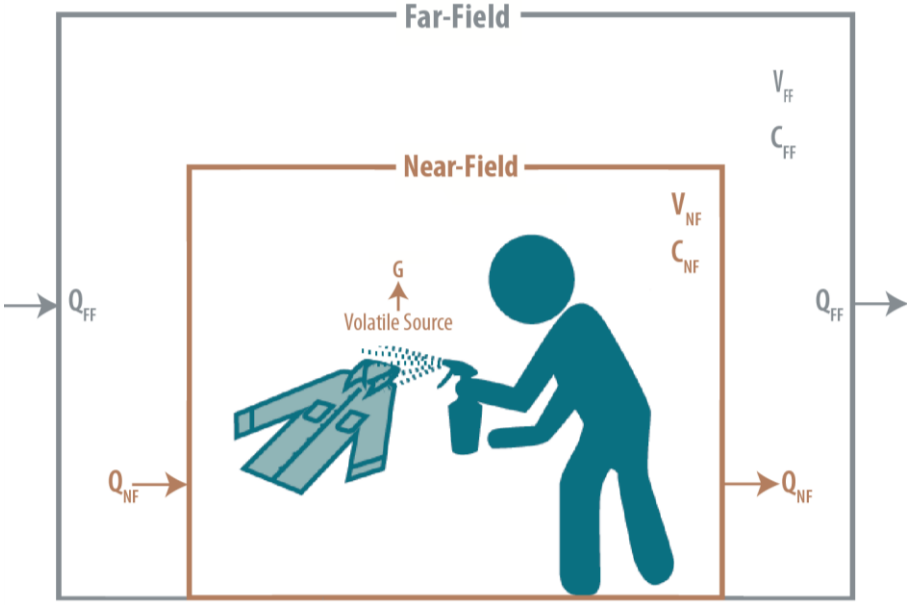
EPA also considered the use of modeling, which is in the middle of the inhalation approach hierarchy. A Monte Carlo simulation with 100,000 iterations was used to capture the range of potential input

parameters. Various model parameters were derived from a CARB study. The primary limitations of the air concentration outputs from the model include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Added uncertainties include that the underlying methodologies used to obtain the values in the CARB study, as well as the assumed TCE concentration in the spot cleaning product. Based on these strengths and limitations of the air concentrations, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Despite these limitation, the modeling and monitoring results match each other very closely. Therefore, the overall confidence is medium.

Wolf and Morris (IRTA, 2007) estimated 42,000 gal of TCE-based spotting agents are sold in California annually. Review of SDS's identified TCE-based spotting agents contain 10% to 100% TCE. The study also estimated approximately 5,000 textile cleaning facilities in California. Results in average of 8.4 gal/site-yr of TCE-based spotting agents used.

Figure_Apx P-6 illustrates the near-field/far-field modeling approach that EPA applied to spot cleaning facilities. As the figure shows, chemical vapors evaporate into the near-field (at evaporation rate G), resulting in near-field exposures to workers at a concentration C_{NF}. The concentration is directly proportional to the amount of spot cleaner applied by the worker, who is standing in the near-field-zone (i.e., the working zone). The volume of this zone is denoted by V_{NF}. The ventilation rate for the near-field zone (Q_{NF}) determines how quickly the chemical of interest dissipates into the far-field (i.e., the facility space surrounding the near-field), resulting in occupational non-user exposures at a concentration C_{FF}. V_{FF} denotes the volume of the far-field space into which the chemical of interest dissipates out of the near-field. The ventilation rate for the surroundings, denoted by Q_{FF}, determines how quickly the chemical dissipates out of the surrounding space and into the outdoor air.



Figure_Apx P-6. Schematic of the Near-Field/Far-Field Model for Spot Cleaning

EPA performed Monte Carlo simulations, applying one hundred thousand iterations and the Latin hypercube sampling method. Table_Apx P-27 presents a statistical summary of the exposure modeling results. The 50th and 95th percentile near-field exposures are 0.96 ppm and 2.77 ppm 8-hr TWA,

respectively. These results are comparable to the monitoring data. For occupational non-users (far-field), model 50th and 95th percentile exposure levels are 0.48 ppm and 1.75 ppm 8-hr TWA, respectively. EPA assumes no engineering controls are used at dry cleaning shops, which are typically small, family owned businesses.

The modeling results are comparable to the monitoring data. However, EPA is unsure of the representativeness of these data toward actual exposures to TCE for all sites covered by this condition of use.

Table_Apx P-27. Summary of Exposure Modeling Results for Spot Cleaning Using TCE

Scenario	8-hr TWA (ppm)	AC (24-hr) (ppm)	ADC (ppm)	LADC (ppm)	Data Quality Rating of Associated Air Concentration Data
Workers (Near-field)					
High-End	2.8	0.9	0.6	0.3	N/A – Modeled Data
Central Tendency	1.0	0.3	0.2	0.1	
Occupational non-users (Far-Field)					
High-End	1.8	0.6	0.4	0.2	N/A – Modeled Data
Central Tendency	0.5	0.2	0.1	0.04	

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

P.14.2 Water Release Assessment

TCE releases to water from spot cleaning will depend upon whether the stained surface is washed with water after spotting. For example, TCE-based cleaners used to pre-spot garments prior to cleaning in water or hydrocarbon-based machines would be a source of TCE in wastewater.

Water releases for spot cleaning were assessed using data reported in the 2016 DMR. No sites discharging TCE from spot cleaning activities were found in the 2016 TRI. EPA assessed annual releases as reported in the 2016 DMR and assessed daily releases by assuming 300 days of operation per year. A summary of the water releases reported to the 2016 DMR can be found in Table_Apx P-28. The annual release for each of the unknown sites is calculated by taking the average annual release of the two sites reporting to DMR.

Table_Apx P-28. Reported Water Releases of Trichloroethylene from Sites Using TCE Spot Cleaning

Site	Annual Release[a] (kg/site-year)	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	Media of Release
Boise State University, Boise, ID	0.02	300	8.0E-05	Surface Water
Venetian Hotel And Casino, Las Vegas, NV	8.8E-3	300	2.9E-05	Surface Water

Site	Annual Release[a] (kg/site-year)	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	Media of Release
63,746 Unknown Sites	0.02	300	5.4E-05	Surface Water or POTW

POTW = Publicly Owned Treatment Works

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 300 days of operation per year.

Sources: 2016 DMR (U.S. EPA, 2016a)

P.15 Industrial Processing Aid

P.15.1 Exposure Assessment

EPA did identify inhalation exposure monitoring data related using TCE when used as an industrial processing aid from one site. The following details the results of EPA's occupational exposure assessment for use of TCE as an industrial processing aid based on inhalation exposure monitoring data.

Table_Apx P-29 summarizes the 12-hr TWA monitoring data and acute TWAs from the monitoring data for the use of TCE as a processing aid for both workers and for ONUs. The data were obtained from a European Commission (EC) Technical Report (EC, 2014). The data was supplied to the EC as supporting documentation in an application for continued use of TCE under the REACH Regulation. The data indicate a full shift is 12 hours. Therefore, all exposures were calculated using a 12-hr shift. Because of the limited data set, EPA is unsure of the representativeness of these data toward actual exposures to TCE for all sites covered by this condition of use.

Table_Apx P-29. Summary of Exposure Monitoring Data for Use as a Processing Aid

Scenario	12-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of 12-hr Data Points	Confidence Rating of Air Concentration Data
Workers						
High-End	12.8	6.4	4.4	2.2	30	Medium to High
Central Tendency	4.2	2.1	1.5	0.6		
Occupational non-users						
High-End	2.9	1.4	1.0	0.5	4	Medium
Central Tendency	1.3	0.7	0.4	0.2		

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

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 12-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 30 data points from 1 source, and the data quality ratings from systematic review for these data were high. The primary limitations of these data include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths

and limitations of the inhalation air concentration data, the overall confidence for these 12-hr TWA data in this scenario is medium to high.

For the ONU inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 4 data points from 1 source, and the data quality ratings from systematic review for the data point was high. The primary limitations of this single data point include the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 12-hr TWA data in this scenario is medium to low.

P.15.2 Water Release Assessment

In general, potential sources of water releases in the chemical industry may include the following: equipment cleaning operations, aqueous wastes from scrubbers/decanters, reaction water, process water from washing intermediate products, and trace water settled in storage tanks (OECD, 2019). Based on the use as a processing aid and the amount of TCE used for this condition of use, EPA expects minimal sources of TCE release to water.

Water releases during use as a processing aid were assessed using data reported in the 2016 TRI as well as 2016 DMR. Four of the 16 sites reporting to TRI provided water releases. The remaining 12 sites reported all releases were to off-site land, incineration or recycling. EPA assessed annual releases as reported in the 2016 TRI and assessed daily releases by assuming 300 days of operation per year. A summary of the water releases reported to the 2016 DMR and 2016 TRI can be found in Table_Apx P-30.

Table_Apx P-30. Reported Water Releases of Trichloroethylene from Industrial Processing Aid Sites Using TCE

Site Identity	Annual Release (kg/site-yr)[a]	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	NPDES Code	Release Media
Entek International LLC, Lebanon, OR	113	300	0.4	Not available	POTW
Occidental Chemical Corp Niagara Plant, Niagara Falls, NY	5.8	300	0.02	NY0003336	Surface Water
National Electrical Carbon Products Dba Morgan Adv Materials, Fostoria, OH	2.3	300	7. 6E-03	Not available	POTW
Daramic LLC, Corydon, IN	2.3	300	0.01	Not available	Surface Water
PPG Industries Inc Barberton, Barberton, OH	1.4	300	4.5E-3	OH0123897	POTW
Stepan Co Millsdale Road, Elwood, IL	0.2	300	5.5E-04	IL0002453	Surface Water

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 300 days of operation per year.
POTW = Publicly Owned Treatment Works
Sources: (U.S. EPA, 2017c, 2016a)

P.16 Commercial Printing and Copying

P.16.1 Exposure Assessment

EPA identified inhalation exposure monitoring data from a NIOSH a Health Hazard Evaluation report (HHE) (Finely and Page, 2005) using TCE in high speed printing presses. The following details the results of EPA's occupational exposure assessment for printing applications based on inhalation exposure monitoring data. Table_Apx P-31 summarizes the 8-hr TWA monitoring data for the use of TCE in printing. The data were obtained from a HHE (Finely and Page, 2005).

EPA considered the assessment approach, the quality of the data, and uncertainties in assessment results to determine a level of confidence for the 8-hr TWA data. For the inhalation air concentration data, the primary strengths include the assessment approach, which is the use of monitoring data, the highest of the inhalation approach hierarchy. These monitoring data include 20 data points from 1 source, and the data quality ratings from systematic review for these data were medium. The primary limitations of these data include a limited dataset, and the uncertainty of the representativeness of these data toward the true distribution of inhalation concentrations for the industries and sites covered by this scenario. Based on these strengths and limitations of the inhalation air concentration data, the overall confidence for these 8-hr TWA data in this scenario is medium to low.

Table_Apx P-31. Summary of Worker Inhalation Exposure Monitoring Data for High Speed Printing Presses

Scenario	8-hr TWA (ppm)	AC (ppm)	ADC (ppm)	LADC (ppm)	Number of Data Points	Confidence Rating of Air Concentration Data
High-End	2.1	0.7	0.5	0.2	20	Medium
Central Tendency	0.1	0.03	0.02	8.0E-3		

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

No monitoring data were available to estimate ONU exposures. EPA estimates that ONU exposures are lower than worker exposures, since ONUs do not typically directly handle the chemical.

P.16.2 Water Release Assessment

A potential source of water releases from Printing/copying use would come from clean-out of printing equipment if the ink is water-based (OECD, 2010). Based on the use in printing/copying and the amount of TCE used for this condition of use, EPA expects minimal sources of TCE release to water.

Water releases during use in printing and copying were assessed using data reported in the 2016 DMR. One site provided water releases. EPA assessed annual releases as reported in the 2016 DMR and assessed daily releases by assuming 250 days of operation per year. A summary of the water releases reported to the 2016 DMR can be found in Table_Apx P-32.

Table_Apx P-32. Reported Water Releases of Trichloroethylene from Commercial Printing and Copying

Site Identity	Annual Release (kg/site-yr)[a]	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	NPDES Code	Release Media

Printing and Pub Sys Div, Weatherford, OK	0.05	250	2.0E-4	OK0041785	Surface Water

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 250 days of operation per year.

As only one site was identified with water releases for this condition of use, EPA acknowledges this site does not represent the entirety of commercial printing and copying sites using TCE. However, data is not reasonably available to estimate water releases from additional sites. Based on reasonably available EPA models releases from containers may be up to: 1) 0.3% to 0.6% for small containers (<20 gal) or drums that are emptied via pouring; or 2) 2.5% to 3% for drums emptied via pumping; however, not all sites are expected to dispose of container residues to water. Additional water release sources of TCE at these sites may exist and will vary depending on the use rate of the TCE-based products.

P.17 Other Commercial Uses

P.17.1 Exposure Assessment

EPA did not identify any inhalation exposure monitoring data related to TCE use in other commercial uses. See Section P.14.1 for the assessment of worker exposure during spot cleaning activities. EPA assumes the exposure sources, routes, and exposure levels are similar to those for spot cleaners.

P.17.2 Water Release Assessment

Specifics of the processes and potential sources of release for these uses are unknown. Based on the volatility of TCE, EPA expects the majority of TCE used for these applications to evaporate and be released to air. EPA expects residuals in containers to be disposed of with general site trash that is either picked up by local waste management or by a waste handler that disposes wastes as hazardous waste.

Table_Apx P-33 summarizes non-zero water releases from sites using TCE in other commercial uses reported in the 2016 DMR. To estimate the daily release for the sites in Table_Apx P-33, EPA assumed a default of 250 days/yr of operation and averaged the annual release over the operating days. These data are not expected to capture the entirety of water releases from these uses; however, EPA does not have information to estimate water releases from sites not reporting to DMR.

Table_Apx P-33. Reported Water Releases of Trichloroethylene from Other Commercial Uses in the 2016 DMR

Site Identity	Annual Release (kg/site-yr)	Annual Release Days (days/yr)	Daily Release (kg/site-day)	NPDES Code	Release Media
Corning Hospital, Corning, NY	3.2	250	0.013	NY0246701	Surface Water
Water Street Commercial Bldg, Dayton, OH	0.7	250	2.8E-03	OH0141496	Surface Water
Union Station North Wing Office Building, Denver, CO	1.0E-01	250	4.0E-04	COG315293	Surface Water
Confluence Park Apartments, Denver, CO	7.1E-02	250	2.8E-04	COG315339	Surface Water
Park Place Mixed Use Development, Annapolis, MD	6.7E-02	250	2.7E-04	MD0068861	Surface Water
Tree Top Inc Wenatchee Plant, Wenatchee, WA	9.0E-03	250	3.6E-05	WA0051527	Surface Water
Wynkoop Denver LLCP St, Denver, CO	7.8E-03	250	3.1E-05	COG603115	Surface Water

Greer Family LLC, South Burlington, VT	1.3E-03	250	5.0E-06	VT0001376	Surface Water
John Marshall III Site, Mclean, VA	4.7E-04	250	1.9E-06	VA0090093	Surface Water

[a] Annual release amounts are based on the site reported values. Therefore, daily releases are calculated from the annual release rate and assuming 250 days of operation per year.
Sources: (U.S. EPA, 2016a)

P.18 Process Solvent Recycling and Worker Handling of Wastes

P.18.1 Exposure Assessment

EPA did not identify any inhalation exposure monitoring data related to waste handling/recycling. See Section P.4.1 for the assessment of worker exposure from chemical unloading activities. EPA assumes the exposure sources, routes, and exposure levels are similar to those at a repackaging facility.

P.18.2 Water Release Assessment

Potential sources of water releases at disposal/recycling sites may include the following: aqueous wastes from scrubbers/decanter, trace water settled in storage tanks, and process water generated during the disposal/recycling process.

EPA assessed water releases using the values reported to the 2016 TRI and DMR by the 30 disposal/recycling sites. In the 2016 TRI, three of sites reported non-zero indirect discharges to off-site wastewater treatment; one site reported discharges to both off-site wastewater treatment as well as discharge to a POTW. All sites in TRI for this condition of use reported zero direct discharges to surface water.

To estimate the daily release, EPA used a default assumption of 250 days/yr of operation as and averaged the annual release over the operating days. Table_Apx P-34 summarizes the water releases from the 2016 DMR and 2016 TRI for sites with non-zero discharges.

Table_Apx P-34. Estimated Water Releases of Trichloroethylene from Disposal/Recycling of TCE

Site Identity	Annual Release (kg/site-yr)[a]	Annual Release Days (days/yr)	Daily Release (kg/site-day)[a]	NPDES Code	Release Media
Veolia Es Technical Solutions LLC, Middlesex, NJ	6035	250	24.1	Not available	POTW WWT (0.02%) and Non-POTW WWT (99.98%)
Clean Harbors Deer Park LLC, La Porte, TX	87.1	250	0.3	TX0005941	Non-POTW WWT
Clean Harbors El Dorado LLC, El Dorado, AR	9.1	250	0.04	AR0037800	Non-POTW WWT
Clean Water Of New York Inc, Staten Island, NY	0.9	250	3.8E-03	NY0200484	Surface Water
Reserve Environmental Services, Ashtabula, OH	3.9E-04	250	1.6E-06	OH0098540	Surface Water

POTW = Publicly-Owned Treatment Works; WWT = Wastewater Treatment
[a] Annual release amounts are based on the site reported values. Therefore, daily releases are back-calculated from the annual release rate and assuming 250 days of operation per year.
Sources: (U.S. EPA, 2017c) and (U.S. EPA, 2016a)

P.19 Appendix P References

AIHA (American Industrial Hygiene Association). (2009). Mathematical models for estimating occupational exposure to chemicals. In CB Keil; CE Simmons; TR Anthony (Eds.), (2nd ed.). Fairfax, VA: AIHA Press.

Arkema Inc. (2018). Arkema Inc. comments to inform EPAs rulemaking on problem formulations for the risk evaluations to be conducted under the Toxic Substances Control Act, and general guiding principles to apply systematic review in TSCA risk evaluations. (EPA-HQ-OPPT-2016-0737-0109). Washington, D.C. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0109

ATSDR (Agency for Toxic Substances and Disease Registry). (2014). Toxicological profile for tetrachloroethylene (Draft for public comment). Atlanta, GA: US Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=265&tid=48

Bakke, B; Stewart, P; Waters, M. (2007). Uses of and exposure to trichloroethylene in U.S. industry: A systematic literature review [Review]. J Occup Environ Hyg 4: 375-390. http://dx.doi.org/10.1080/15459620701301763

Baldwin, PE; Maynard, AD. (1998). A survey of wind speed in indoor workplaces. Ann Occup Hyg 42: 303-313. http://dx.doi.org/10.1016/S0003-4878(98)00031-3

Barsan, ME. (1991). Health hazard evaluation report no. HETA 90-344-2159, A.W. Cash Valve Manufacturing Corporation, Decatur, Illinois. (HETA 90-344-2159). Cincinnati, OH: National Institute for Occupational Safety and Health.

Burton, NC; Monesterskey, J. (1996). Health hazard evaluation report no. HETA 96-0135-2612, Eagle Knitting Mills, Inc., Shawano, Wisconsin. (HETA 96-0135-2612). Cincinnati, OH: National Institute for Occupational Safety and Health.

CARB (California Air Resources Board). (2000). Initial statement of reasons for the proposed airborne toxic control measure for emissions of chlorinated toxic air contaminants from automotive maintenance and repair activities.

CARB. (2006). California Dry Cleaning Industry Technical Assessment Report. Stationary Source Division, Emissions Assessment Branch. https://www.arb.ca.gov/toxics/dryclean/finaldrycleantechreport.pdf

Cherrie, JW; Semple, S; Brouwer, D. (2004). Gloves and Dermal Exposure to Chemicals: Proposals for Evaluating Workplace Effectiveness. Ann Occup Hyg 48: 607-615. http://dx.doi.org/10.1093/annhyg/meh060

Chrostek, WJ, : Levine, M. S. (1981). Health hazard evaluation report no. HHE 30-153-881, Palmer Industrial Coatings Incorp., Williamsport, Pennsylvania. (HHE 30-153-881). Cincinnati, OH: National Institute for Occupational Safety and Health.

Crandall, MS; Albrecht, WN. (1989). Health Hazard Evaluation Report No. HETA-86-380-1957, York International Corporation, Madisonville, Kentucky (pp. 86-380). (NIOSH/00189611). Crandall, MS; Albrecht, WN.

Dancik, Y; Bigliardi, PL; Bigliardi-Qi, M, ei. (2015). What happens in the skin? Integrating skin permeation kinetics into studies of developmental and reproductive toxicity following topical exposure. Reprod Toxicol 58: 252-281. http://dx.doi.org/10.1016/j.reprotox.2015.10.001

Daniels, WJ; Orris, P; Kramkowski, R; Almaguer, D. (1988). Health Hazard Evaluation Report No. HETA-86-121-1923, Modern Plating Corporation, Freeport, Illinois (pp. 86-121). (NIOSH/00184446). Daniels, WJ; Orris, P; Kramkowski, R; Almaguer, D.

Demou, E; Hellweg, S; Wilson, MP; Hammond, SK; Mckone, TE. (2009). Evaluating indoor exposure modeling alternatives for LCA: A case study in the vehicle repair industry. Environ Sci Technol 43: 5804-5810. http://dx.doi.org/10.1021/es803551y

Dow Chemical (Dow Chemical Company). (2014). Product safety assessment: Trichloroethylene.
DOW Deutschland. (2014a). Chemical safety report: Use of trichloroethylene in industrial parts cleaning by vapour degreasing in closed systems where specific requirements (system of use-parameters) exist. Ispra, Italy: European Commission Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau. http://ec.europa.eu/DocsRoom/documents/14369/attachments/1/translations/en/renditions/native
DOW Deutschland. (2014b). Chemical safety report: Use of trichloroethylene in packaging. Ispra, Italy: European Commission Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau. http://ec.europa.eu/DocsRoom/documents/14371/attachments/1/translations/en/renditions/native
Durkee, J. (2014). Cleaning with solvents: Methods and machinery. In Cleaning with solvents: Methods and machinery. Oxford, UK: Elsevier Inc. https://www.sciencedirect.com/book/9780323225205/cleaning-with-solvents-methods-and-machinery
EC (European Commission). (2014). Exposure scenario: Use: Trichloroethylene as an extraction solvent for removal of process oil and formation of the porous structure in polyethylene based separators used in lead-acid batteries. Ispra, Italy: European Commission Joint Research Centre, Institute for Health and Consumer Protection, European Chemicals Bureau. http://ec.europa.eu/DocsRoom/documents/12344/attachments/1/translations/en/renditions/native
ECB (European Chemicals Bureau). (2004). European Union risk assessment report: Trichloroethylene (pp. 1-348). (EUR 21057 EN). European Commission. https://echa.europa.eu/documents/10162/83f0c99f-f687-4cdf-a64b-514f1e26fdc0
Elkin, LM. (1969). Process Economics Program, Chlorinated Solvents, Report No. 48. In Kirk-Othmer Encyclopedia of Chemical Technology. Menlo Park, CA: Stanford Research Institute.
ENTEK International Limited. (2014). Analysis of alternatives: Use of trichloroethylene as an extraction solvent for removal of process oil and formation of the porous structure in polyethylene based separators used in lead-acid batteries. Helsinki, Finland: European Chemicals Agency. https://echa.europa.eu/documents/10162/9a728963-e57f-48de-b977-7d05462c43e9
ESIG. (2012). SPERC fact sheet: Manufacture of substance - industrial (solvent-borne). Brussels, Belgium: European Solvents Industry Group (ESIG). https://www.esig.org/reach-ges/environment/
FH, F. (2012). Dermal Absorption of Finite doses of Volatile Compounds. J Pharm Sci 101: 2616-2619. http://dx.doi.org/10.1080/15287394
Finely, M; Page, E. (2005). Health hazard evaluation report no. HETA 2003-0203-2952, Wallace Computer Services, Clinton, Illinois. (HETA 2003-0203-2952). Cincinnati, OH: National Institute for Occupational Safety and Health.
Frasch, HF; Bunge, AL. (2015). The transient dermal exposure II: post-exposure absorption and evaporation of volatile compounds. J Pharm Sci 104: 1499-1507. http://dx.doi.org/10.1002/jps.24334
Frasch, HF; Dotson, GS; Barbero, AM. (2011). In vitro human epidermal penetration of 1-bromopropane. J Toxicol Environ Health A 74: 1249-1260. http://dx.doi.org/10.1080/15287394.2011.595666
Garrod, AN; Phillips, AM; Pemberton, JA. (2001). Potential exposure of hands inside protective glovesa summary of data from non-agricultural pesticide surveys. Ann Occup Hyg 45: 55-60. http://dx.doi.org/10.1016/S0003-4878(00)00013-2
Gilbert, D; Goyer, M; Lyman, W; Magil, G; Walker, P; Wallace, D; Wechsler, A; Yee, J. (1982). An exposure and risk assessment for tetrachloroethylene. (EPA-440/4-85-015). Washington, DC:

U.S. Environmental Protection Agency, Office of Water Regulations and Standards. http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=2000LLOH.txt

Gilles, D; Anania, TL; Ilka, R. (1977). Health hazard evaluation report no. HHE 77-12-418, Airtex Products, Fairfield, Illinois. (HHE 77-12-418). Cincinnati, OH: National Institute for Occupational Safety and Health.

Gilles, D; Philbin, E. (1976). Health hazard evaluation report no. HHE 76-61-337, TRW Incorporated, Philadelphia, Pennsylvania. (HHE 76-61-337). Cincinnati, OH: National Institute for Occupational Safety and Health.

GmbH, WC. (1940). German Patent 901,774. In Kirk-Othmer Encyclopedia of Chemical Technology. Wacker Chemie GmbH.

Golsteijn, L; Huizer, D; Hauck, M; van Zelm, R; Huijbregts, MA. (2014). Including exposure variability in the life cycle impact assessment of indoor chemical emissions: the case of metal degreasing. Environ Int 71: 36-45. http://dx.doi.org/10.1016/j.envint.2014.06.003

Gorman, R; Rinsky, R; Stein, G; Anderson, K. (1984). Health hazard evaluation report no. HETA 82-075-1545, Pratt & Whitney Aircraft, West Palm Beach, Florida. (HETA 82-075-1545). Cincinnati, OH: National Institute for Occupational Safety and Health.

Halogenated Solvents Industry Alliance, I. (2017). RE: Docket no. EPA-HQ-2016-0737. (EPA-HQ-OPPT-2016-0737-0027). Washington, D.C. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0027

Halogenated Solvents Industry Alliance, I. (2018). Re: Docket no. EPA-HQ-OPPT-2016-0737. (EPA-HQ-OPPT-2016-0737-0103). Washington, D.C. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0103

Hellweg, S; Demou, E; Bruzzi, R; Meijer, A; Rosenbaum, RK; Huijbregts, MA; Mckone, TE. (2009). Integrating human indoor air pollutant exposure within Life Cycle Impact Assessment [Review]. Environ Sci Technol 43: 1670-1679. http://dx.doi.org/10.1021/es8018176

ICF Consulting. (2004). The U.S. solvent cleaning industry and the transition to non ozone depleting substances. https://www.epa.gov/sites/production/files/2014-11/documents/epasolventmarketreport.pdf

IRTA (Institute for Research and Technical Assistance). (2007). Spotting chemicals: Alternatives to perchloroethylene and trichloroethylene in the textile cleaning industry. Prepared for: Cal/EPAs Department of Toxic Substances Control and U.S. Environmental Protection Agency Region IX. http://www.irta.us/reports/DTSC%20Spotting%20Chemical%20for%20Web.pdf

Kanegsberg, B; Kanegsberg, E. (2011). Handbook for critical cleaning, cleaning agents and systems (2nd ed.). Boca Raton, FL: CRC Press.

Kasting, BG; Miller, MA. (2006). Kinetics of finite dose absorption through skin 2: Volatile compounds. J Pharm Sci 95: 268-280. http://dx.doi.org/10.1002/jps.20497

Kinnes, GM. (1998). Health hazard evaluation report no. HETA 97-0214-2689, Dorma Door Controls, Inc., Reamstown Pennsylvania. (HETA 97-0214-2689). Cincinnati, OH: National Institute for Occupational Safety and Health.

Klein, P; Kurz, J. (1994). [Reduction of Solvent Concentrations in Surroundings of Dry-Cleaning Shops]. Bonningheim, Germany: Hohenstein Physiological lnstitute on Clothing.

Lewis, FA. (1980). Health hazard evaluation report no. HHE 80-87-708, Harowe Servo Contorls Inc., West Chester, Pennsylvania. (HHE 80-87-708). Cincinnati, OH: National Institute for Occupational Safety and Health.

Marquart, H; Franken, R; Goede, H; Fransman, W; Schinkel, J. (2017). Validation of the dermal exposure model in ECETOC TRA. Annals of Work Exposures and Health 61: 854-871. http://dx.doi.org/10.1093/annweh/wxx059

Morford, RG. (2017). Comment submitted by Richard G. Morford, General Counsel, Enviro Tech International, Inc. Available online at https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0741-0016

Morris, M; Wolf, K. (2005). Evaluation of New and Emerging Technologies for Textile Cleaning. Institute for Research and Technical Assistance (IRTA). http://wsppn.org/pdf/irta/Emerging_Technologies_Textile_%20Clea.pdf

Most, CC. (1989). Locating and estimating air emissions from sources of perchloroethylene and trichloroethylene. (EPA-450/2-89-013). Research Triangle Park, NC: U.S. EPA. https://www3.epa.gov/ttn/chief/le/perc.pdf

NEWMOA (Northeast Waste Management Officials' Association). (2001). Pollution prevention technology profile - Closed loop vapor degreasing. Boston, MA. http://www.newmoa.org/prevention/p2tech/ProfileVaporDegreasing.pdf

NIH. (2012). Hazardous Substances Data Bank (HSDB): Trichloroethylene. Bethesda, MD: U.S. Department of Health & Human Services, National Institutes of Health, U.S. National Library of Medicine. https://toxnet.nlm.nih.gov/newtoxnet/hsdb.htm

NIOSH (National Institute for Occupational Safety and Health). (1997). Control of health and safety hazards in commercial drycleaners: chemical exposures, fire hazards, and ergonomic risk factors. In Education and Information Division. (DHHS (NIOSH) Publication Number 97-150). Atlanta, GA. http://www.cdc.gov/niosh/docs/97-150/

NIOSH (National Institute for Occupational Safety and Health). (2001). Evaluation of Solvent Exposures from the Degreaser. Trilthic Inc., IN. In Hazard Evaluation Technical Assisstance Branch. (HETA 2000-0233-2845). NIOSH Publishing Office: National Institute of Occupational Safety and Health. http://www.cdc.gov/niosh/hhe/reports/pdfs/2000-0233-2845.pdf

NIOSH. (2002a). In-depth survey report: control of perchloroethylene (PCE) in vapor degreasing operations, site #1. (EPHB 256-19b). Cincinnati, Ohio: National Institute for Occupational Safety and Health (NIOSH).

NIOSH (National Institute for Occupational Safety and Health). (2002b). In-depth survey report: Control of perchloroethylene (PCE) in vapor degreasing operations, site #2. (EPHB 256-16b). CDC. https://www.cdc.gov/niosh/surveyreports/pdfs/256-16b.pdf

NIOSH. (2002c). In-depth survey report: control of perchloroethylene (PCE) in vapor degreasing operations, site #4. (EPHB 256-18b). Cincinnati, Ohio: National Institute for Occupational Safety and Health (NIOSH).

NIOSH (National Institute for Occupational Safety and Health). (2002d). In-depth survey report: Control of perchloroethylene exposure (PCE) in vapor degreasing operations, site #3. (EPHB 256-17b). CDC. https://www.cdc.gov/niosh/surveyreports/pdfs/ECTB-256-17b.pdf

OECD (Organisation for Economic Co-operation and Development). (2004). Emission scenario document on lubricants and lubricant additives. In OECD Series On Emission Scenario Documents. (JT00174617). Paris, France. http://www.olis.oecd.org/olis/2004doc.nsf/LinkTo/env-jm-mono(2004)21

OECD (Organisation for Economic Co-operation and Development). (2009a). Emission scenario document on adhesive formulation. (JT03263583). Paris, France.

OECD (Organisation for Economic Co-operation and Development). (2009b). Emission scenario documents on coating industry (paints, lacquers and varnishes). (JT03267833). Paris, France.

OECD (Organisation for Economic Co-operation and Development). (2010). Scoping Document for Emission Scenario Document on Manufacturing and Use of Printing Inks. OECD Environmental Health and Safety Publications.

OECD (Organisation for Economic Co-operation and Development). (2011a). EMISSION SCENARIO DOCUMENT ON RADIATION CURABLE COATING, INKS AND ADHESIVES. In Series

on Emission Scenario Documents No 27. Paris: OECD Environmental Health and Safety Publications. http://www.oecd-ilibrary.org/docserver/download/9714111e.pdf?expires=1497031939&id=id&accname=guest&checksum=C794B2987D98D1D145225EAF9B482280

OECD (Organisation for Economic Co-operation and Development). (2011b). Emission scenario document on the use of metalworking fluids. In OECD Environmental health and safety publications Series on emission scenario documents Emission scenario document on coating and application via spray painting in the automotive refinishing industry Number 11. (JT03304938). Organization for Economic Cooperation and Development.

OECD (Organisation for Economic Co-operation and Development). (2015). Emission scenario document on use of adhesives. (Number 34). Paris, France. http://www.oecd.org/officialdocuments/publicdisplaydocumentpdf/?cote=ENV/JM/MONO(2015)4&doclanguage=en

OECD. (2017). Draft ESD on Vapor Degreasing - Internal EPA document. Organization for Economic Co-operation and Development (OECD).

OECD. (2019). Emission scenario documents. Available online at http://www.oecd.org/env/ehs/risk-assessment/emissionscenariodocuments.htm (accessed August 29, 2019).

Orris, P; Daniels, W. (1981). Health Hazard Evaluation Report 80-201-816: Peterson/Puritan Company. (HE 80-201-816). NIOSH. https://www.cdc.gov/niosh/hhe/reports/pdfs/80-201-816.pdf?id=10.26616/NIOSHHHE80201816

OSHA (Occupational Safety & Health Administration). (2017). Chemical Exposure Health Data (CEHD) provided by OSHA to EPA. U.S. Occupational Safety and Health Administration.

Products, IAfSDaM. (2012). AISE SPERC fact sheet - wide dispersive use of cleaning and maintenance products. International Association for Soaps Detergents and Maintenance Products. https://www.aise.eu/our-activities/regulatory-context/reach/environmental-exposure-assessment.aspx

Rosensteel, RE; Lucas, JB. (1975). Health hazard evaluation report no. HHE 74-28-212, Westinghouse Air Brake Company, Wilmerding, Pennsyvlania. (HHE 74-28-212). Cincinnati, OH: National Institute for Occupational Safety and Health.

Ruhe, RL. (1982). Health hazard evaluation report no. HETA 82-040-119, Synthes Ltd. (USA), Monument, Colorado. (HETA 82-040-119). Cincinnati, OH: National Institute for Occupational Safety and Health.

Ruhe, RL; Watanabe, A; Stein, G. (1981). Health hazard evaluation report no. HHE 80-49-808, Superior Tube Company, Collegeville, Pennsylvania. (HHE 80-49-808). Cincinnati, OH: National Institute for Occupational Safety and Health. https://www.cdc.gov/niosh/hhe/reports/pdfs/80-49-808.pdf

Saft America, I. (2017). Memorandum: Trichoroethylene, Docket ID number EPA-HQ-OPPT-2016-0737. (EPA-HQ-OPPT-2016-0737-0007). Washington, D.C. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0007

SCG (Scientific Consulting Group, Inc.). (2013). Final peer review comments for the OPPT trichloroethylene (TCE) draft risk assessment. Available online at https://www.epa.gov/sites/production/files/2017-06/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf

Seitz, T; Driscoll, R. (1989). Health hazard evaluation report no. HETA 88-082-1971, Jostens Incorporated, Princeton, Illinois. (HETA 88-082-1971). Cincinnati, OH: National Institute for Occupational Safety and Health. https://www.cdc.gov/niosh/hhe/reports/pdfs/1988-0082-1971.pdf

Smart, BE; Fernandez, RE. (2000). Fluorinated aliphatic compounds [Encyclopedia]. In Kirk-Othmer encyclopedia of chemical technology. Hoboken, NJ: John Wiley and Sons, Inc. http://dx.doi.org/10.1002/0471238961.0612211519130118.a01

Snedecor, G; Hickman, JC; Mertens, JA. (2004). Chloroethylenes and chloroethanes.

Tomer, A; Kane, J. (2015). The great port mismatch. U.S. goods trade and international transportation. The Global Cities Initiative. A joint project of Brookings and JPMorgon Chase. https://www.brookings.edu/wp-content/uploads/2015/06/brgkssrvygcifreightnetworks.pdf

U.S. BLS (U.S. Bureau of Labor Statistics). (2014). Employee Tenure News Release. Available online at http://www.bls.gov/news.release/archives/tenure_09182014.htm

U.S. BLS (U.S. Bureau of Labor Statistics). (2016). May 2016 Occupational Employment and Wage Estimates: National Industry-Specific Estimates. Available online at http://www.bls.gov/oes/tables.htm

U.S. Census Bureau. (2013). Census 2012 Detailed Industry Code List [Database]. Retrieved from https://www.census.gov/topics/employment/industry-occupation/guidance/code-lists.html

U.S. Census Bureau. (2015). Statistics of U.S. Businesses (SUSB). https://www.census.gov/data/tables/2015/econ/susb/2015-susb-annual.html

U.S. Census Bureau. (2019). Survey of Income and Program Participation: SIPP introduction and history. Washington, DC. https://www.census.gov/programs-surveys/sipp/about/sipp-introduction-history.html

U.S. EPA (U.S. Environmental Protection Agency). (1977). Control of volatile organic emissions from solvent metal cleaning [EPA Report]. (EPA-450/2-77-022). Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air and Waste Management, Office of Air Quality Planning and Standards.

U.S. EPA (U.S. Environmental Protection Agency). (1980). Chapter 4.7: Waste Solvent Reclamation. In AP-42 Compilation of air pollutant emission factors (5th ed.). Research Triangle Park, NC: Office of Air and Radiation, Office of Air Quality and Planning Standards. https://www3.epa.gov/ttn/chief/ap42/ch04/index.html

U.S. EPA (U.S. Environmental Protection Agency). (1981). AP-42. Compilation of air pollutant emission factors. Chapter 4.6: Solvent degreasing. Washington, DC. http://www3.epa.gov/ttn/chief/ap42/ch04/final/c4s06.pdf

U.S. EPA (U.S. Environmental Protection Agency). (1985). Occupational exposure and environmental release assessment of tetrachloroethylene. Office of Pesticides and Toxic Substances.

U.S. EPA (U.S. Environmental Protection Agency). (1992). Guidelines for exposure assessment. Federal Register 57(104):22888-22938 [EPA Report]. In Guidelines for exposure assessment. (EPA/600/Z-92/001). Washington, DC. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263

U.S. EPA. (1994). Guidelines for Statistical Analysis of Occupational Exposure Data: Final. United States Environmental Protection Agency :: U.S. EPA.

U.S. EPA (U.S. Environmental Protection Agency). (1997). Solvent Cleaning. Volume III, Chapter 6. pp. 6.2.1. Washington, DC. http://www3.epa.gov/ttnchie1/eiip/techreport/volume03/iii06fin.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2001a). Guide to industrial assessments for pollution prevention and energy efficiency [EPA Report]. (EPA/625/R-99/003). Cincinnati, OH: Office of Research and Development, National Risk Management Research Laboratory, Center for Environmental Research Information.

U.S. EPA (U.S. Environmental Protection Agency). (2001b). Risk assessment guidance for superfund: Volume III - Part A, Process for conducting probabilistic risk assessment [EPA Report]. (EPA 540-R-02-002). Washington, DC: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response.

U.S. EPA (U.S. Environmental Protection Agency). (2006). Risk assessment for the halogenated solvent cleaning source category [EPA Report]. (EPA Contract No. 68-D-01-052). Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. https://www.regulations.gov/document?D=EPA-HQ-OAR-2002-0009-0022

U.S. EPA (U.S. Environmental Protection Agency). (2011). The 2011 National Emissions Inventory. Retrieved from https://www.epa.gov/air-emissions-inventories/2011-national-emissions-inventory-nei-data

U.S. EPA (U.S. Environmental Protection Agency). (2013). ChemSTEER user guide - Chemical screening tool for exposures and environmental releases. Washington, D.C. https://www.epa.gov/sites/production/files/2015-05/documents/user_guide.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2014a). Degreasing with TCE in commercial facilities: Protecting workers [EPA Report]. Washington, DC: U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics.

U.S. EPA (U.S. Environmental Protection Agency). (2014b). TSCA work plan chemical risk assessment. Trichloroethylene: Degreasing, spot cleaning and arts & crafts uses. (740-R1-4002). Washington, DC: Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. http://www2.epa.gov/sites/production/files/2015-09/documents/tce_opptworkplanchemra_final_062414.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2016a). EPA Discharge Monitoring Report Data. Retrieved from https://cfpub.epa.gov/dmr/

U.S. EPA (U.S. Environmental Protection Agency). (2016b). Instructions for reporting 2016 TSCA chemical data reporting. (EPA/600/R-09/052F). Washington, DC: U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics. https://www.epa.gov/chemical-data-reporting/instructions-reporting-2016-tsca-chemical-data-reporting

U.S. EPA (U.S. Environmental Protection Agency). (2017a). Chemical data reporting under the Toxic Substances Control Act. Available online at https://www.epa.gov/chemical-data-reporting (accessed August 29, 2017).

U.S. EPA (U.S. Environmental Protection Agency). (2017b). Preliminary information on manufacturing, processing, distribution, use, and disposal: Trichloroethylene [Comment]. (EPA-HQ-OPPT-2016-0737-003). Washington, DC: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0737-0003

U.S. EPA (U.S. Environmental Protection Agency). (2017c). Toxics Release Inventory (TRI), reporting year 2016. Retrieved from https://www.epa.gov/toxics-release-inventory-tri-program/tri-data-and-tools

U.S. EPA (U.S. Environmental Protection Agency). (2017d). Trichloroethylene market and use report. Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention, Chemistry, Economics, and Sustainable Strategies Division. https://www.epa.gov/sites/production/files/2016-05/documents/instructions_for_reporting_2016_tsca_cdr_13may2016.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2018a). 2014 National Emissions Inventory Report. https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data

U.S. EPA (U.S. Environmental Protection Agency). (2018b). Application of systematic review in TSCA risk evaluations. (740-P1-8001). Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. https://www.epa.gov/sites/production/files/2018-06/documents/final_application_of_sr_in_tsca_05-31-18.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2018c). Problem formulation of the risk evaluation for trichloroethylene. (EPA-740-R1-7014). Washington, DC: Office of Chemical Safety and Pollution Prevention, United States Environmental Protection Agency. https://www.epa.gov/sites/production/files/2018-06/documents/tce_problem_formulation_05-31-31.pdf

U.S. EPA (U.S. Environmental Protection Agency). (2018d). TRI Reporting Forms and Instructions (RFI) Guidance Document. https://ofmpub.epa.gov/apex/guideme_ext/f?p=guideme_ext:41:0::NO:::

U.S. EPA (U.S. Environmental Protection Agency). (2019a). Aluminum forming point source category. (40 CFR Part 467). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=117c2452100f178f42f8141c0887e5f4&mc=true&node=pt40.32.467&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019b). Coil coating point source category. (40 CFR Part 465). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=117c2452100f178f42f8141c0887e5f4&mc=true&node=pt40.32.465&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019c). Electrical and electronic components point source category. (40 CFR Part 469). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=117c2452100f178f42f8141c0887e5f4&mc=true&node=pt40.32.469&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019d). Electroplating Point Source Category. (40 CFR Part 413). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=5c5a19d4dd729db1e53fb9ca47e16706&mc=true&node=pt40.31.413&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019e). Iron and steel manufacturing point source category. (40 CFR Part 420). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=5c5a19d4dd729db1e53fb9ca47e16706&mc=true&node=pt40.31.420&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019f). Metal finishing point source company. (40 CFR Part 433). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=117c2452100f178f42f8141c0887e5f4&mc=true&node=pt40.32.433&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019g). Organic chemicals, plastics, and synthetic fibers. (40 CFR Part 414). Washington, D.C. https://www.ecfr.gov/cgi-bin/text-idx?SID=5c5a19d4dd729db1e53fb9ca47e16706&mc=true&node=pt40.31.414&rgn=div5

U.S. EPA (U.S. Environmental Protection Agency). (2019h). Risk evaluation for trichloroethylene. Washington, D.C.

Vandervort, R; Polakoff, PL. (1973). Health hazard evaluation report no. HHE 72-84-31, Dunham-Bush, Incroprated, West Hartford, Connecticut, Part 2. (HHE 72-84-31). Cincinnati, OH: National Institute for Occupational Safety and Health.

von Grote, J; Hürlimann, C; Scheringer, M; Hungerbühler, K. (2006). Assessing occupational exposure to perchloroethylene in dry cleaning. J Occup Environ Hyg 3: 606-619. http://dx.doi.org/10.1080/15459620600912173

Von Grote, J; Hurlimann, JC; Scheringer, M; Hungerbuhler, K. (2003). Reduction of Occupational Exposure to Perchloroethylene and Trichloroethylene in Metal Degreasing over the Last 30 years: Influence of Technology Innovation and Legislation. J Expo Anal Environ Epidemiol 13: 325-340. http://dx.doi.org/10.1038/sj.jea.7500288

Whittaker, SG; Johanson, CA. (2011). A profile of the dry cleaning industry in King County, Washington: Final report. (LHWMP 0048). Seattle, WA: Local Hazardous Waste Management Program in King County. http://www.hazwastehelp.org/publications/publications_detail.aspx?DocID=Oh73%2fQilg9Q%3d

Young, ML. (2012). Pre-spotting step toward better cleaning. Available online at https://americandrycleaner.com/articles/pre-spotting-step-toward-better-cleaning