Exhibit 375

Expert Report of Lisa A. Bailey, Ph.D.

In the Case of:
Terry Dyer *v.* United States

Prepared by

Lisa Bailey

Lisa A. Bailey, Ph.D.

Prepared for
United States Department of Justice
950 Pennsylvania Avenue NW
Washington, DC 20530

April 8, 2025

GRADIENT
www.gradientcorp.com
One Beacon Street, 17th Floor
Boston, MA 02108
617-395-5000

Table of Contents

Page

List of Tables

List of Figures

Abbreviations

$\mu g/m^3$	Micrograms per Cubic Meter of Air
$(\mu g/m^3)^{-1}$	Per Micrograms per Cubic Meter of Air
1,2-cDCE	*cis*-1,2-dichloroethylene
1,2-tDCE	*trans*-1,2-Dichloroethylene
ACS	American Cancer Society
ADAF	Age-Dependent Adjustment Factor
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BMDL	Lower Confidence Limit on the Benchmark Dose
CI	Confidence Interval
CSF	Cancer Slope Factor
CTE	Central Tendency Exposure
DEC	Daily Exposure Concentration
DED	Daily Exposure Dose
ELCR	Excess Lifetime Cancer Risk
HB	Holcomb Boulevard
HP	Hadnot Point
IARC	International Agency for Research on Cancer
IUR	Inhalation Unit Risk
L	Liter
LADD	Lifetime Average Daily Dose
LADE	Lifetime Average Daily Exposure
LED	Lower Confidence Limit of the Exposure Dose
LNT	Linear No Threshold
MCL	Maximum Contaminant Level
MoE	Margin of Exposure
mg/kg-day	Milligrams per Kilogram Body Weight per Day
$(mg/kg\text{-}day)^{-1}$	Per Milligrams per Kilogram Body Weight per Day
NHL	Non-Hodgkin's Lymphoma
NRC	National Research Council
NTP	National Toxicology Program
OR	Odds Ratio
PBPK	Physiologically Based Pharmacokinetic
PCE	Tetrachloroethylene/Perchloroethylene
POD	Point of Departure
ppm	Parts per Million
ppb	Parts per Billion
RAGS	Risk Assessment Guidance for Superfund
RME	Reasonable Maximum Exposure
SD	Standard Deviation
SDWA	Safe Drinking Water Act
TCE	Trichloroethylene
TT	Tarawa Terrace

TT2	Tarawa Terrace 2
US DOJ	United States Department of Justice
US EPA	United States Environmental Protection Agency
WoE	Weight of Evidence
WTP	Water Treatment Plant

1 Qualifications

I am a Principal at Gradient, an environmental and risk sciences consulting firm that specializes in toxicology, epidemiology, risk assessment, and other disciplines. I have more than 25 years of experience in toxicology and human health risk assessment. I received my Ph.D. in biochemistry from the Massachusetts Institute of Technology in 1996, and I was a post-doctoral fellow at the Harvard School of Public Health from 1996 to 1999. I have expertise in toxicology, molecular biology, genetic toxicology and mutagenesis, mechanisms of carcinogenesis, weight-of-evidence (WoE) evaluations and systematic review, and risk communication.

My expertise in WoE evaluations includes systematic review and in-depth evaluation and integration of all data relevant to a particular chemical and its potential association with human disease (*i.e.*, toxicokinetics data, animal toxicity data, epidemiology data, mechanistic data, and human exposure data). I have conducted in-depth WoE evaluations of many chemicals and have published several papers describing the results of my analyses.

I also have expertise in conducting human health risk assessments for environmental, consumer product, and occupational exposures. In order to assess whether exposure (*via* inhalation, dermal contact, or ingestion) to a particular substance may be associated with potential human health risk, both hazard and exposure (including the level, duration, and frequency of exposure) need to be considered, and only when the two combined are sufficient to cause disease in humans is there cause for concern. Therefore, my expertise in human health risk assessment consistently involves in-depth evaluation of the potential hazards of chemicals in addition to consideration of the extent to which humans are exposed to the chemicals of concern in the environment, consumer products, or the workplace.

I have authored many peer-reviewed articles and book chapters in the field of human health risk and toxicology and have presented my scientific findings and analyses at conferences, to community groups, and to regulatory agencies. I am also a full member of the Society of Toxicology and the Society for Risk Analysis.

Gradient is currently being compensated at the rate of $595 per hour for my work in this matter. My *curriculum vitae* is attached as Appendix A. My testimony experience is attached as Appendix B. Appendix C lists all the materials I considered in the preparation of this report.

2 Introduction and Executive Summary

This report was prepared at the request of the United States Department of Justice (US DOJ). As part of my engagement in this case, I have been asked to review materials relevant to the case *Terry Dyer v. United States* and to develop opinions related to whether there is scientific support for the plaintiff's claim that exposure to chemicals in tap water (trichloroethylene [TCE], tetrachloroethylene [also known as perchloroethylene (PCE)], vinyl chloride, benzene, and *trans*-1,2-dichloroethylene [1,2-tDCE]) while employed and residing at Camp Lejeune is causally associated with the plaintiff's bladder cancer diagnosis.

My report includes:

- An executive summary (Section 2.1);
- An overview of the general risk assessment methodology I applied to evaluate risk for the plaintiff (Section 3);
- A brief discussion of the history of the Marine Corps Base Camp Lejeune Site (Section 4);
- Hazard evaluation summaries (based on the expert report by Dr. Julie Goodman [2025]) and summaries of the regulatory toxicity criteria used to calculate risks for TCE, PCE, vinyl chloride, benzene, and 1,2-tDCE (Section 5);
- A plaintiff-specific risk evaluation, based on exposure information provided in the expert report by Dr. Judy LaKind (2025) (Section 6);
- A comparison of the estimated exposures for the plaintiff to exposures from the animal or human studies that are the basis of the chemical-specific toxicity criteria (Section 7);
- A comparison of the estimated exposures for the plaintiff to exposure information from relevant epidemiology or animal studies (Section 8);
- A rebuttal of the plaintiff's experts' reports (Section 9); and
- A summary of my opinions related to the plaintiff's claim that exposures to chemicals in tap water while employed/residing at Camp Lejeune are related to the plaintiff's diagnosis (Section 10).

2.1 Executive Summary

Section 3 of this report provides a discussion of the general approach to toxicology and risk assessment and regulatory risk assessment guidelines.

- Toxicology is the study of health effects resulting from exposure to chemical, biological, or physical agents. One of the most fundamental concepts in the field of toxicology is the dose-response relationship; dose is the amount of a chemical to which an organism is exposed, and a response is the effect on the organism resulting from the chemical exposure. A dose-response relationship occurs when the chemical exposure and the effect are correlated, and the effect (response) increases directly with increased exposure (dose). For most chemicals, biological effects (with a dose-response relationship) occur only when the dose exceeds a certain exposure level for a sufficient period of time. It is common for dose-response data from toxicology investigations to be used in risk assessment, which is a tool used to predict adverse health effects based on knowledge of the effects of chemicals and exposures.

- Human health risk assessment is the systematic process of characterizing potential adverse human health effects resulting from exposure to environmental chemicals. Risk assessment generally involves four steps:
  - **Hazard Identification:** Identify the potential hazard (*i.e.*, determine whether a particular chemical is causally linked to any health effects).
  - **Dose-Response Assessment:** Determine the relationship between the nature and magnitude of exposure to the hazard and the probability of a health effect occurring.
  - **Exposure Assessment:** Estimate the level of human exposure to the hazard.
  - **Risk Characterization:** Compare the estimated human exposure level of concern to the dose-response assessment for the chemical and characterize the comparison as a risk estimate, then assess the magnitude of uncertainty in the risk estimate.
- The United States Environmental Protection Agency (US EPA) has derived toxicity criteria for many chemicals based on its **hazard and dose-response assessments** of those chemicals.
  - Toxicity criteria are quantitative estimates of risk of the adverse health effects associated with a given chemical exposure level. Toxicity criteria are typically derived from observations of chemical exposures and health effects reported in epidemiology or animal studies, and are conservatively based on the most sensitive endpoint reported in the health effect studies (*i.e.*, the health effect occurring at the lowest exposure level). They are also designed to be protective of the most sensitive populations (*e.g.*, children and the elderly). Therefore, US EPA's toxicity criteria reflect conservative estimates of the relationship between exposures and health effects (*i.e.*, overly protective assumptions about exposures and health effects), particularly for short exposure durations for healthy individuals in a population.
  - The cancer toxicity criteria derived by US EPA are referred to as the cancer slope factor (CSF), which is used to characterize risk from oral and dermal exposures, and the inhalation unit risk (IUR), which is used to characterize risk from inhalation exposure. These criteria are derived based on the most sensitive cancer endpoint evaluated in the available studies. CSFs are described as risks per milligrams per kilogram body weight per day (or $[\text{mg/kg-day}]^{-1}$). IURs are described as risks per microgram per cubic meter of air (or $[\mu g/m^3]^{-1}$]). For example:
    - A CSF of 0.01 $(\text{mg/kg-day})^{-1}$ is equivalent to a risk of 1 in 100 or 1% (1 cancer case in 100 people exposed) from exposure to 1 milligram per kilogram body weight per day (mg/kg-day) of a chemical over a lifetime (70 years) of oral or dermal exposure.
    - An IUR of 0.01 $(\mu g/m^3)^{-1}$ is equivalent to a risk of 1 in 100 or 1% (1 cancer case in 100 people exposed) from continuous exposure to 1 microgram per cubic meter of air ($\mu g/m^3$) of a chemical over a lifetime (70 years) of inhalation exposure.
- In the **exposure assessment** step in the risk assessment, daily oral or dermal doses of a chemical taken into the body, averaged over the appropriate exposure period, and expressed in units of mg/kg-day are estimated for an individual. Similarly, inhalation exposure concentrations, averaged over the appropriate exposure period, and expressed in units of $\mu g/m^3$ are estimated for an individual.
- In her expert report (LaKind, 2025), Dr. LaKind describes the daily exposure doses (DEDs) for oral and dermal exposures and daily exposure concentrations (DECs) for inhalation exposures calculated for the plaintiff for each chemical. Using the plaintiff-specific DED and DEC estimates from Dr. LaKind (2025), the exposure frequency (how often exposure occurs, in terms of days per

year), and exposure duration (how long the exposure was, in terms of years), for the plaintiff, and an averaging time (the period over which the exposure is averaged) of 70 years, or 25,550 days (consistent with US EPA regulatory guidelines for cancer risk estimates), I calculated the plaintiff's lifetime average daily doses (LADDs) for oral and dermal chemical exposures and the lifetime average daily exposures (LADEs) for inhalation chemical exposures for the plaintiff.

- I calculated the plaintiff's lifetime average daily doses (LADDs) as follows:
  - LADD = (DED × EF × ED) ÷ AT, where:

    LADD = Lifetime Average Daily Dose (mg/kg-day)

    DED = Daily Exposure Dose (mg/kg-day)

    EF = Exposure Frequency (days/year)

    ED = Exposure Duration (years)

    AT = Averaging Time (25,550 days)
- I calculated the plaintiff's lifetime average daily exposures (LADEs) as follows:
  - LADE = (DEC × EF × ED) ÷ AT, where:

    LADE = Lifetime Average Daily Exposure (μg/m^3)

    DEC = Daily Exposure Concentration (μg/m^3)

    EF = Exposure Frequency (days/year)

    ED = Exposure Duration (years)

    AT = Averaging Time (25,550 days)

▪ In the **risk characterization** step in the risk assessment, the estimated human exposure levels of concern (LADD or LADE, as described above) are combined with the dose-response assessment (toxicity criteria [CSF or IUR]) for each chemical to calculate risk estimates for each chemical and exposure pathway (*i.e.*, ingestion, dermal contact, or inhalation).

- Cancer toxicity criteria are used in regulatory risk evaluations to estimate the incremental risk of developing cancer following a specific chemical exposure, beyond the background cancer risk. US EPA refers to this risk as the excess lifetime cancer risk (ELCR), which is expressed as a unitless probability (*e.g.*, 1 cancer case in 1 million people exposed, or 1×10^{-6}).
  - US EPA has established a target ELCR range of 1×10^{-6} (1 cancer case in 1,000,000 people exposed) to 1×10^{-4} (1 cancer case in 10,000 people exposed); an exposure that may result in an ELCR that falls within this range, that is calculated using conservative assumptions, is considered acceptable by US EPA (1990, 1991).
  - To provide perspective on what a target ELCR of 1 in 10,000 or 1 in 1,000,000 means, it is helpful to understand how these risks compare to the overall lifetime probability of being diagnosed with cancer. According to the American Cancer Society (ACS), the lifetime probability of developing any cancer (*i.e.*, background lifetime cancer risk for all cancers combined) is approximately 40% on average across the population (ACS, 2024a). Individual risk will vary and is based on a number of different factors, including age, sex, race, lifestyle (*e.g.*, diet, exercise), and family history.
  - US EPA's acceptable ELCR range of 1×10^{-6} (1 cancer case in 1 million people exposed, or 0.0001% probability) to 1×10^{-4} (1 cancer case in 10,000 people exposed, or 0.01% probability) is well below the background lifetime probability of developing cancer (*i.e.*, ~40% overall in the population) – the equivalent of a total cancer risk range of 40.0001-

40.01%. Therefore, any exceedance of the regulatory cancer risk target should be interpreted carefully, and not be taken to mean that health effects are expected to occur for any particular individual as a result of that exceedance.

- Cancer risk (ELCR) from oral or dermal exposures to a chemical is calculated by multiplying the lifetime oral or dermal dose of that chemical (LADD) by the chemical-specific CSF, as follows:
  - ELCR from Oral or Dermal Exposure = LADD (mg/kg-day) × CSF ($[\text{mg/kg-day}]^{-1}$)
- Similarly, cancer risk (ELCR) from inhalation exposure to a chemical is calculated by multiplying the lifetime inhalation exposure concentration of that chemical (LADE) by the chemical-specific IUR, as follows:
  - ELCR from Inhalation Exposure = LADE ($\mu g/m^3$) × IUR ($[\mu g/m^3]^{-1}$)
- As an example risk calculation, applying a CSF of 0.01 $(\text{mg/kg-day})^{-1}$ to an LADD of 0.005 mg/kg-day would result in the following risk calculation: 0.005 mg/kg-day × 0.01 $(\text{mg/kg-day})^{-1}$ = an excess risk (ELCR) of 0.00005 (or 5 cancer cases in 100,000 people exposed, or 5×10^{-5}, or 0.005%). This ELCR falls within US EPA's target risk range and is considered acceptable by US EPA.

Section 4 briefly describes the history of the Marine Corps Base Camp Lejeune Site. Operations at Camp Lejeune started in late 1941. Multiple water treatment plants (WTPs)[1] have serviced the Camp Lejeune base, including Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB). The HP WTP was the first plant to come online in 1942, and serviced the base until the TT and HB WTPs came online in 1952 and in the summer of 1972, respectively (Hennet, 2024). In the early 1980s, the groundwater sources for two of the WTPs that serviced the Camp Lejeune base (HP and TT) were found to be contaminated with volatile organic compounds. Although the groundwater source for the HB WTP was not contaminated, the HB water system was contaminated when its drinking water was supplied by the HP WTP in the spring and summer months from 1972 through 1985 (ATSDR, 2017a). The contaminants identified in the drinking water at the HP WTP were TCE, PCE, vinyl chloride, 1,2-tDCE, and refined petroleum products (including benzene) (ATSDR, 2017a). The contaminants identified in the drinking water at the TT WTP were TCE, PCE, vinyl chloride, and 1,2-tDCE (ATSDR, 2017a).

As summarized in the hazard evaluations in Section 5, the Agency for Toxic Substances and Disease Registry (ATSDR), in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" (ATSDR, 2017b), concluded that there was "sufficient evidence for causation" for PCE exposure and bladder cancer, and the evidence for causation was "below equipoise" for exposure to TCE, benzene, or vinyl chloride and bladder cancer. ATSDR (2017b) provided no comment on whether there is a causal association between 1,2-tDCE exposure and bladder cancer. As discussed in Section 5, US EPA and the International Agency for Research on Cancer (IARC) have concluded that there is limited evidence in humans for an association between bladder cancer and PCE exposure. US EPA concluded that there is some scientific evidence that TCE can cause bladder cancer in humans, but also noted that the available studies have limitations that hinder interpretation of study results. Also as discussed in Section 5, IARC and the National Toxicology Program (NTP) did not conclude exposure to TCE is associated with increased bladder cancer risk in humans. US EPA and IARC do not conclude that there is an association between exposure to benzene, vinyl chloride, or 1,2-tDCE and bladder cancer. Dr. Goodman also concluded that,

[1] Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB) supplied drinking water to residences and workplaces at Camp Lejeune (see Hennet [2024]). Additional Camp Lejeune water-distribution systems which were not contaminated include: Marine Corps Air Station New River, Onslow Beach, Courthouse Bay, Camp Geiger, Rifle Range, and Montford Point/Camp Johnson (Hennet, 2024).

overall, the scientific evidence does not provide support for causal associations between exposure to TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE and bladder cancer (Goodman, 2025).

Section 5 also summarizes the US EPA toxicity criteria used in the cancer risk evaluation for the plaintiff. Because the scientific evidence does not support, or only provides limited support for, an association between bladder cancer and exposure to TCE, PCE, benzene, or vinyl chloride,[2] the cancer toxicity criteria for these chemicals are not based on bladder cancer, and therefore are not predictive of, and are overly conservative for, bladder cancer risk from exposure to these chemicals. However, I conservatively apply the criteria for these chemicals to estimate the plaintiff's overall excess lifetime cancer risk.

In Section 6, I calculate cancer risks based on exposure estimates for Ms. Dyer. Ms. Dyer lived and attended school at Camp Lejeune from August 1958 through January 1973 (from ages 2 through 16), for a total of 14.5 years. Ms. Dyer lived with her family at various addresses in Tarawa Terrace (TT) throughout their time on-base, except for one year (June 1964 to May 1965) when they resided in Jacksonville, North Carolina. Ms. Dyer testified that she attended elementary school in TT and junior high and high schools in Hadnot Point (HP). In contrast to her testimony, Ms. Dyer's school records indicate that she attended school off-base from 1964 to 1965. However, I conservatively assumed she attended school on-base during that year, as alleged in her testimony. In addition, Ms. Dyer stated that she and her family visited swimming pools on-base. For my risk calculations, I used TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE exposure estimates for Ms. Dyer from tap water (*via* ingestion of drinking water, and *via* dermal and inhalation exposure to shower vapor) calculated by Dr. LaKind (2025) (DED and DEC estimates, as discussed earlier) for the two main areas of concern for groundwater contamination at Camp Lejeune (HP and TT). I combined this information with the regulatory toxicity criteria summarized in Section 5 to conduct a conservative regulatory risk evaluation for Ms. Dyer. Risks were calculated for the following exposure pathways and scenarios for the exposure period of concern (approximately 14.5 years) for Ms. Dyer:

- **Baseline Exposure Pathways:**
  - <u>Drinking Water Ingestion</u> – For this exposure pathway, I evaluated three scenarios for both the HP and TT WTPs: (1) central tendency exposure (CTE), which assumes ingestion of 0.34-0.56 liters (L) of tap water per day from ages 2 through 16; (2) reasonable maximum exposure (RME), which assumes ingestion of 0.85-1.8 L of tap water per day from ages 2 through 16; and (3) a custom high-end exposure scenario, which is a combination of the RME tap water consumption rates from ages 2 through 10 combined and a consumption rate of 2.4 L per day for ages 11 through 16.
  - <u>Dermal and Inhalation Exposures from Showering</u> – For these exposure pathways, I calculated risks based on the CTE (50^{th} percentile) and RME (95^{th} percentile) dermal dose and inhalation concentration outputs from both a residential shower model and a communal showering facility exposure model (ATSDR, 2024a). For Ms. Dyer, I calculated residential shower risks for TT based on the plaintiff's locations of residence during her time at Camp Lejeune, and risks from showering at school for HP based on the locations of her junior high and high schools; these doses and concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Note that the residential shower model accounts for additional household water uses, including appliances, sinks, and toilets, and the communal shower model accounts for additional water uses, including sinks and toilets in the communal facility.

[2] Note that as discussed in Section 5, US EPA does not consider *trans*-1,2-dichloroethylene (1,2-tDCE) to be carcinogenic. Therefore, there are no cancer toxicity criteria available for 1,2-tDCE.

- **Additional Exposure Pathway:**
  - Inhalation from Swimming – For this exposure pathway, I calculated risks based on estimated air concentrations for an indoor swimming pool. Because Ms. Dyer did not specify the location of the swimming pool(s) that she visited, I calculated risks for the HP and TT WTPs. Based on statements Ms. Dyer made in her declaration (Dyer, 2025), I assumed that she visited the indoor pools once per week from ages 2-12, then twice per month from age 13 until her family left Camp Lejeune. The air concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025).
- **Exposure Scenarios Evaluated for Ms. Dyer:**
  - The CTE exposure scenario, which includes the following exposure pathways: CTE drinking water ingestion (TT and HP WTPs), CTE dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).
  - The RME exposure scenario, which includes the following exposure pathways: RME drinking water ingestion (TT and HP WTPs), RME dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).
  - The custom high-end exposure scenario, which includes custom high-end drinking water ingestion (TT and HP WTPs), RME dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).

Based on standard risk assessment methodology, which includes overly health-protective assumptions about exposure and risk, the maximum risk estimate calculated for Ms. Dyer's estimated exposures (1×10^{-4}, or 1 cancer cases in 10,000 exposed people, or 0.01% risk), for all the exposure scenarios evaluated, does not exceed US EPA's target excess cancer risk of 1 in 10,000 (*i.e.*, 1 cancer case in 10,000 exposed people, or 0.01%).

It is notable that the majority of this calculated ELCR is driven by vinyl chloride and TCE exposures. As noted above, ATSDR, US EPA, and Dr. Goodman did not conclude that vinyl chloride exposure is associated with increased bladder cancer risk in humans. ATSDR, NTP, IARC, and Dr. Goodman also did not conclude that TCE is associated with increased bladder cancer risk, with US EPA concluding that only limited evidence is available for TCE and bladder cancer. ATSDR (2017b) states that there is "sufficient evidence for causation" for exposure to PCE and bladder cancer based on limited epidemiology evidence. The PCE risk estimates based on Ms. Dyer's estimated exposures (2×10^{-5}, or 2 cancer cases in 100,000 exposed people, or 0.002% risk) are within US EPA's acceptable cancer risk range. In addition, the PCE cancer toxicity criteria are overly protective for bladder cancer, because the values are based on the more sensitive liver cancer endpoint, not bladder cancer. Therefore, the calculated ELCRs for Ms. Dyer are not reflective of bladder cancer risk.

In Section 7, I compare the plaintiff-specific doses and exposure concentrations to the doses or exposure concentrations that are the basis of the toxicity criteria (predicted to be associated with no, or a very low, response from animal or human studies) before linear extrapolation to derive the toxicity criteria. These comparisons are called margins of exposure (MoEs), and are equal to the doses or exposure concentrations that are the basis of the toxicity criteria divided by the plaintiff-specific doses or exposure concentrations. MoEs above 1 provide support that adverse health effects would not be expected for the individual. Based on these comparisons for Ms. Dyer's exposures, the MoEs range from 278 to 300,000,000, all of which are well above 1, providing additional support that Ms. Dyer's exposures would not have been expected to lead to her bladder cancer.

Further, in Section 8, I consider comparisons of the plaintiff's exposure estimates to exposures in relevant epidemiology and animal studies. As discussed, Ms. Dyer's exposure estimates are orders of magnitude below the concentrations in these studies, providing additional support that Ms. Dyer's exposures would not have been expected to lead to her bladder cancer.

Based on the results of my analysis described above, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Ms. Dyer's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 14.5 years that she lived and spent time at Camp Lejeune are causally associated with her bladder cancer.

I reserve the right to amend my opinion in the future should new information become available to me.

3 Methodology

3.1 General Methodology

The opinions herein are based on my training and experience in toxicology and risk assessment, and on a review of documents available to me as of the date of this report. Specific documents I have reviewed are presented in the references section of this report. In addition, there are many documents that I have reviewed in my professional history that supported my understanding of this case but are not cited specifically in this report. The types of information I relied upon for my analyses include the following:

- Case-specific documents, including:
  - Expert report of Dr. Goodman (2025), which address general causation information regarding exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE;
  - Expert report of Dr. LaKind (2025) regarding exposure information for the plaintiff;
  - Expert reports of Drs. Hennet (2024) and Spiliotopoulos (2024) regarding groundwater modeling for Camp Lejeune;
  - Expert reports submitted on behalf of Ms. Dyer by Drs. Reynolds (2025a,b), Hatten (2025), Longo (2025), and Bird (2025);
  - Plaintiff's deposition; and
  - Other plaintiff materials, if available, as cited within (*e.g.*, declaration, housing records, school records).
- Camp Lejeune evaluations conducted by ATSDR related to potential health effects from exposure to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE in groundwater.
- General toxicology and risk assessment guidance documents authored by agencies such as US EPA and ATSDR.
- Publicly available environmental and regulatory documents that are not case specific, but provide data and information relevant to my analyses. Such documents include chemical-specific toxicity criteria and toxicological reviews.
- Scientific literature specifically related to chemicals (TCE, PCE, vinyl chloride, benzene, and 1,2-tDCE) and exposures associated with the Camp Lejeune litigation.

The specific analyses I performed for my evaluation are briefly stated below.

- Reviewed the plaintiff's deposition and other relevant materials (*e.g.*, housing and school records);
- Reviewed information related to possible associations between exposures to TCE, PCE, vinyl chloride, benzene, and 1,2-tDCE in tap water and the health effects alleged by the plaintiff, based on information provided in the expert report prepared by Dr. Goodman (2025);
- Applied standard risk assessment methodology to conduct a risk evaluation for the plaintiff using plaintiff-specific doses calculated and supplied to me by Dr. LaKind (2025), based on Dr. LaKind's and my agreement on exposure assumptions appropriate for the plaintiff;

- Conducted a margin of exposure (MoE) analysis, comparing the estimated exposures for the plaintiff to exposures from the animal or human studies that are the basis of the chemical-specific toxicity criteria; and
- Compared the estimated exposures for the plaintiff to exposure information from relevant epidemiology or animal studies.

The following sections provide more information about methodologies for toxicology, human health risk assessment, and regulatory risk evaluation.

3.2 Introduction to Toxicology

Toxicology is the study of health effects resulting from exposure to chemical, biological, or physical agents. An understanding of the scientific principles in the field of toxicology is necessary for evaluating the potential for a causal relationship between exposure to chemicals and health effects. One of the most fundamental concepts in the field of toxicology is the dose-response relationship; dose is the amount of a chemical to which an organism is exposed, and a response is the effect on the organism resulting from the chemical exposure. A dose-response relationship occurs when the chemical exposure and the effect are correlated, and the effect (response) increases directly with increased exposure (dose). However, for most chemicals, biological effects (with a dose-response relationship) occur only when the dose exceeds a threshold level for a certain period of time. At doses ranging between zero and the threshold, biochemical or physiological mechanisms can negate a chemical's effects, thereby preventing any adverse effects from occurring. As the magnitude and duration of exposure begin to exceed the threshold, these protective mechanisms can become less effective. Consequently, at exposure levels higher than the threshold for a given chemical, the effect begins to appear in a manner that corresponds to the increase in dose. It is common for dose-response data from toxicology investigations to be used in risk assessment, which is a tool used to predict adverse health effects based on knowledge of the effects of chemicals and exposures.

3.3 Introduction to Human Health Risk Assessment

Human health risk assessment is the systematic process of characterizing potential adverse human health effects resulting from exposure to environmental hazards (NRC, 1983). Risk assessment generally involves four steps that were first presented by the National Academy of Sciences in 1983 (NRC, 1983).

1. **Hazard Identification:** Identify the potential hazard (*i.e.*, determine whether a particular chemical is causally linked to any health effects).
2. **Dose-Response Assessment:** Determine the relationship between the nature and magnitude of exposure to the hazard and the probability of a health effect occurring.
3. **Exposure Assessment:** Estimate the level of human exposure to the hazard.
4. **Risk Characterization:** Compare the estimated human exposure level of concern to the dose-response assessment for the chemical and characterize the comparison as a risk estimate, then assess the magnitude of uncertainty in the risk estimate.

The hazard identification steps for TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE are described in more detail in Dr. Goodman's expert report (Goodman, 2025), and are summarized in Section 5 of my report. The exposure assessment for the plaintiff is introduced below and described in more detail in Dr. LaKind's expert report (LaKind, 2025) and in Section 6 of my report.

Below, I provide more detail on the general approach for the dose-response assessment and risk characterization steps of a risk assessment, including discussion of US EPA's hazard and dose-response approach for the derivation of regulatory toxicity criteria. Because bladder cancer is the health effect of concern for this plaintiff, in this section, I have focused the dose-response and risk characterization methodology discussions on cancer risk evaluations.

3.3.1 Dose-Response Assessment

A dose-response assessment characterizes the relationship between the nature and magnitude of exposure to a chemical of concern and the probability that one or more adverse health effects may result from that exposure. Regulatory agencies rely on dose-response assessments to derive chemical-specific toxicity criteria for use in evaluating potential cancer risks from oral, dermal, or inhalation exposures of concern (see Section 3.3.2).

The following section describes the derivation of cancer toxicity criteria used in regulatory risk assessments.

3.3.1.1 Derivation of Cancer Toxicity Criteria

Regulatory toxicity criteria for cancer and noncancer effects, such as those established by US EPA and ATSDR, are typically derived from observations of chemical exposures and health effects reported in epidemiology or animal studies, and are conservatively based on the most sensitive endpoint reported in the health effect studies (*i.e.*, the health effect occurring at the lowest exposure level). They are designed to be protective of the most sensitive populations (*e.g.*, children and the elderly). Therefore, toxicity criteria reflect conservative estimates of the relationship between exposures and health effects (*i.e.*, overly protective assumptions about exposures and health effects), particularly for short exposure durations for healthy individuals in a population.

US EPA and ATSDR apply standard risk assessment methodologies to estimate the dose-response relationship between chemical exposures and health effects in epidemiology or animal studies. Then, based on that relationship and an understanding of the mechanism of action for a particular chemical (if known) and the associated health effect, these regulatory agencies derive an exposure concentration or dose that is predicted to be associated with no (or a very low) response. This exposure concentration or dose is referred to as the point of departure (POD) (US EPA, 2021), from which cancer and noncancer toxicity criteria are typically derived. Because the plaintiff was diagnosed with bladder cancer, the process for deriving regulatory cancer toxicity criteria is described below.

The cancer toxicity criteria derived by US EPA are referred to as the cancer slope factor (CSF), which is used to characterize risk from oral and dermal exposures, and the inhalation unit risk (IUR), which is used to characterize risk from inhalation exposure. Dose-response information from studies used to derive toxicity criteria can be plotted graphically as the relationship between the magnitude of the response (*i.e.*, health effect) observed at each evaluated chemical dose (referred to as a "dose-response curve"). See Figure 3.1 for an example of a dose-response curve. CSF and IUR values are typically derived by drawing a line from the POD (the dose associated with no, or a very low, response in animal or human studies) on the dose-response curve down to the point of origin (or zero-response).

US EPA often uses a benchmark dose (BMD) modeling approach (US EPA, 2012a) to develop dose-response curves and PODs for the derivation of toxicity criteria. US EPA uses the 95% upper bound on the dose-response curves for these derivations, stating that "[t]he use of upper bounds generally is considered to be a health-protective approach for covering the risk to susceptible individuals" (US EPA, 2005). Using the upper bound on the response results in a lower POD, called the lower confidence limit on the benchmark dose (BMDL). See Figure 3.2 for an example of linear extrapolation from a POD, based on a BMD/BMDL, for the derivation of toxicity criteria (*e.g.*, CSF or IUR). Depending on how the POD is derived, it can sometimes be referred to as a lower confidence limit of the exposure dose (LED). For cancer toxicity criteria, both the BMDL and LED values are typically associated with a cancer risk in the range of 1-10%.

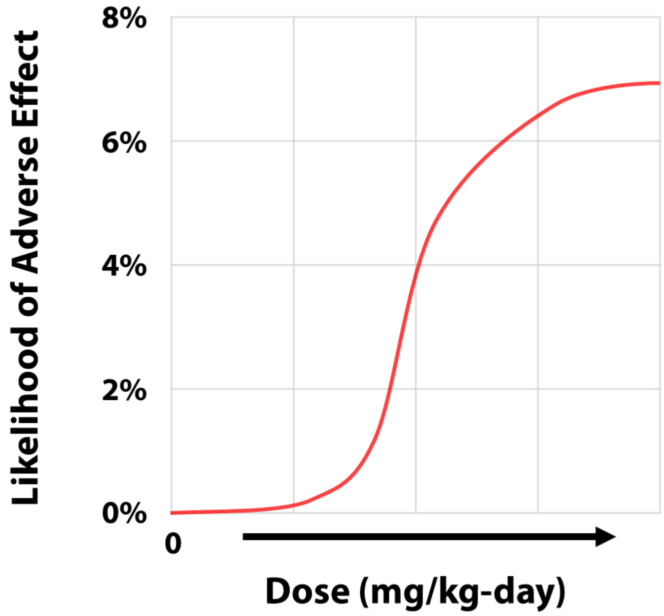

Figure 3.1 Dose-Response Curve

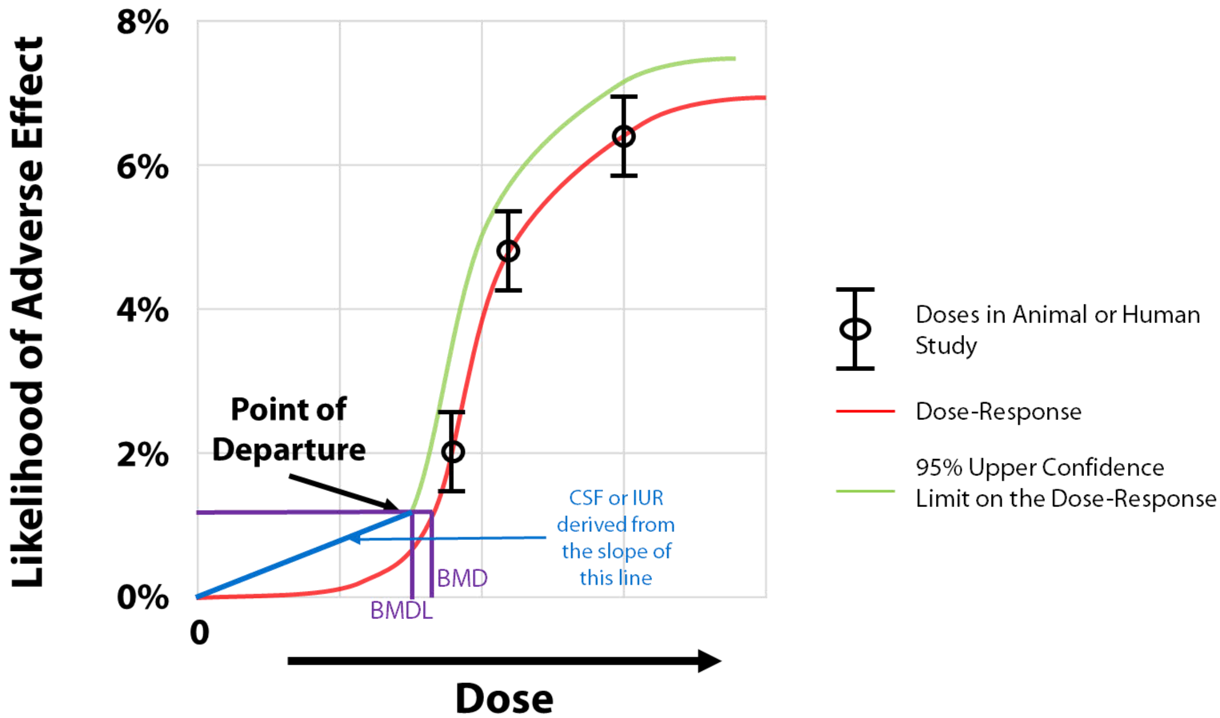

Figure 3.2 Approach for Cancer Slope Factor (CSF) or Inhalation Unit Risk (IUR) Development. BMD = Benchmark Dose; BMDL = Lower Confidence Limit on the Benchmark Dose.

CSFs are used to estimate the probability of an individual developing cancer as a result of a lifetime of oral or dermal exposure to a particular amount of a potential carcinogen, described as risks per mg/kg-day (*i.e.*, $[\text{mg/kg-day}]^{-1}$). For example, a CSF of 0.01 $(\text{mg/kg-day})^{-1}$ is equal to a risk of 1 in 100 or 1% from exposure to 1 mg/kg-day of a substance over a lifetime. Similarly, US EPA defines the IUR as the probability of an individual developing cancer from continuous exposure to a particular amount of a potential carcinogen in air, described as risks per $\mu g/m^3$ (or $[\mu g/m^3]^{-1}$). For example, an IUR of 0.01 $(\mu g/m^3)^{-1}$ is equal to a risk of 1 in 100 or 1% from continuous exposure to 1 $\mu g/m^3$ of a substance in air over a lifetime.

Further, for some chemicals for which there is only reliable observational information (*i.e.*, a human or animal study) to derive either a CSF or an IUR, US EPA might conduct what is called a "route-to-route extrapolation" and derive an IUR from a CSF, or *vice versa*, using information about a chemical's absorption, distribution, metabolism, and excretion for the two exposure pathways, as well as assumptions about human and animal body weights and inhalation rates.

3.3.2 Exposure Assessment

Oral or dermal exposure estimates represent the daily dose of a chemical taken into the body, averaged over the appropriate exposure period and expressed in the units of milligram of chemical per kilogram of human body weight per day (mg/kg-day). Inhalation exposure estimates represent the daily exposure concentration of a chemical taken into the body, averaged over the appropriate exposure period and expressed in the units of microgram of a chemical per cubic meter of air ($\mu g/m^3$). The primary source for the exposure equations used in human health risk assessment is US EPA's "Risk Assessment Guidance for Superfund" (RAGS) (US EPA, 1989).

My risk calculations for the plaintiff, which are described in Section 6, start with Dr. LaKind's plaintiff-specific daily doses and daily inhalation exposure concentrations, which I have termed daily exposure doses (DEDs) and daily exposure concentrations (DECs), respectively. Dr. LaKind provides a detailed discussion of the plaintiff's DED and DEC estimates in her report (LaKind, 2025), including discussion of the dermal and shower inhalation exposure models applied and the exposure parameters used in those models. As described in her report, Dr. LaKind calculated plaintiff-specific daily dose and daily inhalation exposure concentration estimates from exposure point concentrations of chemicals in tap water at Camp Lejeune (LaKind, 2025).

The plaintiff's exposure frequency (EF, how often exposure to chemicals occurred) and exposure duration (ED, how long the exposure to chemicals was) are also considered in the risk calculations. A daily exposure frequency of 365 days per year is typically applied for tap water use (ingestion and showering). Exposure duration generally corresponds to the time period that the plaintiff lived or worked at Camp Lejeune. Finally, consistent with US EPA guidance (US EPA, 2014), an averaging time (the period over which the chemical exposures are averaged) was applied to derive the risk estimates. The averaging time that US EPA recommends using to calculate exposure estimates for cancer risk calculations is a 70-year lifetime (*i.e.*, 25,550 days), because the cancer toxicity criteria are based on a lifetime of exposure (US EPA, 2014).

For evaluating oral and dermal exposures for cancer risk estimates, the relevant dose metric is the lifetime average daily dose (LADD), which is defined as the amount of a chemical taken into the body *via* oral or dermal exposure during the exposure period, averaged over a 70-year life lifetime (*i.e.*, 25,550 days). Using the DED estimates from Dr. LaKind, I calculate LADDs for oral and dermal exposures to the chemicals of interest as follows:

$$\mathrm{LADD} = \frac{\mathrm{DED} \times \mathrm{EF} \times \mathrm{ED}}{\mathrm{AT}}$$

where:

LADD	=	Lifetime Average Daily Dose (mg/kg-day)
DED	=	Daily Exposure Dose (mg/kg-day)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
AT	=	Averaging Time (25,550 days)

For evaluating inhalation exposures for cancer risk estimates, the relevant dose metric is the lifetime average daily exposure (LADE), which is defined as the amount of chemical that someone is exposed to *via* inhalation during the exposure period, averaged over a 70-year lifetime (*i.e.*, 25,550 days). Using the DEC estimates from Dr. LaKind, I calculate LADEs for inhalation exposures to the chemicals of interest as follows:

$$\mathrm{LADE} = \frac{\mathrm{DEC} \times \mathrm{EF} \times \mathrm{ED}}{\mathrm{AT}}$$

where:

LADE	=	Lifetime Average Exposure Concentration (µg/m^3)
DEC	=	Daily Exposure Concentration (µg/m^3)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
AT	=	Averaging Time (25,550 days)

See Appendix D for more details on these calculations.

3.3.2.1 Calculations for the Indoor Swimming Inhalation Exposure Pathway

As described in Dr. LaKind's expert report (LaKind, 2025), an indoor swimming inhalation exposure pathway was also evaluated for the plaintiff. As discussed by Dr. LaKind, and consistent with the ATSDR "Public Health Assessment for Camp Lejeune Drinking Water" (ATSDR, 2017a), only the inhalation exposure pathway is considered for the indoor swimming exposure pathway. For this exposure pathway, Dr. LaKind provided an indoor vapor concentration (VC) for each chemical. I calculated a daily exposure concentration (DEC) from the VC, based on the following equation:

$$\mathrm{DEC} = \frac{\mathrm{VC} \times \mathrm{ET}}{24\ \mathrm{hours/day}}$$

where:

DEC	=	Daily Exposure Concentration ($\mu g/m^3$)
VC	=	Vapor Concentration in Pool Area ($\mu g/m^3$)
ET	=	Exposure Time (hours/day)

The lifetime average daily exposure (LADE) for the indoor swimming inhalation exposure pathway is then calculated as follows (slightly modified from the LADE equation discussed earlier to reflect the total number of events that occurred during the exposure duration):

$$\mathrm{LADE} = \frac{\mathrm{DEC} \times \mathrm{EF} \times \mathrm{EV}}{\mathrm{AT}}$$

where:

LADE	=	Lifetime Average Exposure Concentration ($\mu g/m^3$)
DEC	=	Daily Exposure Concentration ($\mu g/m^3$)
EF	=	Exposure Frequency (days/event)
EV	=	Events During Exposure Duration (number of events)
AT	=	Averaging Time (25,550 days)

See Appendix D for details on this calculation.

3.3.3 Risk Characterization for Cancer Health Effects

In the risk characterization step of the risk assessment, the estimated human exposure levels of concern (LADD or LADE, as described above) are combined with the dose-response assessment (chemical-specific toxicity criteria [CSF or IUR]) for each chemical to calculate risk estimates for each chemical and exposure pathway.

3.3.3.1 Cancer Toxicity Criteria Are Used to Estimate the Excess Lifetime Cancer Risk (ELCR) in a Population

Cancer risks are characterized as the incremental probability that an individual will develop cancer during their lifetime due to exposure to a chemical under the specific exposure scenarios evaluated. All individuals have a background risk of developing cancer at some point in their lifetimes. According to the American Cancer Society (ACS), the lifetime probability of developing any cancer (*i.e.*, background cancer risk for all cancers combined) is slightly less than 1 in 2 (41.6%) for men and slightly more than 1 in 3 (39.6%) for women (ACS, 2024a). As described by ACS (2024a), the lifetime probability of developing bladder cancer is 3.6% for men and 1.1% for women, in the population overall. Background cancer risk is based on cancer incidence within the population and does not mean that all individuals are at 40% risk of developing cancer. Individual risk (background or above background) will vary and is based on a number of different factors, including age, sex, race, lifestyle (*e.g.*, diet, exercise), and family history (ACS, 2024a; Mayo Clinic, 2024).

Cancer toxicity criteria are used in regulatory risk evaluations to estimate the incremental risk of developing cancer as a result of a specific chemical exposure, beyond the background cancer risk. This risk is termed the excess lifetime cancer risk (ELCR), which is expressed as a unitless probability (*e.g.*, 1 cancer case in 1 million people exposed, or 1×10^{-6}). US EPA has established a target ELCR range of 1×10^{-6} (1 cancer case in 1,000,000 people exposed) to 1×10^{-4} (1 cancer case in 10,000 people exposed); an exposure that may result in an ELCR that falls within this range, that is calculated using conservative assumptions, is considered acceptable (US EPA, 1990, 1991). To provide perspective on what a target ELCR of 1 in 10,000 or 1 in 1,000,000 means, it is helpful to understand how these risks compare to the overall lifetime probability of being diagnosed with cancer. A risk of 1 in 1,000,000 is equivalent to a cancer risk of 0.000001, or an ELCR of 0.0001%. A risk of 1 in 10,000 is equivalent to a cancer risk of 0.0001, or an ELCR of 0.01%. Adding these risks to the background risk of developing any cancer over a lifetime (~40%, or about 400,000 cancer cases in a population of 1,000,000) results in total cancer risks of 40.0001-40.01%. Another way to think about these risks is as follows.

- A 40% background cancer risk is the same as 400,000 cancer cases occurring in a population of 1,000,000. Compare that to:
  - 400,001 cancer cases in a population of 1,000,000 (the same as a risk of 40.0001%, or a 1×10^{-6} ELCR), and
  - 400,100 cancer cases in a population of 1,000,000 (the same as a risk of 40.01%, or a 1×10^{-4} ELCR).

Therefore, US EPA's target ELCRs are well below the overall lifetime risk of getting cancer (including bladder cancer) and represent only very slight increases above the background risk of cancer, based on conservative assumptions of exposure and toxicity.

3.3.3.2 Cancer Risk Calculations

Per US EPA (1989) guidance, the excess lifetime cancer risk (ELCR) for oral or dermal exposure to a chemical is calculated by multiplying the lifetime average daily oral or dermal dose (LADD) of that chemical by the chemical-specific CSF, as follows:

$$\text{ELCR from Oral or Dermal Exposure} = \text{LADD (mg/kg-day)} \times \text{CSF}\left([\text{mg/kg-day}]^{-1}\right)$$

Similarly, per US EPA (1989), the excess lifetime cancer risk (ELCR) from inhalation exposure to a chemical is calculated by multiplying the lifetime average daily inhalation exposure concentration (LADE) of that chemical by the chemical-specific IUR, as follows:

$$\text{ELCR from Inhalation Exposure} = \text{LADE}\ (\mu g/m^3) \times \text{IUR}\ \left([\mu g/m^3]^{-1}\right)$$

US EPA does not derive toxicity criteria based specifically on dermal exposure toxicity studies. Instead, risk from dermal exposure to chemicals is assessed based on oral toxicity criteria, under the assumption that once a chemical is absorbed into the blood stream, the health effects caused by that chemical are similar regardless of whether the route of exposure was oral or dermal. Because oral toxicity criteria are based on the amount of a chemical *administered* per unit of time and body weight (*i.e.*, the chemical intake), and not the amount absorbed systemically from the gastrointestinal tract, and because dermal exposures are expressed as absorbed intake levels, the oral criteria need to be adjusted to be applicable to *absorbed* doses before they can be used to assess risk from dermal exposure (US EPA, 1989, 1992, 2004).

This adjustment is made using the chemical's oral absorption efficiency (*i.e.*, the systemic absorption of the chemical following oral exposure). If a chemical's systemic absorption following oral exposure is very high (almost 100%), then the absorbed dose is virtually the same as the administered dose, and no adjustment of the oral toxicity factor is necessary for dermal risk calculations. If a chemical's systemic absorption following oral exposure is very low (*e.g.*, 5%), the chemical's oral toxicity criterion must be adjusted to account for the fact that the absorbed dose is much smaller than the administered dose before the criterion can be used to assess risk from dermal exposure to that chemical. US EPA recommends adjusting a chemical's oral toxicity criterion for use in evaluating dermal exposure and risks only when the systemic absorption of that chemical following oral exposure is less than 50%, to "obviate the need to make comparatively small adjustments in the toxicity value that would otherwise impart on the process a level of accuracy that is not supported by the scientific literature" (US EPA, 2004). Because the oral absorption efficiencies of TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE are not less than 50%, their oral toxicity criteria can be used to assess risks posed by dermal exposure to these chemicals without any adjustment (US EPA, 2004).

For some chemicals that US EPA considers to be carcinogenic *via* a mutagenic mode of action (chemicals considered to react with DNA and lead to permanent changes in DNA, *i.e.*, mutations), such as TCE (for kidney cancer), US EPA (2011a) recommends applying age-dependent adjustment factors (ADAFs) to the cancer toxicity criteria to derive values protective of children in various age ranges. The current ADAFs are 10 for <2-year-olds (*i.e.*, an increase in the ELCR estimate by 10-fold is recommended for this age group), 3 for 2- to <16-year-olds (*i.e.*, an increase in the ELCR estimate by 3-fold is recommended for this age group), and 1 for ≥16-year-olds (no increase to the ELCR estimate is recommended for this age group) (US EPA, 2011a). US EPA recommends multiplying the CSF and IUR by the 10- and 3-fold ADAFs as part of the cancer risk calculations. US EPA does not recommend such adjustments when deriving cancer toxicity criteria for PCE or benzene because the agency does not consider PCE or benzene to cause cancer by a mutagenic mode of action. As discussed in Section 5, US EPA has derived vinyl chloride cancer toxicity values for continuous lifetime exposure from birth that should be applied for early-life (from-birth) scenarios.

After calculating cancer risks from exposure to chemicals *via* each relevant exposure pathway, the ELCR is derived by summing the risks across chemicals and exposure pathways. If the ELCR falls within US EPA's acceptable risk range of 1×10^{-4} to 1×10^{-6} (or 1 additional cancer case in 10,000 people exposed to 1 additional cancer case in 1,000,000 people exposed), there is no need for further evaluation. If the ELCR is calculated to be greater than 1 in 10,000, the *potential* cancer risk from the evaluated chemical exposures requires further evaluation. However, because of the overly conservative nature of regulatory toxicity criteria, as discussed above, the exceedance of an estimated ELCR of 1×10^{-4} does not mean that adverse health effects will occur or are even likely to occur in any one individual.

3.4 The "Linear No-Threshold" Model and the Concept of "No Safe Dose" for Carcinogens

Carcinogenic compounds are often incorrectly described as having "no safe dose." The "no safe dose" concept can be described as meaning that any level of exposure to a carcinogen will lead to some level of increased cancer risk. This concept comes from the linear no-threshold (LNT) or "nonthreshold" mechanism of carcinogenesis that is often conservatively assumed to apply in regulatory cancer risk evaluations. As described by US EPA (and other regulatory agencies), the LNT mechanism of action is applied when there is no known "threshold" dose below which exposure to a carcinogen is not expected to lead to some level of risk, even if it is very low. CSFs and IURs that are derived by extrapolating from the lowest doses in an animal or human study (or POD) down to a response of zero (as discussed above), are derived by applying the LNT approach. In contrast, a threshold model for deriving toxicity criteria is based on the concept that there is some dose below which no adverse effects are expected.

Figure 3.3 depicts a threshold (often the BMDL derived from the animal or human study) and a linear no-threshold (LNT) dose-response model that could be applied to a POD. Figure 3.4 provides a comparison of the LNT and threshold model extrapolations from the PODs. As shown, for a threshold model, US EPA typically applies uncertainty factors (*e.g.*, for sensitive subpopulations, or for the use of an animal study) to the POD to derive a toxicity value, at or below which adverse health effects are not expected. Non-cancer toxicity values are typically derived using a threshold approach.

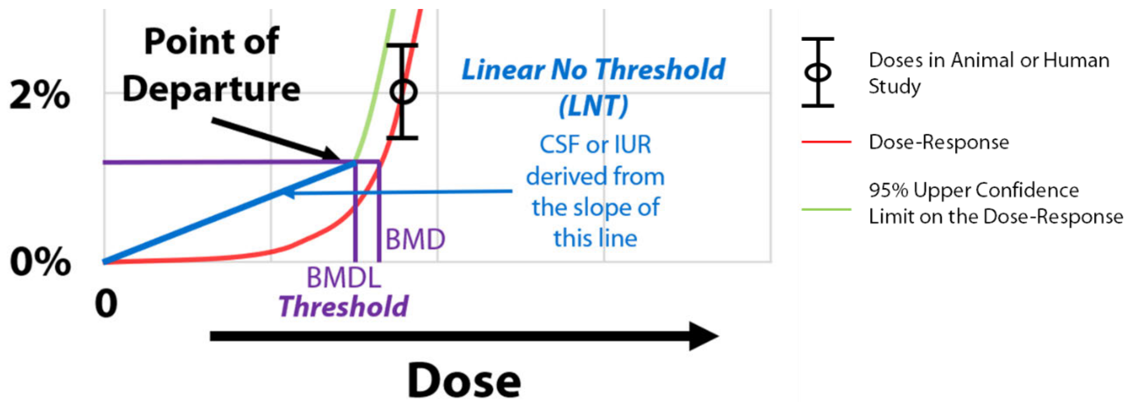

Figure 3.3 Linear No-Threshold (LNT) *vs*. Threshold Models. CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk.

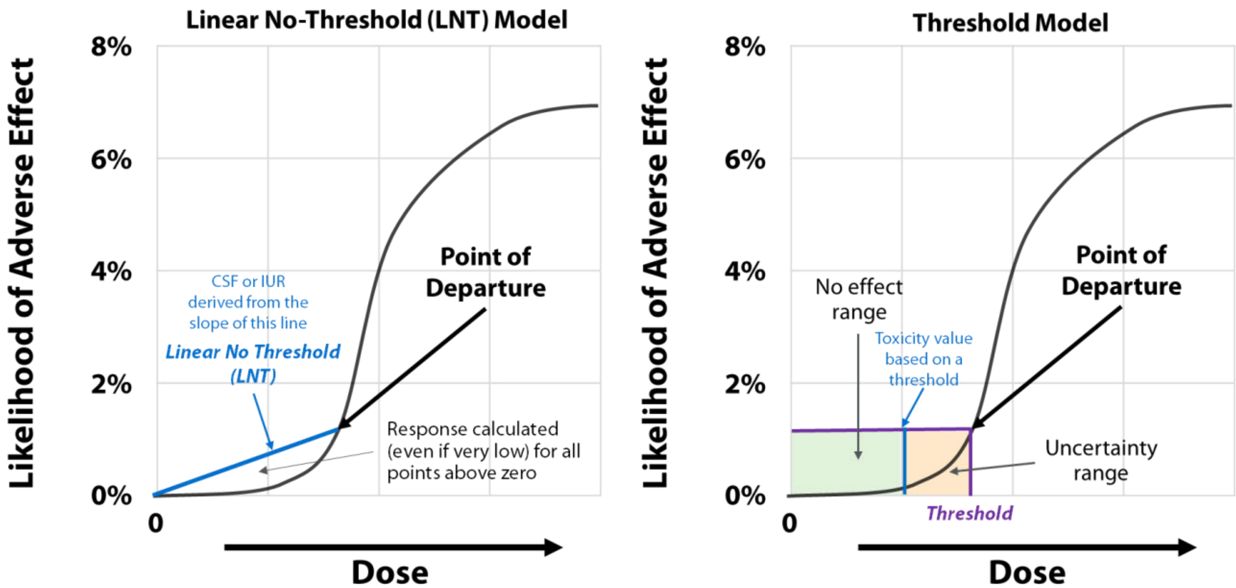

Figure 3.4 Linear No-Threshold (LNT) *vs.* Threshold Model Extrapolations from the Point of Departure (POD)

Regulatory agencies often consider a nonthreshold approach to be the mechanism of carcinogenesis for genotoxic carcinogens (*i.e.*, carcinogens that directly interact with DNA), even when there is no reliable evidence of genotoxicity for these chemicals at low doses. Therefore, for chemicals that have been observed to be genotoxic only at high doses, a nonthreshold approach may be very conservative. Some carcinogens have been found to cause cancer through mechanisms that are not directly genotoxic. For example, some chemicals can cause cytotoxicity (cell death) at certain doses (usually high doses); cytotoxic conditions can result in oxidative stress that can result in the generation of toxic substances (*i.e.*, oxygen radicals) that can react with DNA and cause mutations and cancer. These carcinogens are considered to have a "threshold."

A nonthreshold mechanism of carcinogenesis implies that any level of exposure, even as low as a single molecule of a substance in a cell, potentially presents some level of response because of the possibility that (in theory) one molecule of the substance could react with DNA in a critical gene, and the consequent DNA damage could result in a mutation in that gene (permanent change in DNA) that could then result in carcinogenesis. However, this theoretical carcinogenic mechanism is not biologically plausible, even for carcinogens that are known to react directly with DNA. Several scientific reviews on this topic (*e.g.*, Cardarelli and Ulsh, 2018; Golden *et al.*, 2019; Calabrese, 2023) describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk. Although a nonthreshold approach is reasonable to consider on a theoretical basis, the probability that it will occur in humans (*i.e.*, is it biologically plausible?) needs to be considered in the context of the high levels of DNA damage that human cells experience and efficiently repair on a daily basis.

DNA damage occurs daily in every cell in the human body as a result of normal daily living, and the body readily repairs that damage on a regular basis (Ames *et al.*, 1995). DNA damage from endogenous processes (*i.e.*, processes that naturally occur in the human body) are thought to result in a steady-state (*i.e.*, continuous, or any point in time) background level of about 50,000 damaged DNA bases in every human cell (Swenberg *et al.*, 2011). Although carcinogens have the potential to damage DNA either directly or indirectly, possibly resulting in mutations that may contribute to the development of cancer, these processes are more likely to happen when the body's normal cellular and molecular defense and repair mechanisms are damaged or overwhelmed by high concentrations of a mutagen or carcinogen. The body's normal defense mechanisms can efficiently eliminate low concentrations of a mutagenic or carcinogenic substance and repair DNA damage that exposure to the substance may have caused. Therefore, exposures to low levels of genotoxic carcinogens would be unlikely to lead to increases in genotoxicity, mutations, and carcinogenesis beyond what would be considered background levels. Therefore, the current scientific evidence supports a threshold mode of action even for substances that interact directly with DNA, as long as the exposure levels are low enough to not significantly overwhelm cells' normal defense mechanisms.

The National Research Council (NRC) report "Science and Decisions: Advancing Risk Assessment" described the need for an improved framework for dose-response analysis (NRC, 2009). The authors of this report discuss the nonthreshold (LNT) approach and address some of the points I have raised here. The authors state the following regarding the nonthreshold dose-response approach (referred to by NRC as the "low-dose linear" approach):

> *Low-dose linear individual and population dose-response*. For this conceptual model, both individual risk and population risk have no threshold and are linear at low doses.... Note that low-dose linear means that at low doses "added risk" (above background) increases linearly with increasing dose; **it does not mean that the dose-response relationship is linear throughout the dose range between zero dose and high doses.** (NRC, 2009 [emphasis added]).

The NRC goes on to illustrate that the linear dose-response approach is based on an assumption of linearity above background from the hypothetical average of a number of different nonlinear (or threshold) dose-response curves in the population, showing that for a given individual, the dose-response is not linear throughout the dose range between zero and high doses. That is, for every individual, there is an exposure level, even for genotoxic substances, below which DNA damage and mutagenesis would not be expected to occur because of cellular defense mechanisms that are able to fully function at low exposures to exogenous (*i.e.*, environmental) substances. However, in the absence of information about the shape of the population dose-response curve in the low dose background range, regulators often conservatively assume the relationship is linear below the background level.

Therefore, the concept that there is "no safe dose" for carcinogens, or that there is no threshold below which increased cancer risk is unlikely, is not biologically plausible. Although US EPA and other regulatory agencies often apply the nonthreshold model (the basis of the "no safe dose" concept) to derive cancer toxicity criteria, the scientific evidence supports the conclusion that this approach is overly conservative when evaluating low exposures to genotoxic and mutagenic carcinogens in the population, and likely even more conservative when evaluating these exposures on an individual basis.

Further, based on the conservative derivations of cancer toxicity values using a nonthreshold approach, cancer risks calculated using these toxicity values are overly conservative, particularly at low doses; *i.e.*, for low exposures that typically occur in the population, a threshold approach is likely to be more scientifically appropriate.

3.5 Regulatory Toxicology and Risk Assessment *vs*. Risk Evaluation to Assess Potential Causation

There are substantial differences between how toxicological data are used in a regulatory framework to protect public health *vs.* how they are used to evaluate the potential for causation between an individual's chemical exposures and health effects (Aleksunes and Eaton, 2019). The approach to regulatory decision-making is, in part, directed by policy. As practitioners of public health, regulatory toxicologists are more concerned with avoiding adverse health effects than with estimating the likelihood of health effects actually occurring in a population or an individual (Rodricks and Rieth, 1998; ATSDR, 2018a,b). This difference in perspective is important, because, as discussed above, regulators often use high-end estimates of exposure and toxicity (which can result in overprediction of potential health risks) to be protective of human health. The aim of US EPA and other public health agencies is not to precisely define which effects are expected to occur at any given exposure level, but to define the level at which health effects are *unlikely* to occur (US EPA, 1993; ATSDR, 2018a,b). Thus, regulatory criteria are designed to "protect the health of everyone in general and no one in particular" (Rodricks and Rieth, 1998, p. 23). As such, guidelines developed by US EPA and other agencies for deriving regulatory toxicity criteria state that such criteria are designed to be applicable to "susceptible groups," or sensitive subpopulations, which include life stages (*e.g.*, developing fetus) and other factors that may predispose certain individuals to experience a greater response to a given exposure (US EPA, 2002; ATSDR, 2018a,b). Thus, a regulatory risk assessment is designed to be protective of the population overall, and should not be the sole method used to evaluate risks on an individual basis. However, because of the conservative nature of regulatory toxicity criteria, if individual exposures are at or below those criteria, it can be concluded that the individual exposures do not pose concern for potential adverse health effects.

In contrast to risk assessments performed for regulatory or guidance purposes, assessing the likelihood of a chemical exposure causing health effects for an individual requires a risk evaluation specifically for that individual, based on an individual exposure assessment, dose characterization, and an understanding of the potential health effects that the chemical of interest may have on humans at the exposure levels relevant to the individual (Olsen *et al*., 2014). This type of evaluation can include a risk calculation, using regulatory toxicity criteria, based on the individual's exposure information, as a screening-level conservative first step in a causation analysis. However, as discussed above, it is important to consider the conservative nature of these regulatory criteria, and the fact that they often reflect exposure levels that are much lower than the exposure levels in the animal or human studies at which effects were reported. Therefore, application of regulatory risk calculations for an individual causation analysis is overly conservative and should not be used by itself in a causation analysis. However, if the conservative regulatory risk estimates fall at or below US EPA's acceptable risk range, those results provide strong support for the conclusion that the exposures of concern are not likely to be causally associated with the health effect of concern.

Further, given the conservative nature of the regulatory risk calculations, even if there is an exceedance of US EPA's risk target, that does not mean that health effects are likely to occur. Therefore, for a causation analysis, it is also useful to evaluate potential causal relationships by comparing the estimated doses for the individual to doses or exposure information from the health effect studies (animal or human) that are the basis of the toxicity criteria. These relationships are called margins of exposure (MoEs), as discussed in the next section

In some cases, it is also helpful to compare plaintiff-specific exposure information to exposure information from reliable epidemiology studies that evaluated the potential relationships between exposures to the chemicals of concern and the disease of concern.

3.6 Margin of Exposure Estimates

As discussed above, the exposure levels at which health effects are predicted to be associated with no (or very low) responses in animal or human studies are the starting points (*i.e.*, PODs) used to derive regulatory toxicity criteria. PODs are the doses from which linear extrapolation is conducted to lower doses for the derivation of cancer toxicity criteria. I describe the PODs for TCE, PCE, benzene, and vinyl chloride in Section 5 of this report. In Section 7, I compare the plaintiff's exposure estimates for these chemicals to the appropriate POD. These types of comparisons provide what is called margins of exposure (MoE) between the exposure predicted for an individual and the lowest exposure levels at which health effects have been observed (or exposure levels at which no effects have been observed, for some chemicals) in human or animal studies. In comparison to the conservative regulatory risk calculations that are designed to assess risk for the most sensitive individual in a population, and for any concentration above zero (for carcinogens), MoEs provide a comparison of individual exposure estimates to concentrations much closer to those at which health effects have been reported in human studies (or extrapolated to humans from animal studies). The equation used to calculate MoEs is as follows:

$$\text{MoE} = \frac{\text{POD for the Cancer Toxicity Value}}{\text{Individual LADD or LADE}}$$

If the MoE is greater than 1, that indicates that the POD (*i.e.*, estimated to reflect exposures related to no or very low responses) is higher than exposures estimated for the individual, providing support that adverse health effects would not be expected for the individual.

These MoE calculations, in addition to comparisons of individual exposure information to exposure information from other relevant epidemiology studies, are important for causation analyses because they provide a more useful comparison of the plaintiff's exposures to exposures where health effects have been observed in people. If the plaintiff's exposures are well below exposures where effects have been observed in epidemiology or toxicology studies, even if there is a risk calculation greater than US EPA's targets, these results provide support that the individual exposures are not likely to be associated with the health effect of concern.

4 Brief History of the US Marine Corps Base Camp Lejeune Site

4.1 Site Description and History

In the early 1940s, the United States Marine Corps developed a water-distribution system at its Camp Lejeune base, which is located in Onslow County, North Carolina, approximately 70 miles northeast of Wilmington, North Carolina (ATSDR, 2013a). The sole source of drinking water at Camp Lejeune is groundwater wells that pump water from the Castle Hayne aquifer system (ATSDR, 2013a).

Operations at Camp Lejeune started in late 1941. Multiple water treatment plants (WTPs)[3] have serviced Camp Lejeune, including Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB) (the three at issue in this litigation). The HP WTP was the first plant to come online, in 1942 and serviced the base until the TT and HB WTPs came online in 1952 and in the summer of 1972, respectively (Hennet, 2024). Because the WTPs were connected to many more groundwater wells than were needed to supply drinking water to the base, the wells' service was rotated and water from different wells was sometimes mixed at the WTPs before being delivered to Camp Lejeune residences and facilities as tap water (ATSDR, 2013a).

4.2 Investigations of Groundwater Contamination

In 1974, the Safe Drinking Water Act (SDWA) was established to protect the quality of drinking water in the United States (US Congress, 1974). Under the SDWA, US EPA developed national drinking water regulations that included the derivation of maximum contaminant levels (MCLs), *i.e.*, the highest level of a contaminant that is allowed in drinking water.

In the early 1980s, the groundwater sources for two of the WTPs that serviced Camp Lejeune (HP and TT) were found to be contaminated with volatile organic compounds. Although the groundwater source for the HB WTP was not contaminated, the HB WTP was contaminated when HB drinking water was supplied by the HP WTP in the spring and summer months from 1972 through 1985 (ATSDR, 2017a). The contaminants identified in the drinking water at the HP WTP were TCE, PCE, vinyl chloride, and refined petroleum products (including benzene) (ATSDR, 2017a). The HP contamination is believed to have been related to historical base operations and disposal practices (ATSDR, 2017a). TCE was the primary contaminant identified at the HP WTP. Groundwater modeling conducted by ATSDR estimated that the maximum mean monthly reconstructed level of TCE was 783 parts per billion (ppb), in November 1983 (ATSDR, 2017a). The maximum reconstructed mean monthly concentrations of benzene and PCE were 12 ppb (in April 1984) and 39 ppb (in November 1983), respectively (ATSDR, 2017a). The maximum reconstructed mean monthly concentration of vinyl chloride was 67 ppb, in November 1983 (Maslia *et al.*,

[3] Hadnot Point (HP), Tarawa Terrace (TT), and Holcomb Boulevard (HB) supplied drinking water to residences and workplaces at Camp Lejeune (see Hennet [2024]). Additional Camp Lejeune water-distribution systems which were not contaminated include: Marine Corps Air Station New River, Onslow Beach, Courthouse Bay, Camp Geiger, Rifle Range, and Montford Point/Camp Johnson (Hennet, 2024).

2016; ATSDR, 2017a). The maximum reconstructed mean monthly concentration of 1,2-tDCE was 435 ppb, in November 1983 (ATSDR, 2017a).[4]

Contamination of the TT WTP supply wells was found to be due to an off-site dry cleaner (Bove *et al.*, 2014), with PCE identified as the primary contaminant. TCE, vinyl chloride, and 1,2-tDCE were also detected at this WTP as PCE degradation products (ATSDR, 2017a; Bove *et al.*, 2014).[5] Groundwater modeling conducted by ATSDR, including a multispecies degradation model of PCE, estimated that the maximum reconstructed mean monthly concentration of PCE in the TT WTP was 158 ppb, in June 1984 (ATSDR, 2017a). Applying the same model, ATSDR estimated maximum reconstructed mean monthly concentrations of TCE and vinyl chloride of 7 and 12 ppb, respectively (ATSDR, 2017a). The maximum reconstructed mean monthly concentration of 1,2-tDCE was 22 ppb (ATSDR, 2017a).[6]

The wells directly serving the other Camp Lejeune water-distribution systems – Holcomb Boulevard (HB), Marine Corps Air Station, Courthouse Bay, Camp Johnson, Camp Geiger, and Rifle Range – were not contaminated with solvents. As stated previously, the HB WTP was largely uncontaminated except when HB drinking water was supplied by the HP WTP (ATSDR, 2017a).

By February 1985, the most highly contaminated wells servicing the HP and TT WTPs had been removed from service (ATSDR, 2017b).

[4] Drs. Hennet and Spiliotopoulos explain in their expert reports that ATSDR's modeled groundwater concentrations are unreliable and likely biased high as a result of several conservative assumptions used in ATSDR's modeling (Hennet, 2024; Spiliotopoulos, 2024).

[5] Refined petroleum products were not contaminants of the TT WTP; therefore, benzene was not identified as a contaminant of concern at the TT WTP, and ATSDR did not model groundwater concentrations for benzene for the TT WTP (ATSDR, 2013b; Hennet, 2024).

[6] Drs. Hennet and Spiliotopoulos explain in their expert reports that ATSDR's modeled groundwater concentrations are unreliable and likely biased high as a result of several conservative assumptions used in ATSDR's modeling (Hennet, 2024; Spiliotopoulos, 2024).

5 Hazard Assessments and Toxicity Criteria

This section summarizes the TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE hazard assessments that have been conducted by regulatory agencies, and the hazard evaluations conducted by Dr. Goodman (2025) that are specifically focused on exposure to each of these chemicals and bladder cancer. In addition, I summarize the US EPA cancer toxicity criteria for TCE, PCE, benzene, and vinyl chloride that are applied in the plaintiff-specific risk evaluation (Section 6). Note that US EPA has not derived oral or inhalation toxicity criteria for 1,2-tDCE, because US EPA concluded that there was inadequate evidence with which to assess the carcinogenic potential of 1,2-tDCE (US EPA, 2010a,b).

5.1 Hazard Assessments

5.1.1 Trichloroethylene (TCE)

To understand the potential association between TCE exposure and bladder cancer, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the conclusions from several regulatory agency TCE toxicological reports. Overall, US EPA (2011a, 2020a) concluded that there is some scientific evidence that TCE can cause bladder cancer in humans from high cumulative exposures, but also note that the available studies do not completely account for potential confounding factors, therefore, limiting the conclusions that can be drawn from these studies. ATSDR (2019a), NTP (2015), and IARC (2014) did not conclude that exposure to TCE is associated with increased bladder cancer risk in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that there was "below equipoise evidence for causation" for TCE and bladder cancer.

Based on the available epidemiology studies and agency reviews that evaluated TCE exposure and bladder cancer, Dr. Goodman concluded that the majority of "cohort and case-control studies did not report associations between TCE exposure overall and bladder cancer or any dose-response relationships" (Goodman, 2025). Dr. Goodman also concluded the animal bioassays "do not provide evidence that TCE can cause bladder cancer" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between TCE exposure and bladder cancer concluded that overall, "scientific evidence does not support a causal association between TCE and bladder cancer" (Goodman, 2025).

5.1.2 Tetrachloroethylene (PCE)

To understand the potential association between PCE exposure and bladder cancer, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the conclusions from several regulatory agency PCE toxicological reports (ATSDR, 2019b; US EPA, 2012b, 2020b); these agencies concluded that there is weak scientific evidence for an association between PCE and bladder cancer. IARC (2014) concluded that there is limited evidence in humans for carcinogenicity of PCE, stating that "positive associations have been observed for cancer of the bladder." In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that there is "sufficient evidence for causation" for exposure to PCE and bladder cancer based on limited epidemiology evidence.

Based on the available epidemiology studies and agency reviews that evaluated PCE exposure and bladder cancer, Dr. Goodman concluded that the majority of "cohort and case-control studies did not report associations between PCE exposure and bladder cancer or demonstrate exposure-response relationships" (Goodman, 2025). Dr. Goodman also concluded that chronic oral and inhalation animal studies, "do not provide evidence that PCE can cause bladder cancer" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between PCE exposure and bladder cancer concluded that overall, "scientific evidence does not support a causal association between PCE and bladder cancer" (Goodman, 2025).

5.1.3 Benzene

To understand the potential association between benzene exposure and bladder cancer, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the conclusions from several regulatory agency benzene toxicological reports. Overall, US EPA (2003a), ATSDR (2007a, 2015), and IARC (2018) did not conclude that there is a causal association between exposure to benzene and bladder cancer in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that the evidence for an association between benzene exposure and bladder cancer was "below equipoise evidence for causation."

Based on the available epidemiology studies and agency reviews that evaluated benzene exposure and bladder cancer, Dr. Goodman concluded that the majority of "cohort and case-control studies did not report associations between benzene exposure and bladder cancer or demonstrate exposure-response relationships" (Goodman, 2025). Dr. Goodman also concluded the animal studies "do not provide evidence that benzene can cause bladder cancer" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between benzene exposure and bladder cancer concluded that "the evidence does not support a causal association between benzene and bladder cancer" (Goodman, 2025).

5.1.4 Vinyl Chloride

To understand the potential association between vinyl chloride exposure and bladder cancer, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the conclusions from regulatory agency vinyl chloride toxicological reports. Overall, ATSDR (2024b) and US EPA (2003b) did not conclude that vinyl chloride exposure is a known cause of bladder cancer in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that the evidence for an association between vinyl chloride exposure and bladder cancer was "below equipoise evidence for causation."

Based on the available epidemiology studies and agency reviews that evaluated vinyl chloride exposure and bladder cancer, Dr. Goodman concluded that "[n]o study reported associations between vinyl chloride exposure and bladder cancer or demonstrated clear exposure-response relationships" (Goodman, 2025). Dr. Goodman also concluded that the "animal evidence does not support vinyl chloride as a cause of bladder cancer in humans" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between vinyl chloride exposure and bladder cancer concluded that overall, "the evidence does not support a causal association between vinyl chloride and bladder cancer" (Goodman, 2025).

5.1.5 *trans*-1,2-Dichloroethylene (1,2-tDCE)

To understand the potential association between 1,2-tDCE exposure and bladder cancer, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the conclusions from regulatory agency 1,2-tDCE toxicological reports. Dr. Goodman concluded that currently available scientific evidence is too limited to address whether there is a causal association between 1,2-tDCE and bladder cancer (Goodman, 2025). Overall, US EPA (2010a,b) and ATSDR (2023) have not concluded that 1,2-tDCE is associated with increased bladder cancer risk in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) did not comment on 1,2-tDCE potential bladder carcinogenicity.

5.2 Toxicity Criteria

This section summarizes the cancer toxicity criteria that US EPA derived for TCE, PCE, benzene, and vinyl chloride based on the methodology described in Section 3, and US EPA's hazard assessment of these chemicals as described in the documents cited below.

5.2.1 Trichloroethylene (TCE)

Table 5.1 summarizes the cancer types, points of departure (PODs), and oral cancer toxicity criteria (cancer slope factors [CSFs]) that US EPA derived for TCE (US EPA, 2011b). Table 5.2 summarizes the cancer types, PODs, and inhalation cancer toxicity criteria (inhalation unit risks [IURs]) that US EPA derived for TCE (US EPA, 2011b). Note that US EPA does not provide TCE oral PODs for renal cell carcinoma (kidney cancer), non-Hodgkin's lymphoma (NHL), or liver cancer, or TCE inhalation PODs for NHL or liver cancer. Because PODs are used in the MoE analyses in Section 7, I estimated PODs for these pathways and endpoints as described in Appendix E.

Based on its hazard assessment for TCE, US EPA first derived the TCE IURs for renal cell carcinoma, NHL, and liver cancer based on two human occupational TCE inhalation studies (Charbotel *et al*., 2006; Raaschou-Nielsen *et al*., 2003; US EPA, 2011b) (Table 5.2). US EPA then applied a TCE physiologically based pharmacokinetic (PBPK) model to conduct a route-to-route (inhalation-to-oral) extrapolation to derive the TCE CSFs from the IURs (US EPA, 2011b) (Table 5.1).

Table 5.3 summarizes the TCE toxicity criteria used in the risk evaluation for the plaintiff. Because the evidence overall does not support an association between TCE exposure and bladder cancer, increased bladder cancer risk is not expected from TCE exposure, and TCE cancer toxicity values specific to bladder cancer are not available. Therefore, I conservatively apply the CSF and IUR that US EPA derived for kidney cancer, liver cancer, and NHL combined to estimate cancer risk for the plaintiff. It should be noted that applying US EPA's TCE cancer toxicity criteria that are based on these three cancers combined is not predictive, and is overly conservative, for estimating bladder cancer risk.

Table 5.1 US EPA TCE Oral Cancer Toxicity Values (Cancer Slope Factors [CSFs])

Chemical	Oral CSF[a,b] ([mg/kg-day]$^{-1}$)	POD (mg/kg-day)	Cancer Type	Sources
TCE	4.6×10^{-2}	$LED_{01} = 0.21$[c]	Renal cell carcinoma, NHL, and liver cancer	US EPA (2011a,b)
	9.33×10^{-3}	$LED_{01} = 1.07$[d]	Renal cell carcinoma	
	2.16×10^{-2}	$LED_{01} = 0.46$[d]	NHL	
	1.55×10^{-2}	$LED_{01} = 0.65$[d]	Liver cancer	

Notes:
IUR = Inhalation Unit Risk; LED_{01} = Lower Confidence Limit of the Exposure Dose at an Extra Risk Level of 1%; mg/kg-day = Milligrams per Kilogram Body Weight per Day; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; NHL = Non-Hodgkin's Lymphoma; PBPK = Physiologically Based Pharmacokinetic; POD = Point of Departure; ppm = Parts per Million; (ppm)$^{-1}$ = Per Parts per Million; TCE = Trichloroethylene; US EPA = United States Environmental Protection Agency.
(a) Individual CSFs for the three cancers were derived by US EPA (2011a) by extrapolating from the IURs for these cancers. Each IUR was multiplied by a cancer-specific PBPK model-derived adjustment for route-to-route extrapolation (from inhalation to oral exposure) (US EPA, 2011a), as follows. Renal Cell Carcinoma: 5.49×10^{-3} (ppm)$^{-1}$ × 1.7 ppm per mg/kg-day = 9.33×10^{-3} (mg/kg-day)$^{-1}$. NHL: 1.10×10^{-2} (ppm)$^{-1}$ × 1.97 ppm per mg/kg-day = 2.16×10^{-2} (mg/kg-day)$^{-1}$. Liver Cancer: 5.49×10^{-3} (ppm)$^{-1}$ × 2.82 ppm per mg/kg-day = 1.55×10^{-2} (mg/kg-day)$^{-1}$.
(b) The CSF for the three cancer types combined was derived by US EPA (2011a) as follows: (5.49×10^{-3} [ppm]$^{-1}$ × 1.7 ppm per mg/kg-day) × 5 = 4.6×10^{-2} (mg/kg-day)$^{-1}$. The factor of 5 is equal to the total risks summed across all three endpoints (4.6×10^{-2} [mg/kg-day]$^{-1}$) divided the by the kidney cancer risk (9.33×10^{-3} [mg/kg-day]$^{-1}$).
(c) US EPA (2011a) calculated the LED_{01} in mg/kg-day for the three cancers combined using the following equation: LED_{01} = (kidney cancer LED_{01} in ppm ÷ 1.70 ppm per mg/kg-day) ÷ 5 = (1.82 ÷ 1.70) ÷ 5 = 0.21 mg/kg-day.
(d) See Appendix E for derivation.

Table 5.2 US EPA TCE Inhalation Cancer Toxicity Values (Inhalation Unit Risks [IURs])

Chemical	IUR ([μg/m^3]$^{-1}$; [ppm]$^{-1}$)	POD (μg/m^3 [ppb])	Cancer Type	Sources
TCE	4.1×10^{-6} (μg/m^3)$^{-1}$; 2.2×10^{-2} (ppm)$^{-1}$	LEC_{01} = 2,445[a] (455)	Renal cell carcinoma, NHL, and liver cancer	Charbotel *et al.* (2006); Raaschou-Nielsen *et al.* (2003); US EPA (2011a,b)
	1.0×10^{-6} (μg/m^3)$^{-1}$; 5.5×10^{-3} (ppm)$^{-1}$	LEC_{01} = 9,781 (1,820)	Renal cell carcinoma	
	2.0×10^{-6} (μg/m^3)$^{-1}$ 1.1×10^{-2}(ppm)$^{-1}$	LEC_{01} = 4,890[b] (910)	NHL	
	1.0×10^{-6} (μg/m^3)$^{-1}$; 5.5×10^{-3} (ppm)$^{-1}$	LEC_{01} = 9,781[b] (1,820)	Liver cancer	

Notes:
μg/m^3 = Micrograms per Cubic Meter; (μg/m^3)$^{-1}$ = Per Micrograms per Cubic Meter; LEC_{01} = Lower Confidence Limit of the Exposure Concentration at an Extra Risk Level of 1%; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; (ppm)$^{-1}$ = Per Parts per Million; NHL = Non-Hodgkin's Lymphoma; TCE = Trichloroethylene; US EPA = United States Environmental Protection Agency.
(a) US EPA (2011a) calculated the LEC_{01} for all three cancers combine using the following equation and the LEC_{01} for kidney cancer of 1.82 ppm: LEC_{01} = kidney cancer LEC_{01} ÷ 4 = 1.82 ppm ÷ 4 = 0.455 ppm (equivalent to 2,445 μg/m^3). The factor of 4 is equal to the total risks summed across all three endpoints (4.1×10^{-6} [μg/m^3]$^{-1}$) divided by the kidney cancer risk (1.0×10^{-6} [μg/m^3]$^{-1}$).
(b) See Appendix E for derivation.

Table 5.3 TCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Cancer Type	Value
TCE	Oral CSF	Renal, NHL, liver	4.6×10^{-2} (mg/kg-day)$^{-1}$
	IUR	Renal, NHL, liver	4.1×10^{-6} (μg/m^3)$^{-1}$

Notes:
(μg/m^3)$^{-1}$ = Per Microgram per Cubic Meter; CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; TCE = Trichloroethylene.
Source: US EPA (2011b).

5.2.1.1 Age-Dependent Adjustment Factors

As discussed in Section 3, for some chemicals that US EPA considers to be carcinogenic *via* a mutagenic mode of action, such as TCE, US EPA recommends applying age-dependent adjustment factors (ADAFs) to estimate TCE cancer risks protective of children in various age ranges. The current ADAFs are 10 for <2-year-olds and 3 for 2- to <16-year-olds, and no ADAF is necessary for ≥16-year-olds (US EPA, 2011a). Because the modes of action for each of the three cancer endpoints for the TCE toxicity criteria (NHL, kidney cancer, and liver cancer) are not all considered to be mutagenic (*i.e.*, only TCE-induced kidney cancer is considered by US EPA to have a mutagenic mode of action [US EPA, 2011a]), US EPA provides toxicity value adjustment factors so that the ADAFs can be applied to only the kidney cancer portion of the risk calculation.

Oral LADD for TCE

The oral carcinogenicity and mutagenicity adjustment factors are calculated based on the different endpoint-specific oral toxicity values (oral cancer slope factors [CSFs]) for TCE, as follows (US EPA, 2024).

$$\text{CAF}_\text{o}\ (0.804) = \frac{\text{NHL + Liver CSF}_\text{o}\ \left(3.7 \times 10^{-2}\ (\text{mg/kg-day})^{-1}\right)}{\text{Adult-Based CSF}_\text{o}\ (4.6 \times 10^{-2}\ (\text{mg/kg-day})^{-1})}$$

$$\text{MAF}_\text{o}\ (0.202) = \frac{\text{Kidney CSF}_\text{o}\ \left(9.3 \times 10^{-3}\ (\text{mg/kg-day})^{-1}\right)}{\text{Adult-Based CSF}_\text{o}\ (4.6 \times 10^{-2}\ (\text{mg/kg-day})^{-1})}$$

where:

CAF_o = Carcinogenicity Adjustment Factor for the TCE Cancer Oral Slope Factor
MAF_o = Mutagenicity Adjustment Factor for the TCE Cancer Oral Slope Factor

To adjust the lifetime average daily dose (LADD) (equation shown in Section 3) for TCE prior to multiplying by the oral TCE cancer toxicity value shown in Table 5.3, the LADD is apportioned by applying the carcinogenicity and mutagenicity adjustment factors (CAF_o and MAF_o), and then the ADAF of 3 (for ages 2 to <16 years) is applied to only the MAF_o portion of the equation, as follows (US EPA, 2024):

$$\text{LADD}_{\text{TCE adj}} = (\text{LADD} \times 3 \times \text{MAF}_\text{o}) + (\text{LADD} \times \text{CAF}_\text{o})$$

Inhalation LADE for TCE

Similarly, the inhalation carcinogenicity and mutagenicity adjustment factors are calculated based on the different endpoint-specific inhalation toxicity values (inhalation unit risks [IURs]) for TCE, as follows (US EPA, 2024).

$$\text{CAF}_\text{i}\ (0.756) = \frac{\text{NHL + Liver IUR}\ \left(3.0 \times 10^{-6}\ \left(\mu\text{g/m}^3\right)^{-1}\right)}{\text{Adult-Based IUR}\ (4.1 \times 10^{-6}\ (\mu\text{g/m}^3)^{-1})}$$

$$\text{MAF}_\text{i}\ (0.244) = \frac{\text{Kidney IUR}\ \left(1.0 \times 10^{-6}\ \left(\mu\text{g/m}^3\right)^{-1}\right)}{\text{Adult-Based IUR}\ (4.1 \times 10^{-6}\ (\mu\text{g/m}^3)^{-1})}$$

where:

CAF_i = Carcinogenicity Adjustment Factor for the TCE Inhalation Unit Risk Value
MAF_i = Mutagenicity Adjustment Factor for the TCE Inhalation Unit Risk Value

To adjust the lifetime average daily exposure (LADE) (equation shown in Section 3) for TCE prior to multiplying by the IUR for TCE shown in Table 5.3, the LADE is apportioned by applying the carcinogenicity and mutagenicity adjustment factors (CAF_i and MAF_i), and then the ADAF of 3 (for ages 2 to <16 years) is applied to only the MAF_i portion of the equation, as follows (US EPA, 2024):

$$\mathrm{LADE}_{\mathbf{TCE\ adj}} = (\mathrm{LADE} \times 3 \times \mathrm{MAF_i}) + (\mathrm{LADE} \times \mathrm{CAF_i})$$

5.2.2 Tetrachloroethylene (PCE)

Table 5.4 summarizes the cancer type, point of departure (POD), and oral cancer toxicity criterion (CSF) that US EPA derived for PCE (US EPA, 2012b). Table 5.5 summarizes the cancer type, POD, and inhalation cancer toxicity criterion (IUR) that US EPA derived for PCE (US EPA, 2012b).

Based on its hazard assessment for PCE, US EPA (2012b) first derived a PCE IUR based on an inhalation tumor bioassay conducted in mice that reported hepatocellular adenomas and carcinomas (liver cancer) (JISA, 1993) and applying a PCE PBPK model to extrapolate from animal to human doses (Table 5.4). US EPA then applied the same PBPK model to conduct a route-to-route (inhalation-to-oral) and animal-to-human extrapolation to derive the PCE CSF from the IUR (US EPA, 2012b) (Table 5.5).

Table 5.6 summarizes the PCE toxicity criteria used in the cancer risk evaluation for the plaintiff. Because the evidence overall is limited for an association between PCE exposure and bladder cancer, PCE cancer toxicity values specific to bladder cancer are not available. Therefore, using US EPA's PCE cancer toxicity criteria that are based on liver cancer is not predictive of bladder cancer risk. However, I conservatively apply the criteria to estimate cancer risk for the plaintiff.

Table 5.4 US EPA PCE Oral Cancer Toxicity Value (Cancer Slope Factor [CSF])

Chemical	Oral CSF ([mg/kg-day]$^{-1}$)	POD (mg/kg-day)	Cancer Type (Sex/Species)	Sources
PCE	2.1×10^{-3}	50	Hepatocellular adenomas or carcinomas (male mice)	US EPA (2012b,c)

Notes:
mg/kg-day = Milligrams per Kilogram Body Weight per Day; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; PCE = Tetrachloroethylene; POD = Point of Departure; US EPA = United States Environmental Protection Agency.

Table 5.5 US EPA PCE Inhalation Cancer Toxicity Value (Inhalation Unit Risk [IUR])

Chemical	IUR ([µg/m^3]$^{-1}$; [ppm]$^{-1}$)	POD (µg/m^3 [ppb])	Cancer Type (Sex/Species)	Source
PCE	2.6×10^{-7} (µg/m^3)$^{-1}$; 1.8×10^{-3} (ppm)$^{-1}$	390,000 (60,000)	Hepatocellular adenomas or carcinomas (male mice)	JISA (1993); US EPA (2012b,c)

Notes:
µg/m^3 = Micrograms per Cubic Meter; (µg/m^3)$^{-1}$ = Per Micrograms per Cubic Meter; ppb = Parts per Billion; (ppm)$^{-1}$ = Per Parts per Million; PCE = Tetrachloroethylene; POD = Point of Departure; US EPA = United States Environmental Protection Agency.

Table 5.6 PCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Cancer Type (Sex/Species)	Value
PCE	Oral CSF	Hepatocellular adenomas or carcinomas (male mice)	2.1×10^{-3} (mg/kg-day)$^{-1}$
	IUR		2.6×10^{-7} (µg/m^3)$^{-1}$

Notes:
(µg/m^3)$^{-1}$ = Per Micrograms per Cubic Meter; CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; PCE = Tetrachloroethylene.
Source: US EPA (2012b).

5.2.3 Benzene

Table 5.7 summarizes the cancer type, point of departure (POD), and oral cancer toxicity criterion (CSF) that US EPA derived for benzene (US EPA, 2003a). Table 5.8 summarizes the cancer type, POD, and inhalation cancer toxicity criterion (IUR) that US EPA derived for benzene (US EPA, 2003a).

Based on its hazard assessment for benzene, US EPA (2003a) first derived a benzene IUR based on two sets of benzene exposure estimates derived from the Rinsky *et al*. (1981, 1987) Pliofilm rubber worker cohort studies that evaluated leukemia: (1) exposure estimates from Paustenbach *et al*. (1992), and (2) exposure estimates from Crump and Allen (1984) (Table 5.7). US EPA then conducted route-to-route (inhalation-to-oral) extrapolation to derive the benzene CSF from the IUR (US EPA, 2003a) (Table 5.8).

Table 5.9 summarizes the benzene toxicity criteria used in the risk evaluation for the plaintiff. I chose to use the higher end of the range of benzene CSFs and IURs provided by US EPA (2003a) in my risk calculations. Because the evidence overall does not support an association between benzene exposure and bladder cancer, increased bladder cancer risk is not expected from benzene exposure and benzene cancer toxicity values specific to bladder cancer are not available. Therefore, using US EPA's benzene cancer toxicity criteria that are based on leukemia is not predictive of bladder cancer risk. However, I conservatively apply the criteria to estimate cancer risk for the plaintiff.

Table 5.7 US EPA Benzene Oral Cancer Toxicity Values (Cancer Slope Factors [CSFs])

Chemical	Oral CSF ([mg/kg-day]$^{-1}$)	POD (mg/kg-day)	Cancer Type	Sources
Benzene	1.5×10^{-2} to 5.5×10^{-2}	0.055	Leukemia	Rinsky *et al*. (1981, 1987); US EPA (1999, 2003a)

Notes:
mg/kg-day = Milligrams per Kilogram Body Weight per Day; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; POD = Point of Departure; US EPA = United States Environmental Protection Agency.

Table 5.8 US EPA Benzene Inhalation Cancer Toxicity Values (Inhalation Unit Risks [IURs])

Chemical	IUR ([µg/m^3]$^{-1}$; [ppm]$^{-1}$)	POD (µg/m^3 [ppb])	Cancer Type	Sources
Benzene	2.2×10^{-6} to 7.8×10^{-6} (µg/m^3)$^{-1}$; 7.1×10^{-3} to 2.5×10^{-2} (ppm)$^{-1}$	383 (120)	Leukemia	Rinsky *et al*. (1981, 1987); US EPA (1998, 2003a)

Notes:
µg/m^3 = Micrograms per Cubic Meter; (µg/m^3)$^{-1}$ = Per Micrograms per Cubic Meter; POD = Point of Departure; ppb = Parts per Billion; (ppm)$^{-1}$ = Per Parts per Million; US EPA = United States Environmental Protection Agency.

Table 5.9 Benzene Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Cancer Type	Value
Benzene	Oral CSF	Leukemia	5.5×10^{-2} (mg/kg-day)$^{-1}$
	IUR		7.8×10^{-6} (μg/m^3)$^{-1}$

Notes:
(μg/m^3)$^{-1}$ = Per Micrograms per Cubic Meter; CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk; (mg/kg-day)$^{-1}$ = Per Milligrams per Kilogram Body Weight per Day.
Source: US EPA (2003a).

5.2.4 Vinyl Chloride

Table 5.10 summarizes the cancer type, points of departure (PODs), and oral cancer toxicity criteria (CSFs) that US EPA derived for vinyl chloride (US EPA, 2000, 2003b). Table 5.11 summarizes the cancer type and inhalation cancer toxicity criteria (IURs) that US EPA derived for vinyl chloride (US EPA, 2003b). Note that US EPA does not provide vinyl chloride oral or inhalation PODs. Because PODs are used in the MoE analyses in Section 7, I estimated PODs for these pathways as described in Appendix E.

Based on its hazard assessment for vinyl chloride, US EPA (2000, 2003b) derived a vinyl chloride CSF for continuous lifetime exposure during adulthood based on an increased incidence of liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules in female rats in the oral study by Feron *et al*. (1981) and applying a vinyl chloride PBPK model to extrapolate from animal to human doses. US EPA derived two very similar CSFs using two extrapolation methods and they recommend using the lower of the two values for risk calculations (US EPA, 2000, 2003b). US EPA (2000, 2003b) also recommends a two-fold higher CSF to account for continuous lifetime exposure from birth. Values are summarized in Table 5.10.

US EPA (2000, 2003b) derived a vinyl chloride IUR for continuous lifetime exposure during adulthood based on an increased incidence of liver angiosarcomas, angiomas, hepatomas, and neoplastic nodules in female rats in the inhalation studies by Popper *et al*. (1981) and Maltoni *et al*. (1984) and applying a vinyl chloride PBPK model to extrapolate from animal to human doses. US EPA (2000, 2003b) also recommends a two-fold higher CSF to account for continuous lifetime exposure from birth. Values are summarized in Table 5.11

Table 5.12 summarizes the vinyl chloride toxicity criteria used in the risk evaluation for the plaintiff. Because the evidence overall does not support an association between vinyl chloride exposure and bladder cancer, increased bladder cancer risk is not expected from vinyl chloride exposure and vinyl chloride cancer toxicity values specific to bladder cancer are not available. Therefore, use of US EPA's vinyl chloride cancer toxicity criteria that are based on liver cancer is not predictive of bladder cancer risk. However, I conservatively apply the criteria to estimate cancer risk for the plaintiff.

Table 5.10 US EPA Vinyl Chloride Oral Cancer Toxicity Values (Cancer Slope Factors [CSFs])

Chemical	Oral CSF ($[mg/kg\text{-}day]^{-1}$)	POD[a] (mg/kg-day)	Cancer Type (Sex/Species)	Sources
Vinyl Chloride	**Continuous Lifetime Exposure, Not Exposed at Birth**			
	7.2×10^{-1}; 7.5×10^{-1}	$LED_{10} = 0.133$	Liver angiosarcomas, hepatocellular carcinomas, and neoplastic liver nodules (female rat)	Feron *et al.* (1981); US EPA (2000, 2003b)
	Continuous Lifetime Exposure from Birth			
	1.4; 1.5	$LED_{10} = 0.067$	Liver angiosarcomas, hepatocellular carcinomas, and neoplastic liver nodules (female rat)	Feron *et al.* (1981); US EPA (2000, 2003b)

Notes:
LED_{10} = Lower Confidence Limit of the Exposure Dose at an Extra Risk Level of 10%; mg/kg-day = Milligrams per Kilogram Body Weight per Day; $(mg/kg\text{-}day)^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; POD = Point of Departure; US EPA = United States Environmental Protection Agency.
(a) See Appendix E for derivation.

Table 5.11 US EPA Vinyl Chloride Inhalation Cancer Toxicity Values (Inhalation Unit Risks [IURs])

Chemical	IUR ($[\mu g/m^3]^{-1}$)	POD[a] ($\mu g/m^3$ [ppb])	Cancer Type (Sex/Species)	Sources
Vinyl Chloride	**Continuous Lifetime Exposure, Not Exposed at Birth**			
	4.4×10^{-6}	LEC_{10} = 22,727 (8,900)	Liver angiosarcomas, angiomas, hepatomas, and neoplastic liver nodules (female rat)	Popper *et al.* (1981); Maltoni *et al.* (1984); US EPA (2000, 2003b)
	Continuous Lifetime Exposure from Birth			
	8.8×10^{-6}	LEC_{10} = 11,364 (4,445)	Liver angiosarcomas, angiomas, hepatomas, and neoplastic liver nodules (female rat)	Popper *et al.* (1981); Maltoni *et al.* (1984); US EPA (2000, 2003b)

Notes:
$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Micrograms per Cubic Meter; LEC_{10} = Lower Confidence Limit of the Exposure Concentration at an Extra Risk Level of 10%; ppb = Parts per Billion; POD = Point of Departure; US EPA = United States Environmental Protection Agency.
(a) See Appendix E for derivation.

Table 5.12 Vinyl Chloride Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Cancer Type	Value
Vinyl Chloride	Oral CSF	Liver angiosarcomas, angiomas, hepatomas, and neoplastic liver nodules (female rat)	7.2×10^{-1} $(mg/kg\text{-}day)^{-1}$ (continuous lifetime exposure, not exposed at birth)
	IUR		4.4×10^{-6} $(\mu g/m^3)^{-1}$ (continuous lifetime exposure, not exposed at birth)

Notes:
$(\mu g/m^3)^{-1}$ = Per Microgram per Cubic Meter; CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk; $(mg/kg\text{-}day)^{-1}$ = Per Milligrams per Kilogram Body Weight per Day.
Source: US EPA (2003b).

5.2.5 *trans*-1,2-Dichloroethylene (1,2-tDCE)

US EPA (2010a,b) concluded that there was inadequate evidence from which to assess the carcinogenic potential of 1,2-cDCE, 1,2-tDCE, or their mixtures. Therefore, US EPA has not derived a CSF or IUR for 1,2-cDCE, 1,2-tDCE, or their mixtures. ATSDR (2017a) did not evaluate cancer risk from exposure to 1,2-tDCE.

6 Plaintiff-Specific Regulatory Risk Evaluation

This section summarizes the plaintiff's residential and employment history, including the duration of time that the plaintiff lived and spent time at Camp Lejeune, and a risk evaluation for the plaintiff based on the plaintiff's estimated exposures. I perform regulatory risk calculations based on exposure estimates for the plaintiff from the expert report of Dr. LaKind (2025), plaintiff-specific information about exposure duration (*i.e.*, time spent on-base), information about exposure frequency for the activities evaluated (*e.g.*, number of times per week), and US EPA's toxicity criteria for the chemicals of interest (when available), as summarized in Section 5, and applying standard risk assessment methodology as summarized in Section 3.

6.1 Plaintiff Background

As discussed in Ms. Dyer's deposition (Dyer, 2024), she was born Terry Fristoe on [redacted], 1956, and lived with her family and attended school at Camp Lejeune from August 1958 through January 1973 (from ages 2 through 16), for a total of 14.5 years, due to her father's position as a school principal at two schools on-base (first at Tarawa Terrace 2 [TT2] Elementary School and then at Berkeley Manor, located in the Mainside). According to Ms. Dyer's testimony and official housing records, the Fristoe family lived at four different addresses in Tarawa Terrace (TT) throughout their time on-base, with the exception of one year (June 1964 to May 1965) when they resided in Jacksonville, North Carolina (Dyer, 2024). Ms. Dyer attended TT2 Elementary School, Brewster Junior High School, and Lejeune High School until January 1973, when the family moved off-base (Dyer, 2024). TT2 Elementary School was located in TT, and Brewster Junior High School and Lejeune High School were both located in Hadnot Point (HP). Ms. Dyer testified that during the year they lived off-base (1964-1965), she still attended school at Camp Lejeune and would visit the base for social and recreational activities; however, her school records indicate that she attended school off-base, at Northwoods Elementary School, from 1964 to 1965 (Dyer, 2024). To be conservative, I assumed that Ms. Dyer attended school on-base and visited the base for activities from 1964 to 1965. With respect to drinking water consumption, Ms. Dyer stated that she and her family drank tap water and used it to make drinks, and estimated that she drank "at least ten" glasses of water per day (Dyer, 2024), though she did not specify the age or time period when this occurred. With respect to showering and bathing, Ms. Dyer recalled taking baths at home, with her sister when she was young and then by herself as she got older (Dyer, 2024). Ms. Dyer stated that at school, she had gym class every day and was required to shower afterward, though she did not specify the grade in which this began (Dyer, 2024). Ms. Dyer also indicated that she visited swimming pools on-base as a child, "often" in summer and "as much as once a week" in the winter (Dyer, 2025). Ms. Dyer did not provide details regarding the specific location of the pools, whether they were indoor or outdoor pools, or at which specific ages or in which specific years the visits were made (Dyer, 2024).

Ms. Dyer is claiming that her exposure to the water at Camp Lejeune is the cause of her bladder cancer, which she states she was diagnosed with in May 2009 (Dyer, 2024).

6.2 Plaintiff Exposure Estimates

Exposure estimates for the plaintiff were calculated based on the average of the monthly average concentrations of TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE over the duration of the plaintiff's exposure period from modeled treatment plant finished water concentrations for both the HP and TT WTPs, which are available in ATSDR's "Public Health Assessment for Camp Lejeune Drinking Water" (ATSDR, 2017a), as described in Dr. LaKind's report (LaKind, 2025). In general, exposures from drinking water were evaluated for both the HP and TT WTPs, while dermal and inhalation exposures from showering were evaluated only for the WTP that supplied water to the plaintiff's place of residence during the plaintiff's time on-base. Because Ms. Dyer resided and went to elementary school in TT, I considered water ingestion from the TT WTP. I also considered water ingestion from the HP WTP, because Ms. Dyer attended junior high and high schools in locations serviced by the HP WTP. To bound upper and lower water ingestion risks, I calculated ingestion exposures and risks from both areas assuming exposure for the whole 14.5 years. Because Ms. Dyer resided in TT, I evaluated residential shower exposures for Ms. Dyer from the TT WTP only. Because Ms. Dyer also testified to showering at school after gym class, I evaluated shower exposures at school (junior high and high schools) from the HP WTP. I assumed that Ms. Dyer's gym classes began in junior high school (September 1968) and lasted through high school, until January 1973, when her family left Camp Lejeune. Assuming exposure during the school year only (September through June), her total exposures from using the junior high and high school showers total 972 days (~4.4 years from ages 12-16, for ~221 days in school per year).

Plaintiff-specific TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE daily tap water exposure estimates from drinking water (ingestion exposure pathway) and showering (dermal and inhalation exposure pathways), are described in more detail in Dr. LaKind's expert report (LaKind, 2025). Because Ms. Dyer testified that she attended school and social and recreational activities on-base while living off-base for one year (June 1964 to May 1965), I included all exposures during that year other than exposures from bathing, because she would have bathed off-base, in her home, during that year. Risks were calculated for the following exposure pathways and scenarios for the exposure period of concern (approximately 14.5 years) for the plaintiff:

- <u>Drinking Water Ingestion</u> – For this exposure pathway, I evaluated three scenarios for both the HP and TT WTPs: (1) central tendency exposure (CTE), which assumes ingestion of 0.34-0.56 L of tap water per day from ages 2 through 16; (2) reasonable maximum exposure (RME), which assumes ingestion of 0.85-1.8 L of tap water per day from ages 2 through 16; and (3) custom high-end exposure scenario, which is based on a weighted average of RME ingestion rates for ages 2 through 5 (0.85 L per day) and 6 through 10 (1.3 L per day), and an ingestion rate of 2.4 L per day (equivalent to ten 8-ounce glasses) for ages 11 through 16.
    - Ms. Dyer claims to have ingested at least 10 glasses of water per day in her deposition (Dyer, 2024). Assuming 8-ounce glasses, this is equivalent to 2.4 L per day, and is reflected in the custom high-end scenario. As discussed in Dr. LaKind's report (LaKind, 2025), 2.4 L/day is likely overly conservative, as it is higher than the RME intake value for the 11- through 16-year-old age group (1.8 L/day).
- <u>Dermal and Inhalation Exposures from Showering</u> – For these exposure pathways, I calculated risks based on the CTE (50^{th} percentile) and RME (95^{th} percentile) dermal dose and inhalation concentration outputs from both a residential exposure model and a communal showering facility exposure model (ATSDR, 2024a), and based on the plaintiff's locations of residence and school attendance during the plaintiff's time at Camp Lejeune; these doses and concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025).
    - **Residential Shower Model (TT WTP):** Based on Ms. Dyer's period of residence at various addresses in TT (August 1958 through May 1964, and June 1965 through January 1973), her dermal and inhalation exposures from showering and bathing at home had a duration of 14.5 years. This model estimates average daily dermal and inhalation exposures from showering and bathing, assuming a five-person household. Exposure estimates were provided by Dr. LaKind and details of this model are further described in her report (LaKind, 2025).

Two scenarios were modeled: (1) CTE, which assumes there are two consecutive 7-minute showers in the morning and two consecutive 7-minute showers in the evening, followed by a 7-minute tub bath; and (2) RME, which assumes two consecutive 20-minute showers in the morning and two consecutive 20-minute showers in the evening, followed by a 20-minute tub bath. For both scenarios, Ms. Dyer's exposures are represented by the individual taking the tub bath. Note that the residential shower model accounts for additional household water uses, including appliances, sinks, and toilets.

- **Communal Shower Model (HP WTP, School Bathroom):** As discussed above, Ms. Dyer indicated that while at school, she had gym class every day and was required to shower afterward (Dyer, 2024). For this model, I calculated risks based on the CTE (50^{th} percentile) and RME (95^{th} percentile) dermal dose and inhalation concentration outputs from a communal showering facility exposure model (ATSDR, 2024a); these inhalation concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Both the CTE and RME exposures from the communal showering facility model are estimated assuming a mean daily shower duration of 8 minutes and a standard deviation of 5.3 minutes (the model's default values) for the population of people who use that facility (LaKind, 2025). As discussed in Dr. LaKind's report, using these inputs results in a range of shower durations of up to 18 minutes for 95% of the people being modeled (LaKind, 2025). Note that the communal shower model accounts for additional water uses, including sinks and toilets in the facility.

In addition to the baseline exposure pathways outlined above, Dr. LaKind's expert report also includes a summary of air exposure concentrations relevant to the indoor swimming pool exposure pathway that was evaluated for Ms. Dyer (LaKind, 2025). As discussed in the ATSDR "Public Health Assessment for Camp Lejeune Drinking Water," the main exposure pathway for an indoor swimming pool scenario is the inhalation pathway, with potential exposures from other pathways (*e.g.*, dermal pathway) being very minor and contributing little to the overall risk estimate (ATSDR, 2017a). Ms. Dyer mentioned in her deposition that she and her family would visit Camp Lejeune for recreational activities, including going to swimming pools, but did not provide details on the location of the pools and whether they were indoors or outdoors. As stated above, Ms. Dyer testified that she would go to the swimming pool "often" as a child and "in the winter as much as once a week" (Dyer, 2025). Therefore, I conservatively assumed that she visited indoor swimming pools once per week from age 2 through 12 and then twice per month in her teen years (ages 13-16), for a total of 642 visits, for 1 hour each visit. Without information on the specific location of the pool(s), I calculated swimming pool risks based on water concentrations from both the TT and HP WTPs. Additional details, including air exposure concentrations in the pool area, can be found in Appendix D.

In summary, the following exposure scenarios are evaluated for Ms. Dyer:

- The CTE exposure scenario, which includes the following exposure pathways: CTE drinking water ingestion (TT and HP WTPs), CTE dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).
- The RME exposure scenario, which includes the following exposure pathways: RME drinking water ingestion (TT and HP WTPs), RME dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).
- The custom high-end exposure scenario, which includes custom high-end drinking water ingestion (TT and HP WTPs), RME dermal and inhalation exposures from showering at home (TT WTP) and at school (junior high and high schools) (HP WTP), and inhalation exposure from swimming (TT and HP WTPs).

For the CTE, RME, and custom high-end scenarios, there are four combinations of ingestion and swimming pool pathways that could be considered in combination with TT WTP bathing and HP WTP school showering. I considered three combinations for each scenario, including the one likely to have the highest exposures (based on the exposure estimates summarized in Appendix D).

Pathways were summed in the following combinations for the CTE, RME, and custom high-end scenarios:

- TT WTP bathing, HP WTP school showering, and HP WTP ingestion and swimming pool;
- TT WTP bathing, HP WTP school showering, and TT WTP ingestion and swimming pool; and
- TT WTP bathing, HP WTP school showering, TT WTP ingestion, and HP WTP swimming pool.

6.3 Regulatory Risk Calculations

Risk calculations for the plaintiff based on the estimates of oral and dermal daily exposure doses (DEDs) and daily inhalation exposure concentrations (DECs), which were based on Dr. LaKind's expert report (LaKind, 2025), considering the exposure duration for the plaintiff (approximately 14.5 months) and applying the toxicity values summarized in Section 5, are shown in Table 6.1. More detail on the risk calculations (including chemical- and pathway-specific calculations) is presented in Appendix D. As shown, the ELCRs calculated for Ms. Dyer's estimated exposures are within US EPA's acceptable cancer risk range of 1×10^{-6} and 1×10^{-4} for all of the exposure pathways/scenarios and both water sources evaluated.

Table 6.1 ELCRs by Exposure Pathway for the Plaintiff[a]

Exposure Pathway	Water Source	Excess Lifetime Cancer Risks		
		Central Tendency	Reasonable Maximum	Custom High-End
Baseline Exposure Pathways				
Ingestion of Drinking Water	HP WTP	4×10^{-6}	1×10^{-5}	1×10^{-5}
	TT WTP	9×10^{-6}	2×10^{-5}	3×10^{-5}
Dermal Contact from Bathing at Home	TT WTP	9×10^{-6}	1×10^{-5}	1×10^{-5}
Inhalation from Bathing at Home	TT WTP	3×10^{-6}	1×10^{-5}	1×10^{-5}
Additional Exposure Pathways				
Dermal Contact from Showering at School	HP WTP	1×10^{-7}	2×10^{-7}	2×10^{-7}
Inhalation from Showering at School	HP WTP	4×10^{-8}	1×10^{-7}	1×10^{-7}
Inhalation from Swimming	HP WTP	6×10^{-5}	6×10^{-5}	6×10^{-5}
	TT WTP	3×10^{-5}	3×10^{-5}	3×10^{-5}
Total ELCR (All Pathways)				
Assuming Bathing from TT WTP, School Showering from HP WTP, and Drinking Water and Swimming from HP WTP		7×10^{-5}	9×10^{-5}	9×10^{-5}
Assuming Bathing from TT WTP, School Showering from HP WTP, and Drinking Water and Swimming from TT WTP		5×10^{-5}	8×10^{-5}	9×10^{-5}
Assuming Bathing from TT WTP, School Showering from HP WTP, Drinking Water from TT WTP, and Swimming from HP WTP		8×10^{-5}	1×10^{-4}	1×10^{-4}

Notes:
ELCR = Excess Lifetime Cancer Risk; HP = Hadnot Point; TT = Tarawa Terrace; WTP = Water Treatment Plant.
(a) All ELCRs are rounded to 1 significant digit and are based on values from tables in Appendix D.

As shown in Table 6.1, the maximum ELCR calculated for Ms. Dyer's estimated exposures (*i.e.*, custom high-end exposure scenarios for drinking water and swimming exposures from both the TT and HP WTPs, combined with residential shower/bath exposures from TT WTP and school shower exposures from the HP WTP) is 1×10^{-4} (or 1 cancer case in 10,000 exposed people, or 0.01% increased risk), which does not exceed US EPA's acceptable excess cancer risk range. As discussed above, the custom high-end exposure scenario uses a dose that is based on a water consumption rate of 10 glasses of water each day for an older child, and is likely overly conservative for evaluating Ms. Dyer's exposures for ages 11 through 16 years. Further, the ELCR estimate is for all cancer types for all chemical exposures evaluated, with the majority of the calculated ELCR being driven by ingestion of vinyl chloride and inhalation of TCE (see Appendix D). As discussed in Section 5, none of the regulatory agency documents concluded that TCE, benzene, or vinyl chloride exposures are known causes of bladder cancer in humans. Although it is inconsistent with Dr. Goodman's conclusions (Goodman, 2025), as discussed in Section 5, ATSDR (2017b) concluded there is "sufficient evidence" for an association between PCE and bladder cancer risk. US EPA's cancer toxicity criteria (and cancer risk estimates) for these chemicals are not based on bladder cancer. The cancer toxicity criteria for PCE and vinyl chloride are based on liver cancer; the criteria for TCE are based on kidney cancer, liver cancer, and NHL combined; and the criteria for benzene are based on leukemia. Thus, cancer risk estimates from these chemicals are overly protective for bladder cancer risks. As shown in Appendix D, the highest estimated cancer risks for PCE (the only chemical for which ATSDR [2017b] concludes there is "sufficient evidence" of an association between exposure and bladder cancer) are within US EPA's target cancer risk range, with a maximum ELCR of 2×10^{-5}, or 2 cancer cases in 100,000 exposed people, or 0.002% increased cancer risk from the overly conservative custom high-end exposure scenarios (again, with cancer risks based on liver cancer and not bladder cancer).

It is also important to keep in mind that, as discussed in Section 3, these risk estimates are protective of the whole population (including sensitive individuals). In addition, some of the toxicity criteria that are based on inhalation studies are extrapolated to toxicity criteria that can be applied to oral and dermal exposure pathways (or *vice versa*). These extrapolations include conservative assumptions, and therefore, the toxicity values derived based on these extrapolations likely overpredict both exposures and risks.

It is also important to note that there is some uncertainty in the modeled finished water concentrations of TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from the two WTPs that are available from ATSDR (2007b, 2013b). As described in the expert reports by Dr. Hennet (2024) and Dr. Spiliotopoulos (2024), ATSDR's modeled finished water concentrations are likely biased high as a result of several conservative assumptions in the modeling. These results suggest that exposures and risks calculated based on ATSDR's modeled concentrations may be overestimated.

6.4 Risk Evaluation Conclusion

Overall, the regulatory risk calculations support the conclusion that Ms. Dyer was not exposed to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE in tap water at Camp Lejeune at levels that are of concern for human health. Even at the highest potential exposures for Ms. Dyer, and applying conservative, health-protective assumptions, Ms. Dyer's exposures to chemicals in Camp Lejeune tap water did not increase her overall cancer risk by more than 0.01% (*i.e.*, 1×10^{-4}, or 1 cancer case in 10,000 exposed people) over her background cancer risk. This ELCR is within US EPA's acceptable risk range. The cancer risk from the only chemical for which ATSDR (2017b) concludes there is "sufficient evidence" of an association between exposure and bladder cancer – PCE (2×10^{-5}, or 2 cancer cases in 100,000 exposed people, or 0.002%) – is also within US EPA's cancer risk range. Therefore, one cannot reasonably conclude that Ms. Dyer's exposures to chemicals in Camp Lejeune tap water are causally associated with her bladder cancer.

Because the cancer risks presented in Table 6.1 are based on the application of conservative exposure assumptions and toxicity values, in Section 7, I have also conducted margin of exposure (MoE) comparisons of the exposures predicted for the plaintiff and the lowest exposure levels at which health effects have been observed (or exposure levels at which no effects have been observed, for some chemicals) in the human or animal studies that are the basis of the toxicity criteria. In Section 8, I have also conducted a comparison of the plaintiff's estimated exposures to exposures reported in epidemiology and animal studies relevant to bladder cancer.

7 Plaintiff-Specific Margins of Exposure

As discussed in Section 3, the exposure levels at which health effects are predicted to be associated with no (or a very low) response from animal or human studies are the starting points (*i.e.*, points of departure [PODs]) used to derive regulatory toxicity criteria. PODs are the doses from which linear extrapolation is conducted to lower doses for the derivation of cancer toxicity criteria. In this section, I compare the plaintiff's exposure estimates for the chemicals evaluated in this report to the chemical-specific PODs. These types of comparisons provide what is called margins of exposure (MoEs) between the exposure predicted for an individual and the lowest exposure levels at which health effects have been observed (or exposure levels at which no effects have been observed, for some chemicals) in human or animal studies. In comparison to the conservative regulatory risk calculations (described in Section 6) that are designed to assess risk for the most sensitive individual in a population, and for any concentration above zero (for carcinogens), MoEs provide a comparison of individual exposure estimates to concentrations much closer to those at which health effects have been reported in human studies (or in animal studies used to extrapolate to humans). As discussed in Section 3, the equation used to calculate MoEs is as follows:

$$\mathrm{MoE} = \frac{\text{POD for the Cancer Toxicity Value}}{\text{Individual LADD or LADE}}$$

If the MoE is greater than 1, that indicates that the POD (*i.e.*, estimated to reflect exposures related to no or very low responses) is higher than exposures estimated for the individual, providing support that adverse health effects would not be expected for the individual.

The PODs for the ingestion (or dermal) and inhalation pathways for each chemical assessed herein are presented in Section 5.2. The plaintiff-specific exposure levels and MoEs are presented in Appendix D. As shown in Tables D.1-D.3, the MoEs for the plaintiff range from 278 to 300,000,000. Therefore, the MoEs are orders of magnitude above 1, indicating that the plaintiff's estimated exposure levels to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE in tap water (*via* inhalation, ingestion, and dermal exposure) at Camp Lejeune were well below the exposure doses and concentrations used to derive the toxicity criteria for these chemicals, providing additional support that the plaintiff's exposures would not have been expected to lead to adverse health effects.

8 Consideration of Epidemiology and Animal Studies Relevant to Bladder Cancer

In this section, I compare the exposure estimates for the plaintiff to exposure information I identified from epidemiology and toxicology studies summarized in Dr. Goodman's expert report that evaluated the possible association between TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE exposure and bladder cancer risk (Goodman, 2025).

Although Dr. Goodman reviewed epidemiology studies that evaluated potential correlations between chemical exposures and bladder cancer in study participants who were stationed at Camp Lejeune, I did not consider exposure estimates from those studies because of the methodological limitations in the studies (*e.g.*, high likelihood of exposure misclassification) as discussed by Dr. Goodman (2025). Further, as discussed by Dr. Goodman (2025) with regard to these studies:

> Overall, there were no consistent associations reported between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and bladder cancer. Almost all risk estimates were statistically null and close to 1. The few reported statistically significant risk estimates were not reported across other analyses of the Camp Lejeune population. (Goodman, 2025)

8.1 TCE

As summarized in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" report (ATSDR, 2017b), concluded that there is "below equipoise evidence for causation" for TCE exposure and bladder cancer. US EPA (2011a, 2020a) concluded that there is some scientific evidence that high cumulative exposures to TCE can cause bladder cancer in humans; ATSDR (2019a), NTP (2015), and IARC (2014) did not conclude that exposure to TCE is associated with increased bladder cancer risk in humans (see Section 5). Dr. Goodman concluded that the "scientific evidence does not support a causal association between TCE and bladder cancer" (Goodman, 2025).

For plaintiff exposure comparisons, I relied on the only bladder cancer epidemiology study that Dr. Goodman evaluated that reported exposure estimates for TCE – a Nordic occupational case-control study conducted by Hadkhale *et al*. (2017). Hadkhale *et al*. (2017) reported statistically significant associations between TCE exposure and bladder cancer incidence; however, all of the other six TCE bladder cancer studies Dr. Goodman discusses (none of which reported exposure information) did not report statistically significant associations. Further, there are limitations to the Hadkhale *et al*. (2017) study, as discussed by Dr. Goodman (2025), including the use of self-reported occupational history to estimate exposures, and a lack of adjustment for smoking, which is a known risk factor for bladder cancer (ACS, 2024b). The TCE inhalation exposures evaluated by Hadkhale *et al*. (2017) ranged from 32.8 to 129.5 ppm-years. I converted the lowest exposure estimate in this range from occupational exposure to continuous daily exposure for a resident (32.8 ppm-years × [5 ÷ 7 days/week] × [8 ÷ 24 hours/day] = 7.8 ppm-years). This TCE exposure estimate is much higher (70-fold) than those estimated for Ms. Dyer (0.11 ppm-years). I calculated Ms. Dyer's cumulative inhalation exposure to TCE in ppm-years by summing the cumulative ppm-year exposures from bathing at home, showering at school, and swimming pool vapor.[7,8]

Ms. Dyer's TCE exposure estimates are well below those reported in the oral animal bioassays discussed by Dr. Goodman (2025). Dr. Goodman's report indicates that there are no significant increases or trends in bladder tumors in chronic animal bioassays at oral TCE doses up to 2,339 mg/kg-day (Goodman, 2025). This dose is orders of magnitude higher than Ms. Dyer's maximum estimated TCE oral LADD of 0.00018 mg/kg-day.

See Appendix D for Ms. Dyer's inhalation exposure and oral dose estimates.

8.2 PCE

As summarized in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" report (ATSDR, 2017b), concluded that there is "sufficient evidence for causation" for PCE exposure and bladder cancer. ATSDR (2019b), in its toxicological profile for PCE, and US EPA (2012b, 2020b) concluded that there is weak scientific evidence for an association between PCE and bladder cancer (see Section 5). IARC (2014) concluded that there is limited evidence in humans for the carcinogenicity of PCE, stating that "positive associations have been observed for cancer of the bladder" (see Section 5). Dr. Goodman concluded that the "scientific evidence does not support a causal association between PCE and bladder cancer" (Goodman, 2025).

For plaintiff exposure comparisons, I relied on the Hadkhale *et al*. (2017) study, which was the only bladder cancer epidemiology study that Dr. Goodman evaluated that reported exposure estimates for PCE. Hadkhale *et al*. (2017) reported no significant associations (and no trends) between bladder cancer and PCE inhalation exposures at concentrations as high as 87.55 ppm-years. I converted this exposure estimate from occupational exposure to continuous daily exposure for a resident (87.55 ppm-years × [5 ÷ 7 days/week] × [8 ÷ 24 hours/day] = 21 ppm-years). This PCE exposure estimate is ~50-fold higher than those estimated for Ms. Dyer (0.45 ppm-years). I calculated Ms. Dyer's cumulative PCE exposure in ppm-years by summing the cumulative ppm-year exposures from bathing and swimming pool vapor.[9,10] Note that PCE was not detected in the water at HP during Ms. Dyer's time at Camp Lejeune; therefore, there is no PCE inhalation exposure for Ms. Dyer from showering at school.

---

[7] 1 ppm TCE = 5,370 μg/m^3 (CDC, 2019a).

[8] I estimated the ppm-year concentration for bathing exposure by converting the daily TCE concentration from bath vapor (2.9 μg/m^3) to ppm (0.00054 ppm) and multiplying by the number of years Ms. Dyer took tub baths at Camp Lejeune (14.5 years): 0.00054 ppm × 14.5 years = 0.0078 ppm-years. I estimated the ppm-year concentration for school showering exposure by converting the daily TCE concentration from shower vapor (0.57 μg/m^3) to ppm (0.00011 ppm) and multiplying by the number of years Ms. Dyer showered at school (4.4 years × 221 days/year ÷ 365 days/year = 2.66 years): 0.00011 ppm × 2.66 years = 0.00028 ppm-years. I estimated the ppm-year concentration for swimming vapor exposure by converting the daily TCE concentration from swimming vapor (350 μg/m^3) to ppm (0.065 ppm) and multiplying by the number of years Ms. Dyer spent at indoor swimming pools ([2 days/month × 12 months/year × 4.1 years] + [4 days/month × 12 months/year × 10.4 years] ÷ 365 days/year = 1.6 years): 0.065 ppm × 1.6 years = 0.10 ppm-years. I then summed the ppm-year cumulative exposure concentrations from bathing, school showering, and swimming: 0.0078 ppm-years + 0.00028 ppm-years + 0.10 ppm-years = 0.11 ppm-years.

[9] 1 ppm PCE = 6,780 μg/m^3 (CDC, 2019b).

[10] I estimated the ppm-year concentration for bathing exposure by converting the daily PCE concentration from bath vapor (53 μg/m^3) to ppm (0.0078 ppm) and multiplying by the number of years Ms. Dyer took tub baths at Camp Lejeune (14.5 years): 0.0078 ppm × 14.5 years = 0.11 ppm-years. I estimated the ppm-year concentration for swimming vapor exposure by converting the daily PCE concentration from swimming vapor (1,421 μg/m^3) to ppm (0.21 ppm) and multiplying by the number of years Ms. Dyer spent at indoor swimming pools ([2 days/month × 12 months/year × 4.1 years] + [4 days/month × 12 months/year × 10.4 years] ÷ 365 days/year = 1.6 years) : 0.21 ppm × 1.6 years = 0.34 ppm-years. I then summed the ppm-year cumulative exposure concentrations from bathing and swimming: 0.11 ppm-years + 0.34 ppm-years = 0.45 ppm-years.

Ms. Dyer's PCE exposure estimates are well below those reported in the oral animal bioassays discussed by Dr. Goodman (2025). Dr. Goodman's report indicates that there are no significant increases or trends in bladder tumors in chronic animal bioassays at oral PCE doses up to 1,072 mg/kg-day (Goodman, 2025). This dose is orders of magnitude higher than Ms. Dyer's maximum estimated PCE oral LADD of 0.00042 mg/kg-day.

See Appendix D for Ms. Dyer's inhalation exposure and oral dose estimates.

8.3 Benzene

As summarized in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" (ATSDR, 2017b), concluded that the evidence for an association between benzene exposure and bladder cancer was "below equipoise… for causation." US EPA (2003a), ATSDR (2007a, 2015), and IARC (2018) did not conclude that there is a causal association between exposure to benzene and bladder cancer in humans (see Section 5). Dr. Goodman concluded that "the evidence does not support a causal association between benzene and bladder cancer" (Goodman, 2025).

For plaintiff exposure comparisons, I relied on the Hadkhale *et al*. (2017) study, which was the only bladder cancer epidemiology study that Dr. Goodman evaluated that reported exposure estimates for benzene. Hadkhale *et al*. (2017) reported no significant associations (and no trends) between bladder cancer and benzene inhalation exposures at concentrations as high as 15.04 ppm-years. I converted this exposure estimate from occupational exposure to continuous daily exposure for a resident (15.04 ppm-years × [5 ÷ 7 days/week] × [8 ÷ 24 hours/day] = 3.6 ppm-years). This exposure estimate is orders of magnitude (~790-fold) higher than those estimated for Ms. Dyer (0.0045 ppm-years). I calculated Ms. Dyer's cumulative benzene exposure in ppm-years by summing the cumulative ppm-year exposures from school showering and swimming pool vapor.[11,12] Note that benzene was not detected in water at TT; therefore, there is no benzene inhalation exposure for Ms. Dyer from bathing.

Ms. Dyer's benzene exposure estimates are well below those reported in the oral animal bioassays discussed by Dr. Goodman (2025). Dr. Goodman's report indicates that there are no significant increases or trends in bladder tumors in chronic animal bioassays at benzene oral doses up to 200 mg/kg-day (Goodman, 2025). This dose is orders of magnitude higher than Ms. Dyer's maximum estimated benzene oral LADD of 0.0000081 mg/kg-day.

See Appendix D for Ms. Dyer's inhalation exposure and oral dose estimates.

8.4 Vinyl Chloride

As discussed in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" report (ATSDR, 2017b), concluded that the evidence for an association between vinyl chloride exposure and bladder cancer was "below equipoise… for causation."

[11] 1 ppm benzene = 3,190 μg/m^3 (CDC, 2019c).

[12] I estimated the ppm-year concentration for school showering exposure by converting the daily benzene concentration from shower vapor (0.037 μg/m^3) to ppm (0.000016 ppm) and multiplying by the number of years Ms. Dyer showered at school (4.4 years × 221 days/year ÷ 365 days/year = 2.66 years): 0.000016 ppm × 2.66 years = 0.000031 ppm-years. I estimated the ppm-year concentration for swimming vapor exposure by converting the daily benzene concentration from swimming vapor (9 μg/m^3) to ppm (0.0028 ppm) and multiplying by the number of years Ms. Dyer spent at indoor swimming pools ([2 days/month × 12 months/year × 4.1 years] + [4 days/month × 12 months/year × 10.4 years] ÷ 365 days/year = 1.6 years) : 0.0028 ppm × 1.6 years = 0.0045 ppm-years. I then summed the ppm-year cumulative exposure concentrations from school showering and swimming: 0.000031 ppm-years + 0.0045 ppm-years = 0.0045 ppm-years.

ATSDR (2024b) and US EPA (2003b) did not conclude that vinyl chloride exposure is a known cause of bladder cancer in humans. Dr. Goodman concluded that overall, "the evidence does not support a causal association between vinyl chloride and bladder cancer" (Goodman, 2025).

Dr. Goodman did not identify any epidemiology studies that evaluated potential associations between vinyl chloride and bladder cancer that also included vinyl chloride exposure estimates (Goodman, 2025). Dr. Goodman also did not identify any animal inhalation bioassays that evaluated potential associations between vinyl chloride and bladder cancer (Goodman, 2025). Dr. Goodman does discuss several chronic animal oral bioassays for vinyl chloride, which I relied for plaintiff exposure comparisons.

Ms. Dyer's vinyl chloride exposure estimates are well below those reported in the oral animal bioassays discussed by Dr. Goodman (2025). Dr. Goodman's report indicates that there are no significant increases or trends in bladder tumors in chronic animal bioassays at oral vinyl chloride doses up to 300 mg/kg-day (Goodman, 2025). This dose is orders of magnitude higher than Ms. Dyer's maximum estimated vinyl chloride oral LADD of 0.000034 mg/kg-day.

See Appendix D for Ms. Dyer's oral dose estimates.

8.5 1,2-tDCE

As summarized in Section 5, Dr. Goodman concluded that overall, the scientific evidence (including epidemiology and toxicology studies) is too limited to address whether there is a causal association between 1,2-tDCE and bladder cancer (Goodman, 2025). ATSDR (2017b) provided no comment on whether there is a causal association between 1,2-tDCE exposure and bladder cancer. US EPA and ATSDR do not conclude that there is an association between exposure to 1,2-tDCE and bladder cancer (see Section 5). Therefore, exposure comparisons cannot be made for 1,2-tDCE.

8.6 Conclusions from Epidemiology and Toxicology Studies

As described above, Ms. Dyer's exposures to TCE, PCE, benzene, and vinyl chloride were well below levels in epidemiology and animal studies that overall did not observe significant increases (or significant trends) in bladder cancer. Therefore, these results provide additional support that Ms. Dyer's estimated exposures would not have been expected to lead to her bladder cancer.

9 Rebuttal of the Plaintiff's Experts' Reports

I reviewed the reports of the plaintiff's experts Dr. Kelly Reynolds (2025a,b), who provided exposure estimates for the plaintiff, and Drs. Benjamin Hatten (2025), Thomas Longo (2025), and Steven M. Bird (2025), who provided opinions on specific causation for Ms. Dyer. Below, I note the methodological flaws in their analyses, with respect to risk assessment.

9.1 Dr. Reynolds

Dr. Reynolds' report (Reynolds, 2025a,b) does not provide reliable estimates of TCE, PCE, vinyl chloride, and benzene exposures for Ms. Dyer with which to evaluate potential adverse health effects.

Dr. Reynolds relies on ATSDR's monthly modeled concentrations (in µg/L) of TCE, PCE, vinyl chloride, and benzene to calculate total cumulative amounts (in µg) of each chemical summed over time, based on plaintiff-specific drinking water ingestion rates and exposure durations for the total time the plaintiff spent at Camp Lejeune (Reynolds, 2025a,b). Dr. Reynolds describes that her exposure scenarios are based on military field manuals and plaintiff depositions. Dr. Reynolds provides these estimates in plaintiff-specific "exposure assessment charts" in her report (Reynolds, 2025a,b).

Although Dr. Reynolds' calculations are not clearly explained, it appears that she first calculated a cumulative µg/L-month concentration for the plaintiff based on the chemical concentrations and the number of months the plaintiff resided at Camp Lejeune. She also calculated a total chemical mass (in µg) for each plaintiff based on the water concentration and the daily ingestion rate; these calculations were further explained in her calculation summary (Reynolds, 2025c). With respect to Dr. Reynolds' use of total amount (µg) as an oral exposure estimate – this is not a standard exposure metric used in risk assessment. As previously discussed in Section 3.3.2, oral and dermal exposure estimates are represented by the daily dose of a chemical taken into the body, averaged over the appropriate exposure period and expressed in units of milligrams of chemical per kilogram of human body weight per day (mg/kg-day). Inhalation exposure estimates represent the daily exposure concentration of a chemical taken into the body, averaged over the appropriate exposure period and expressed in units of micrograms of a chemical per cubic meter of air ($\mu g/m^3$). As discussed in Section 3, doses and inhalation exposure estimates can then be used to calculate excess lifetime cancer risks (ELCRs) using US EPA's chemical-specific toxicity criteria, and then the results can be compared to US EPA guidelines for acceptable ELCRs. Therefore, Dr. Reynolds representation of exposure as the total ingested amount of a chemical (µg) cannot be used directly to evaluate potential health effects for the plaintiff. That is, the mass of ingested chemical needs to be divided by body weight for the plaintiff and averaged over the appropriate averaging time, as described in Section 3, and as presented in my report for the plaintiff (in Section 6), so that the oral doses can be used to calculate ELCRs per US EPA risk assessment guidelines.

Further, total mass is not a useful metric for comparisons to exposure estimates in most reliable animal or epidemiology studies. Doses (mg/kg-day) or inhalation concentrations ($\mu g/m^3$) are typically used in animal bioassays for evaluating potential health effects from chemical exposures. Most reliable epidemiology studies provide cumulative exposure estimates in ppm-year (*i.e.*, inhalation exposure concentration × number of years exposed) and ppb-month or ppb-year (*i.e.*, ingested water concentration × number of months or years exposed). Thus, there is no risk-based comparison that can be made between total ingested mass and exposure information from relevant animal or epidemiology studies.

9.2 Drs. Longo. Hatten, and Bird

The reports of Drs. Hatten (2025), Longo (2025), and Bird (2025) do not provide a reliable analysis of specific causation or risk of bladder cancer with regard to Ms. Dyer's alleged exposures.

- Dr. Hatten concludes that Ms. Dyer's exposures while at Camp Lejeune were "substantial" and "at levels recognized to be hazardous to humans and consistently demonstrate elevated measures of association with bladder cancer" (Hatten, 2025).
- Dr. Longo concludes that Ms. Dyer's exposures to TCE and PCE at Camp Lejeune were "significant and substantial," and "at least as likely as not a cause of her bladder cancer" (Longo, 2025).
- Dr. Bird concludes that Ms. Dyer's exposures to chemicals at Camp Lejeune were "at a substantial level that is generally capable of causing the development of cancer and of bladder cancer. These exposures were significant and were not minimal or insignificant" (Bird, 2025).

All three experts make these conclusions without providing a robust analysis of the best available scientific information relevant to the potential specific causal association between exposure to these chemicals and Ms. Dyer's bladder cancer. In addition, all three experts' reliance on Dr. Reynolds' exposure estimates is seriously flawed, is not consistent with US EPA risk assessment guidelines, and cannot be relied upon to evaluate risks for Ms. Dyer. Below, I describe several flaws in the analyses of these three experts:

- Dr. Hatten's, Dr. Longo's, and Dr. Bird's risk evaluations are not consistent with US EPA's risk assessment guidelines, which consider not only exposure concentrations, but also exposure frequency and duration.
  - As discussed in Sections 3 and 5, exposure frequency and duration are critical components of US EPA's risk assessment methodology. It is only when the exposure concentrations, in combination with exposure frequencies and durations, result in doses exceeding US EPA's toxicity criteria (*i.e.*, result in a risk estimate that exceeds US EPA's acceptable targets) that there is concern for potential adverse health effects. And even with slight exceedances of US EPA's conservative risk targets, health effects are not necessarily expected to occur (as discussed in Section 3).
- Drs. Hatten, Longo, and Bird all rely on Dr. Reynolds' exposure charts as support that Ms. Dyer's exposures were "significant" and "substantial," but provide no basis for this conclusion other than simply pointing to the total mass values reported by Dr. Reynolds. As discussed in Section 9.1, estimates of total chemical mass exposure over time cannot be used directly to evaluate potential health effects for the plaintiff, because there are no total mass exposure estimates from relevant animal or epidemiology studies against which to make reliable risk-based comparisons. Exposures need to be estimated as oral doses of mg/kg-day or inhalation doses of $\mu g/m^3$, per US EPA risk assessment guidelines. Adding up mass over many days and years will, undoubtedly, result in a very large value. But it is an incorrect value for the purpose of risk evaluation. Therefore, Dr. Hatten's, Dr. Longo's and Dr. Bird's conclusions based on estimates of total chemical mass exposure for Ms. Dyer are meaningless and misleading and cannot be relied upon for risk evaluation for Ms. Dyer.

- Dr. Hatten, Dr. Longo, and Dr. Bird rely on a study by Aschengrau *et al*. (1993) as supporting an association between PCE exposure and bladder cancer. However, as discussed by Dr. Goodman (2025), there are limitations in this study, including a very small number of exposed cases (four), and a statistically unstable odds ratio (OR) because of the large confidence interval (CI) (OR = 6.04, 95% CI 1.32-21.84).
  - In addition, Dr. Bird relies on the 27.1 and 44.1 mg amounts of PCE cited in Aschengrau *et al*. (1993) as levels necessary to cause bladder cancer. However, the masses that Aschengrau *et al*. (1993) refer to are not oral doses from drinking water. Aschengrau *et al*. (1993) describe these amounts as modeled masses of PCE estimated to enter a household over a certain period of time. However, Aschengrau *et al*. (1993) do not provide PCE exposure concentrations or doses for individuals in the study. Therefore, the study cannot be relied upon for conclusions regarding specific estimates of exposure to PCE and bladder cancer.
- Dr. Hatten's, Dr. Longo's, and Dr. Bird's reports also refer to exposure information from several Camp Lejeune studies to support their conclusions. However, as discussed in Dr. Goodman's report (Goodman, 2025), there are methodological limitations in these studies (*e.g.*, high likelihood of exposure misclassification). In addition, with regard to the Camp Lejeune studies, Dr. Goodman states the following:

  > Overall, there were no consistent associations reported between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and bladder cancer. Almost all risk estimates were statistically null and close to 1. The few reported statistically significant risk estimates were not reported across other analyses of the Camp Lejeune population. (Goodman, 2025)
- In addition, as discussed in Section 5, based on a comprehensive review of the best available and most current epidemiology and animal studies, Dr. Goodman (2025) concludes that the scientific evidence does not support a causal association between TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE exposure and bladder cancer.

As discussed in my report (Section 6), applying standard risk assessment methodology (*i.e.*, considering exposure concentrations in addition to exposure frequency and duration for the plaintiff), the excess lifetime cancer risks (ELCRs) estimated for Ms. Dyer's exposures do not exceed US EPA's acceptable cancer risk range.

Therefore, Dr. Reynolds', Dr. Hatten's, Dr. Longo's, and Dr. Bird's expert reports do not change my opinions, as discussed in my report and summarized in Section 10, regarding Ms. Dyer's claim that exposures from Camp Lejeune are the cause of her bladder cancer.

10 Conclusion and Summary of Opinions

Based on the conservative regulatory risk calculations discussed in Section 6, the MoE calculations discussed in Section 7, and consideration of the bladder cancer animal and epidemiology studies discussed in Section 8, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Ms. Dyer's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 14.5 years that she lived and spent time at Camp Lejeune are causally associated with her bladder cancer.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007a. "Toxicological Profile for Benzene." 438p., August.

Agency for Toxic Substances and Disease Registry (ATSDR). 2007b. "Analyses of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water at Tarawa Terrace and Vicinity, U.S. Marine Corps Base Camp Lejeune, North Carolina: Historical Reconstruction and Present-Day Conditions: Chapter A." July.

Agency for Toxic Substances and Disease Registry (ATSDR). 2013a. "Chapter A - Supplement 8: Field Tests, Data Analyses, and Simulation of the Distribution of Drinking Water with Emphasis on Intermittent Transfers of Drinking Water Between the Hadnot Point and Holcomb Boulevard Water-Distribution Systems." In "Analyses and Historical Reconstruction of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water Within the Service Areas of the Hadnot Point and Holcomb Boulevard Water Treatment Plants and Vicinities, U.S. Marine Corps Base Camp Lejeune, North Carolina." 212p., March. Accessed at https://www.atsdr.cdc.gov/sites/lejeune/docs/Chapter_A_Supplement_8.pdf.

Agency for Toxic Substances and Disease Registry (ATSDR). 2013b. "Analyses and Historical Reconstruction of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water Within the Service Areas of the Hadnot Point and Holcomb Boulevard Water Treatment Plants and Vicinities, U.S. Marine Corps Base Camp Lejeune, North Carolina: Chapter A." May.

Agency for Toxic Substances and Disease Registry (ATSDR). 2015. "Addendum to the Toxicological Profile for Benzene." 59p., June.

Agency for Toxic Substances and Disease Registry (ATSDR). 2017a. "Public Health Assessment for Camp Lejeune Drinking Water, U.S. Marine Corps Base Camp Lejeune, North Carolina." 202p., January 20.

Agency for Toxic Substances and Disease Registry (ATSDR). 2017b. "ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases." 152p., January 13.

Agency for Toxic Substances and Disease Registry (ATSDR). 2018a. "Minimal Risk Levels (MRLs) for professionals: MRL information for the general public." June 21. Accessed at https://www.atsdr.cdc.gov/mrls/index.html.

Agency for Toxic Substances and Disease Registry (ATSDR). 2018b. "Minimal Risk Levels (MRLs)." June 4. Accessed at https://www.atsdr.cdc.gov/minimalrisklevels/index.html.

Agency for Toxic Substances and Disease Registry (ATSDR). 2019a. "Toxicological Profile for Trichloroethylene." 511p., June. Accessed at https://www.atsdr.cdc.gov/ToxProfiles/tp19.pdf.

Agency for Toxic Substances and Disease Registry (ATSDR). 2019b. "Toxicological Profile for Tetrachloroethylene." 435p., June. Accessed at https://www.atsdr.cdc.gov/ToxProfiles/tp18.pdf.

Agency for Toxic Substances and Disease Registry (ATSDR). 2023. "Toxicological Profile for 1,2-Dichloroethene (Draft for Public Comment)." 222p., August. Accessed at https://www.atsdr.cdc.gov/toxprofiledocs/index.html.

Agency for Toxic Substances and Disease Registry (ATSDR). 2024a. "Shower and Household Water-use Exposure (SHOWER) Model v4.0." September 26. Accessed at https://www.atsdr.cdc.gov/pha-guidance/toolbox/index.html.

Agency for Toxic Substances and Disease Registry (ATSDR). 2024b. "Toxicological Profile for Vinyl Chloride (Final)." 312p., January. Accessed at https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=282&tid=51.

Aleksunes, LM; Eaton, DL. 2019. "Principles of toxicology." In *Casarett and Doull's Toxicology: The Basic Science of Poisons (Ninth Edition)*. (Ed.: Klaassen, CD), McGraw-Hill Education, New York, NY. p25-64.

American Cancer Society (ACS). 2024a. "Cancer Statistics Center." Accessed at https://cancerstatisticscenter.cancer.org.

American Cancer Society (ACS). 2024b. "Bladder cancer risk factors." March 12. Accessed at https://www.cancer.org/cancer/types/bladder-cancer/causes-risks-prevention/risk-factors.html.

Ames, BN; Gold, LS; Willett, WC. 1995. "The causes and prevention of cancer." *Proc. Natl. Acad. Sci. USA* 92:5258-5265.

Aschengrau, A; Ozonoff, D; Paulu, C; Coogan, P; Vezina, R; Heeren, T; Zhang, Y. 1993. "Cancer risk and tetrachloroethylene contaminated drinking water in Massachusetts." *Arch. Environ. Health* 48(5):284-292.

Bird, S. [University of Massachusetts, Chan Medical School]. 2025. "Corrected Expert Report of Steven Bird, MD with Regard to Track One Plaintiff Terry Dyer." 15p., February 13.

Bove, FJ; Ruckart, PZ; Maslia, M; Larson, TC. 2014. "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. doi: 10.1186/1476-069X-13-10.

Calabrese, EJ. 2023. "Dose-response: A fundamental concept in toxicology." In *Hayes' Principles and Methods of Toxicology (Seventh Edition)*. (Eds.: Hayes, AW; Kobets, T), CRC Press, Boca Raton, FL. p95-141. doi: 10.1201/9781003390008-4.

Cardarelli, JJ II; Ulsh, BA. 2018. "It is time to move beyond the linear no-threshold theory for low-dose radiation protection." *Dose Response* 16(3):1559325818779651. doi: 10.1177/1559325818779651.

Centers for Disease Control and Prevention (CDC). 2019a. "NIOSH Pocket Guide to Chemical Hazards record for trichloroethylene (CAS No. 79-01-6)." October 30. Accessed at https://www.cdc.gov/niosh/npg/npgd0629.html.

Centers for Disease Control and Prevention (CDC). 2019b. "NIOSH Pocket Guide to Chemical Hazards record for tetrachloroethylene (CAS No. 127-18-4)." October 30. Accessed at https://www.cdc.gov/niosh/npg/npgd0599.html.

Centers for Disease Control and Prevention (CDC). 2019c. "NIOSH Pocket Guide to Chemical Hazards record for benzene (CAS No. 71-43-2)." October 30. Accessed at https://www.cdc.gov/niosh/npg/npgd0049.html.

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(8):777-787. doi: 10.1093/annhyg/mel039.

Crump, KS; Allen, BC. [Science Research Systems, Inc.]. 1984. "Quantitative Estimates of Risk of Leukemia from Occupational Exposure to Benzene." Submitted to Occupational Safety and Health Administration (OSHA) Docket H-059B, Exhibit 152. 133p.

Dyer, TF. 2024. "Videotaped deposition and other relevant plaintiff-specific materials of Terry F. Dyer [re: Terry F. Dyer *vs*. United States of America]." Submitted to US District Court, Eastern District of North Carolina. File No.: 7:23-CV-0357.

Dyer, T. 2025. "Declaration of Terry Dyer." 4p., January 30. [PLG-EXPERT_BIRD_DYER_0000000001 - PLG-EXPERT_BIRD_DYER_0000000004]

Feron, VJ; Hendriksen, CFM; Speek, AJ; Til, HP; Spit, BJ. 1981. "Lifespan oral toxicity study of vinyl chloride in rats." *Food Cosmet. Toxicol.* 19:317-333.

Golden, R; Bus, J; Calabrese, E. 2019. "An examination of the linear no-threshold hypothesis of cancer risk assessment: Introduction to a series of reviews documenting the lack of biological plausibility of LNT." *Chem. Biol. Interact.* 301:2-5. doi: 10.1016/j.cbi.2019.01.038.

Goodman, JE. [Gradient]. 2025. "Trichloroethylene, Perchloroethylene, Benzene, Vinyl Chloride, and trans-1,2-Dichloroethylene Exposure and Bladder Cancer Risk." 370p., February 7.

Hadkhale, K; Martinsen, JI; Weiderpass, E; Kjaerheim, K; Sparen, P; Tryggvadottir, L; Lynge, E; Pukkala, E. 2017. "Occupational exposure to solvents and bladder cancer: A population-based case control study in Nordic countries." *Int. J. Cancer* 140(8):1736-1746. doi: 10.1002/ijc.30593.

Hatten, B. [Toxicology Associates, Prof. LLC]. 2025. "Expert Report re: Camp Lejeune (Dyer)." 7p., February 7.

Hennet, RJC. [S.S. Papadopulos & Associates, Inc.]. 2024. "Expert Report of Remy J.-C. Hennet, In the United States District Court, Eastern District of North Carolina, No. 7:23-cv-897, In Re: Camp Lejeune Water Litigation." Report to US Dept. of Justice (US DOJ). 221p., December 9.

International Agency for Research on Cancer (IARC). 2014. "Trichloroethylene." In "IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 106: Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents." IARC Monograph No. 106. International Agency for Research on Cancer (IARC), World Health Organization (WHO). p35-218. Accessed at http://monographs.iarc.fr/ENG/Monographs/vol106/mono106-001.pdf.

International Agency for Research on Cancer (IARC). 2018. "IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 120: Benzene." IARC Monograph No. 120. International Agency for Research on Cancer (IARC); World Health Organization (WHO). 307p.

Japan Industrial Safety Association (JISA). 1993. "Carcinogenicity Study of Tetrachloroethylene by Inhalation in Rats and Mice: Data No. 3-1." 53p., March 31.

LaKind, JS. [LaKind Associates, LLC]. 2025. "Expert Report of Judy S. LaKind, Ph.D. in the Matter of *Dyer v. United States*." April 8.

Longo, T. [Associated Urologists of North Carolina]. 2025. "Specific Causation Expert Report for Terry Dyer." 32p., February 7.

Maltoni, C; Lefemine, G; Ciliberti, A; eds. 1984. *Experimental Research on Vinyl Chloride Carcinogenesis. Part I: Review of Available Data on General Pathology and Carcinogenicity*. Princeton Archives of Research on Industrial Carcinogenesis: Volume 2. Scientific Publishing, Co., Princeton, NJ. p1-35.

Maslia, ML; Aral, MM; Ruckart, PZ; Bove, FJ. 2016. "Reconstructing historical VOC concentrations in drinking water for epidemiological studies at a U.S. Military base: Summary of results." *Water* 8(10):449. doi: 10.3390/w8100449.

Mayo Clinic. 2024. "Cancer risk: What the numbers mean." March 29. Accessed at https://www.mayoclinic.org/diseases-conditions/cancer/in-depth/cancer/art-20044092.

National Research Council (NRC). 1983. "Risk Assessment in the Federal Government: Managing the Process." Committee on the Institutional Means for Assessment of Risks to Public Health, National Academy Press, Washington, DC. 191p. Accessed at https://www.nap.edu/catalog/366/risk-assessment-in-the-federal-government-managing-the-process.

National Research Council (NRC). 2009. "Toward a unified approach to dose-response assessment: The need for an improved dose-response framework." In *Science and Decisions: Advancing Risk Assessment*. Committee on Improving Risk Analysis Approaches Used by the US EPA. The National Academies Press, Washington, DC. p118-173.

National Toxicology Program (NTP). 2015. "Report on Carcinogens Monograph on Trichloroethylene (Draft)." 406p., January. Accessed at https://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf.

Olsen, GW; Butenhoff, JL; Cook, RR. 2014. "Epidemiology for toxicologists." In *Hayes' Principles and Methods of Toxicology (Sixth Edition)*. (Eds.: Hayes, AW; Kruger, CL), CRC Press, Boca Raton, FL. p527-570.

Paustenbach, DJ; Price, PS; Ollison, W; Blank, C; Jernigan, JD; Bass, RD; Peterson, HD. 1992. "Reevaluation of benzene exposure for the Pliofilm (rubberworker) cohort (1936-1976)." *J. Toxicol. Environ. Health* 36(3):177-231.

Popper, H; Maltoni, C; Selikoff, IJ. 1981. "Vinyl chloride-induced hepatic lesions in man and rodents. A comparison." *Liver* 1:7-20.

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(12):1182-1192.

Reynolds, KA. [University of Arizona, Mel and Enid Zuckerman College of Public Health]. 2025a. "Cumulative Exposure Expert Report, Kelly A Reynolds, MSPH, PhD." February 7. [PLG-EXPERT_REYNOLDS_0000000001 - PLG-EXPERT_REYNOLDS_0000000027]

Reynolds, KA. [University of Arizona, Mel and Enid Zuckerman College of Public Health]. 2025b. "Corrected Appendix 3: Terry F Dyer (Bladder Cancer)" 12p., February 7.

Reynolds, KA. [University of Arizona, Mel and Enid Zuckerman College of Public Health]. 2025c. "Calculations Summary." 28p., March 28.

Rinsky, RA; Smith, AB; Hornung, R; Filloon, TG; Young, RJ; Okun, AH; Landrigan, PJ. 1987. "Benzene and leukemia: An epidemiologic risk assessment." *N. Engl. J. Med.* 316:1044-1050.

Rinsky, RA; Young, RJ; Smith, AB. 1981. "Leukemia in benzene workers." *Am. J. Ind. Med.* 2(3):217-245. doi: 10.1002/ajim.4700020305.

Rodricks, JV; Rieth, SH. 1998. "Toxicological risk assessment in the courtroom: Are available methodologies suitable for evaluating toxic tort and product liability claims?" *Regul. Toxicol. Pharmacol.* 27:21-31.

Spiliotopoulos, A. [S.S. Papadopulos & Associates, Inc.]. 2024. "Expert Report of Alexandros Spiliotopoulos, PhD, In the United States District Court for the Eastern District of North Carolina, No. 7:23-cv-897, In Re: Camp Lejeune Water Litigation." Report to US Dept. of Justice (US DOJ). 138p., December 9.

Swenberg, JA; Lu, K; Moeller, BC; Gao, L; Upton, PB; Nakamura, J; Starr, TB. 2011. "Endogenous versus exogenous DNA adducts: Their role in carcinogenesis, epidemiology, and risk assessment." *Toxicol. Sci.* 120(Suppl. 1):S130-S145. doi: 10.1093/toxsci/kfq371.

US Congress. 1974. "Public Law 93-525: Safe Drinking Water Act of 1974." PL 93-525, 88 Stat 1660. December 16.

US EPA. 1989. "Risk Assessment Guidance for Superfund (RAGS). Volume I: Human Health Evaluation Manual (Part A) (Interim final)." Office of Emergency and Remedial Response. NTIS PB90-155581, EPA-540/1-89-002. 287p., December.

US EPA. 1990. "National Oil and Hazardous Substances Pollution Contingency Plan; Final rule." *Fed. Reg.* 55(46):8666-8865.

US EPA. 1991. "Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions." Office of Solid Waste and Emergency Response (OSWER). National Technical Information Service (NTIS). OSWER Directive 9355.0-30, NTIS PB91-92139, 10p., April 22.

US EPA. 1992. "Dermal Exposure Assessment: Principles and Applications (Interim)." Office of Health and Environmental Assessment, Exposure Assessment Group; Office of Research and Development, Center for Environmental Research Information. EPA-600/8-91-011B, January.

US EPA. 1993. "Reference Dose (RfD): Description and Use in Risk Assessments." IRIS Background Document 1A, March 15. Accessed at https://www.epa.gov/iris/reference-dose-rfd-description-and-use-health-risk-assessments.

US EPA. 1998. "Carcinogenic Effects of Benzene: An Update." National Center for Environmental Assessment (NCEA). PA/600/P-97/001F, 57p., April.

US EPA. 1999. "Extrapolation of the Benzene Inhalation Unit Risk Estimate to the Oral Route of Exposure." National Center for Environmental Assessment (NCEA). NCEA-W-0517, 21p., November.

US EPA. 2000. "Toxicological Review of Vinyl Chloride (CAS No. 75-01-4)." EPA/635/R-00/004. 197p., May.

US EPA. 2002. "A Review of the Reference Dose and Reference Concentration Processes (Final)." Risk Assessment Forum, Reference Dose/Reference Concentration (RfD/RfC) Technical Panel. EPA/630-P-02/002F, 192p., December.

US EPA. 2003a. "IRIS Chemical Assessment Summary for Benzene (CAS No. 71-43-2)." 43p., April 17. Accessed at https://www.epa.gov/iris.

US EPA. 2003b. "IRIS Chemical Assessment Summary for Vinyl Chloride (CAS No. 75-01-4)." 63p., October 28. Accessed at https://www.epa.gov/iris.

US EPA. 2004. "Risk Assessment Guidance for Superfund (RAGS). Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (Final)." Office of Superfund Remediation and Technology Innovation. EPA/540/R/99/005, OSWER 9285.7-02EP, PB99-963312. 156p., July.

US EPA. 2005. "Guidelines for Carcinogen Risk Assessment." Risk Assessment Forum. EPA/630/P-03/001F, 166p., March.

US EPA. 2010a. "IRIS Chemical Assessment Summary for trans-1,2-Dichloroethylene (CAS No. 156-60-5)." 21p., September 30. Accessed at https://www.epa.gov/iris.

US EPA. 2010b. "Toxicological Review of cis-1,2-Dichloroethylene and trans-1,2-Dichloroethylene (CAS Nos. cis: 156-59-2; trans: 156-60-5; mixture: 540-59-0) in Support of Summary Information on the Integrated Risk Information System (IRIS)." EPA/635/R-09/006F, 174p., September.

US EPA. 2011a. "Toxicological Review of Trichloroethylene (CAS No. 79-01-6) in Support of Summary Information on the Integrated Risk Information System (IRIS) (Final)." EPA/635/R-09/011F, 2469p., September.

US EPA. 2011b. "IRIS Chemical Assessment Summary for Trichloroethylene (CAS No. 79-01-6)." 65p., September 28. Accessed at https://www.epa.gov/iris.

US EPA. 2012a. "Benchmark Dose Technical Guidance." Risk Assessment Forum. EPA/100/R-12/001, 99p., June.

US EPA. 2012b. "Toxicological Review of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) in Support of Summary Information on the Integrated Risk Information System (IRIS) (Final)." EPA/635/R-08/011F, 1077p., February.

US EPA. 2012c. "IRIS Chemical Assessment Summary for Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4)." 38p., February 10. Accessed at https://www.epa.gov/iris.

US EPA. 2014. Memorandum from D. Stalcup to Superfund National Policy Managers, Regions 1-10 re: Human Health Evaluation Manual, Supplemental Guidance: Update of standard default exposure factors. Office of Solid Waste and Emergency Response (OSWER). OSWER Directive 9200.1-120, 7p., February 6.

US EPA. 2020a. "Risk Evaluation for Trichloroethylene (CASRN: 79-01-6) (Final)." EPA Document # 740R18008, 803p., November.

US EPA. 2020b. "Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro-) (CASRN: 127-18-4) (Final)." EPA Document # 740-R1-8011, 714p., December.

US EPA. 2021. "IRIS Glossary." March 11. Accessed at https://www.epa.gov/iris/iris-glossary.

US EPA. 2024. "Regional Screening Levels (RSLs) - User's Guide." November. Accessed at https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide.

Appendix A

***Curriculum Vitae* Lisa A. Bailey, Ph.D.**

GRADIENT

Lisa Bailey, Ph.D.
Principal
Lisa.Bailey@gradientcorp.com

Areas of Expertise

Human health risk assessment, exposure assessment, toxicology, DNA repair, mutagenesis, carcinogenesis.

Education

Ph.D., Biochemistry, Massachusetts Institute of Technology, 1995

B.A., *cum laude*, Chemistry, Skidmore College, 1989

Professional Experience

2006 – Present GRADIENT, Boston, MA
Principal. Provides expertise in human exposure assessment and toxicology in support of human health risk assessment and toxic tort litigation projects. Evaluates chemical toxicology data and reviews specific environmental chemical exposures to assess potential human health risks. Special emphasis on exposure assessment, toxicology, mode of action, genotoxicity, and carcinogenesis.

1999 – 2006 MENZIE-CURA & ASSOCIATES, INC., Winchester, MA
Senior Scientist. Managed human health risk assessments under the Massachusetts Contingency Plan and the US Environmental Protection Agency Superfund Program.

1996 – 1999 HARVARD SCHOOL OF PUBLIC HEALTH, Boston, MA
Post-Doctoral Fellow, Department of Molecular and Cellular Toxicology. Investigated the contribution of spontaneously generated abasic site DNA damage to spontaneous mutagenesis in the yeast *Saccharomyces cerevisiae* system. Compiled data regarding the origin of spontaneous mutations to better understand their role in the carcinogenesis process.

1989 – 1995 MASSACHUSETTS INSTITUTE OF TECHNOLOGY, Cambridge, MA
Ph.D. Student, Department of Biochemistry and Division of Toxicology. Investigated the mutational specificity of aflatoxin B_1 (AFB_1), a potent mutagen and carcinogen, in *Escherichia coli* through the use of an M13 genome containing the AFB_1-N7-Gua adduct in a defined position. Compared the mutational specificity observed in *E. coli* to that found in human liver cancers believed to be caused by aflatoxin.

Professional Affiliations

Society of Toxicology (Full Member); Society for Risk Analysis

2/14/2025

Select Projects

Confidential Client: In support of toxic tort litigation, reviewed toxicology, epidemiology, mechanistic, and exposure information related to claims of causal associations between trichloroethylene and perchloroethylene inhalation exposures and health effects (*e.g.*, pancreatic cancer and fetal heart malformation).

Confidential Client: In support of toxic tort litigation, reviewed toxicology, epidemiology, and exposure information related to claims of causal associations between exposures to chemicals associated with employment as an oil spill response worker and health effects (*e.g.*, respiratory and dermal effects).

Confidential Client: In support of toxic tort litigation, conducted an in-depth review of toxicology, epidemiology, mechanistic, and biomonitoring data related to claims of a causal association between exposure to glyphosate-based herbicides and Non-Hodgkin's Lymphoma.

Industrial Client: Performed an evaluation of occupational exposure and toxicity information for trichloroethylene to provide support in responding to US EPA's request for information under the 2016 Toxic Substances Control Act (TSCA).

Confidential Client: In support of toxic tort litigation, reviewed toxicology, epidemiology, and exposure information related to claims of causal associations between exposure to diesel exhaust, diesel fuel, silica, asbestos, and cancer endpoints (e.g., lung cancer, colon cancer, and hematological cancers).

Consumer Product Company: Assessed toxicity and human health risk related to potential leaching of chemicals (*i.e.*, nitrosamines) into a household appliance and into consumer tap water.

Consumer Product Company: Assessed toxicity and human health risk related to potential leaching of chemicals from a medical device.

Trade Association: Assessed the current state of the science on neurotoxicity from exposure to manganese in welding fumes and proposed a manganese occupational exposure limit for welders.

Consumer Product Company: Assessed toxicity and human health risk information related to exposure to mold and bacterial species identified in a children's toy product.

Trade Association: Performed in-depth evaluation of naphthalene toxicity and exposure data available in US EPA's ToxCast and ExpoCast programs in comparison to toxicity information from *in vivo* toxicity studies and ambient naphthalene exposure information.

Industrial Client: Performed an evaluation of occupational exposure and toxicity information for carbon tetrachloride, methylene chloride, and perchloroethylene to provide support in responding to US EPA's request for information under the 2016 Toxic Substances Control Act (TSCA).

Industrial Client: In support of toxic tort litigation, performed in-depth toxicological and risk evaluation for hexavalent chromium exposure for stainless steel welders.

Confidential Client: In support of toxic tort litigation, reviewed exposure information and medical records related to a claim of a causal association between inhalation exposure to naphthalene in mothballs and hemolytic anemia for the Plaintiffs.

Insurance Company: In support of toxic tort litigation, reviewed exposure information and medical records related to a claim of a causal association between formaldehyde inhalation exposure and acute myeloid leukemia.

Industrial Clients: In support of toxic tort litigation, assessed the current state of science on manganese neurotoxicity and human health, from exposure to manganese in air and soil, for workers and the general population.

Industrial Client: In support of toxic tort litigation, assessed the weight of epidemiological and toxicological evidence regarding the association between nitrosamine/amide inhalation and brain cancer.

Consumer Product Company: In support of toxic tort litigation, assessed the weight of epidemiological evidence regarding a causal association between inhalation exposures to trichloroethylene and perchloroethylene and cancer and non-cancer health effects.

Industrial Client: In support of toxic tort litigation, performed an extensive review of the mode-of-action data for asbestos and the epidemiology literature on vehicle brake repair and lung cancer and mesothelioma to assess whether there is a causal association.

Industrial Client: In support of toxic tort litigation, evaluated human health risk from exposure to chlorinated volatiles, including trichloroethylene and perchloroethylene, in groundwater *via* drinking water and showering.

Trade Association: Performed in-depth analysis of trichloroethylene and tetrachloroethylene toxicology and mechanistic data to evaluate whether the weight of the evidence supports the plausibility of trichloroethylene and tetrachloroethylene as a human renal carcinogen.

Trade Association: Performed in-depth analysis of methyl methacrylate toxicology and mechanistic data to evaluate the weight of evidence and propose an occupational exposure level.

Trade Association: Through Toxicology Excellence for Risk Assessment (TERA), participated in a peer review process of our proposed manganese reference concentration (RfC) (Bailey *et al*., 2009), which resulted in the values being posted on the National Library of Medicine's National Institute of Health TOXNET compilation of databases as an ITER (International Toxicity Estimates for Risk Assessment) value for manganese dioxide.

Industrial Client(s): For several industrial clients, reviewed current status of manganese inhalation toxicity criteria (reference concentration [RfC], American Conference of Governmental Industrial Hygienists Threshold Limit Value [ACGIH TLV]), and current manganese inhalation toxicity literature, in support of regulatory comment/communication and public communication regarding potential health effects from both occupational and residential exposure to manganese in air.

Trade Association: Performed in-depth analysis of methanol toxicology and mechanistic data to evaluate whether the weight of evidence supports the plausibility that methanol exposure is associated with human lymphoma.

Trade Association: Performed in-depth analysis of naphthalene toxicology and mechanistic data to evaluate whether the weight of evidence supports the plausibility of naphthalene as a human carcinogen.

Trade Association: Performed in-depth analysis of formaldehyde toxicology and mechanistic data to evaluate whether the weight of the evidence supports the plausibility of formaldehyde as a human leukemogen.

Chemical Company: Provided comments on US EPA's 2009 trichloroethylene draft reassessment, focusing on the use of novel methods for reference concentration (RfC) and reference dose (RfD) determination, such as US EPA's use of physiologically based pharmacokinetic (PBPK) modeling.

Industrial Client: Reviewed toxicity data and various agency derivations of perchlorate toxicity criteria.

Pharmaceutical Company: Performed in-depth analysis of the toxicology data of a specific drug to determine whether the company could have anticipated potential adverse side effects in humans.

Confidential Client: Performed literature review of health effects from inhalation of mercury vapor, focusing on reversibility and latency of effects.

Medical Device Manufacturing Company: Participated in evaluation of potential for adverse side effects from residual contamination on medical implant device.

Industrial Company: Reviewed current status of US EPA's manganese inhalation toxicity value, and current manganese inhalation toxicity literature, in support of litigation regarding claims of elevated manganese air concentrations.

Industrial Client: Managed a Superfund risk assessment for US EPA Region I, including a number of chemicals and human exposure pathways for children and adults: direct contact with sediment and soil, direct contact with surface water and groundwater, ingestion of fish, inhalation of indoor air and trench vapor, and inhalation of asbestos in resuspended sediment and soil. This risk assessment required application of US EPA's "Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens" for carcinogenic polycyclic aromatic hydrocarbons (PAHs) in all media.

Industrial Client: Performed a human health Superfund risk assessment for residential exposure to chlorinated volatile organic compounds (VOCs) and metals in drinking water and indoor air, and from potential exposure to metals in sediment and surface water. Part of the project involved participating in public meetings to address concerned citizen groups.

Industrial Client: Performed a risk assessment for the state of Connecticut, for potential residential risk from lead in sediment and blue crab. The risk assessment involved use of the Integrated Exposure Uptake Biokinetic (IEUBK) Model for lead and the Adult Lead Model.

Municipal Facility: Helped design a sampling plan and performed a risk evaluation for an asbestos site that was developed into an urban park. This project was carried out in conjunction with the Massachusetts Department of Environmental Protection (MassDEP), and was used as a model for development of the Draft MassDEP Asbestos in Soil Regulations.

Awards and Honors

Best Overall Abstract Award, "Evaluation of US EPA's Proposed Rule for the Occupational use of Carbon Tetrachloride and Proposal for a Revised Occupational Exposure Value," Risk Assessment Specialty Section (RASS), Society of Toxicology (SOT) 64th Annual Meeting and ToxExpo, 2025

Best Abstract Award, "Hypothesis-Based Weight-of-Evidence Evaluation and Risk Assessment for Naphthalene Carcinogenesis," Risk Assessment Specialty Section (RASS), Society of Toxicology (SOT) 54th Annual Meeting and ToxExpo, 2015

One of the Top Ten Abstracts, "Health-Protective Manganese Guideline for Welding and Other Occupations," Risk Assessment Specialty Section (RASS), Society of Toxicology (SOT) 53rd Annual Meeting and ToxExpo, 2014

One of the Best Published Papers, "Hypothesis-Based Weight-of-evidence Evaluation of Methyl Methacrylate Olfactory Effects in Humans and Derivation of an Occupational Exposure Level," Risk Assessment Specialty Section (RASS), Society of Toxicology (SOT), 2013

One of the Top Ten Best Published Papers, "Hypothesis-Based Weight-of-Evidence Evaluation of Methanol as a Human Carcinogen," Risk Assessment Specialty Section (RASS), Society of Toxicology (SOT), 2012

DNA Damage and Repair NASA Conference Travel Award, Antalya, Turkey, 1997

Mutagenesis Gordon Conference Travel Award, Plymouth, NH, 1996.

Publications and Book Chapters

Bailey, L; Marchitti, S. 2024 (Spring). "Evolving chemical risk evaluation and management under the Toxic Substances Control Act: Trichloroethylene as an example." *Gradient Trends* 90.

Mayfield, DB; Bailey, LA; Cohen, JM; Beck, BD. 2022. "Properties and effects of metals." In *Principles of Toxicology: Environmental and Industrial Applications (Fourth Edition)*. (Eds.: Roberts, SM; James, RC; Williams, PL), John Wiley & Sons, Inc., Hoboken, NJ. p357-380.

Bailey LA, Boomhower SR. 2021. "Potential implications of new information concerning manganese Ohio community health effects studies." *Regul. Toxicol. Pharmacol.* doi: 10.1016/j.yrtph.2021.105069.

Langseth, D; Chien, J; Bailey, L. 2021 (Spring). "Opening the Malden River for recreational boating." *Gradient Trends - Risk Science & Application* 81:1-2.

Bailey, L. 2021 (Spring) "Collaborating to promote chemical safety and animal welfare." *Gradient Trends - Risk Science & Application* 81:6.

Bailey, L. 2020 (Fall). "Worker risk evaluations under TSCA: What we know so far." *Gradient Trends - Risk Science & Application* 79:3,7.

Bailey, LA; Rhomberg, LR. 2020. "Incorporating ToxCast™ data into naphthalene human health risk assessment." *Toxicol. In Vitro.* doi: 10.1016/j.tiv.2020.104913.

Bailey, LA; Zu, K; Beck, BD. 2018. "Comment on 'Impact of air manganese on child neurodevelopment in East Liverpool, Ohio' by Haynes *et al.* (2018)." *Neurotoxicology* 68:A1-A2. doi: 10.1016/j.neuro.2018.07.017.

Bailey, LA; Beck, BD. 2017. "Comment on 'Environmental exposure to manganese in air: Associations with tremor and motor function' by Bowler *et al.* (2016)." *Sci. Total Environ.* 595:839-841. doi: 10.1016/j.scitoenv.2017.03.277.

Bailey, LA; Kerper, LE; Goodman, JE. 2017. "Derivation of an occupational exposure level for manganese in welding fumes." *Neurotoxicology* 64:166-176. doi: 10.1016/j.neuro.2017.06.009.

Bailey, L; Nascarella, M; Kerper, L; Rhomberg, L. 2015. "Hypothesis-based weight-of-evidence evaluation and risk assessment for naphthalene carcinogenesis." *Crit. Rev. Toxicol.* 46(1):1-42. doi: 10.3109/10408444.2015.1061477.

Bailey, LA; Kerper, LE; Rhomberg, LR. [Gradient]. 2015. "Naphthalene." In *Hamilton and Hardy's Industrial Toxicology (Sixth Edition)*. (Eds.: Harbison, RD; Bourgeois, MM; Johnson, GT), John Wiley & Sons, Inc., Hoboken, NJ, p663-668.

Goodman, JE; Peterson, MK; Bailey, LA; Kerper, LE; Dodge, DG. 2014. "Electricians' chrysotile asbestos exposure from electrical products and risks of mesothelioma and lung cancer." *Regul. Toxicol. Pharmacol.* 68(1):8-15.

Pemberton, M; Bailey, LA; Rhomberg, LR. 2013. "Hypothesis-based weight-of-evidence evaluation of methyl methacrylate olfactory effects in humans and derivation of an occupational exposure level." *Regul. Toxicol. Pharmacol.* 66:217-233.

Goodman, JE; Prueitt, RL; Sax, SN; Bailey, LA; Rhomberg, LR. 2013. "Evaluation of the causal framework used for setting National Ambient Air Quality Standards." *Crit. Rev. Toxicol.* 43(10):829-849.

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." *Crit. Rev. Toxicol.* 43(9):753-784.

Mayfield, DB; Lewis, AS; Bailey, LA; Beck, BD. 2015. "Properties and effects of metals." In *Principles of Toxicology: Environmental and Industrial Applications (Third Edition)*. (Eds.: Roberts, SM; James, RC; Williams, PL), John Wiley & Sons, Inc., Hoboken, NJ, p283-307.

Bailey, LA; Prueitt, RL; Rhomberg, LR. 2012. "Hypothesis-based weight-of-evidence evaluation of methanol as a human carcinogen." *Regul. Toxicol. Pharmacol.* 62:278-291.

Rhomberg, LR; Bailey, LA; Goodman, JE; Hamade, A; Mayfield, D. 2011. "Is exposure to formaldehyde in air causally associated with leukemia? - A hypothesis-based weight-of-evidence analysis." *Crit. Rev. Toxicol.* 41(7):555-621.

Prueitt, RL; Goodman, JE; Bailey, LA; Rhomberg, LR. 2011. "Hypothesis-based weight of evidence evaluation of the neurodevelopmental effects of chlorpyrifos." *Crit. Rev. Toxicol.* 42(10):822-903.

Rhomberg, LR; Bailey, LA; Goodman, JE. 2010. "Hypothesis-based weight of evidence – A tool for evaluating and communicating uncertainties and inconsistencies in the large body of evidence in proposing a carcinogenic mode of action – Naphthalene as an example." *Crit. Rev. Toxicol.* 40(8):671-696.

Goodman, JE; Dodge, DG; Bailey, LA. 2010. "A framework for assessing adverse effects in humans with a case study of sulfur dioxide." *Regul. Toxicol. Pharmacol.* 58:308-322.

Bailey, LA; Goodman, JE; Beck BD. 2009. "Proposal for a revised Reference Concentration (RfC) for manganese based on recent epidemiological studies." *Regul. Toxicol. Pharmacol.* 55:330-339.

Baird, SJS; Bailey, EA; Vorhees, DJ. 2007. "Evaluating human risk from exposure to alkylated polycyclic aromatic hydrocarbons in an aquatic system." *Hum. Ecol. Risk Assess.* 13:322-338.

Auerbach, P; Bennett, RAO; Bailey, EA; Krokan, HE; Demple, B. 2005. "Mutagenic specificity of endogenously generated abasic sites in *Saccharomyces cerevisiae* chromosomal DNA." *Proc. Natl. Acad. Sci. USA* 102:17711-17716.

Bailey, L. 2005. "Evaluating risk from asbestos in soil under the MCP." *LSP Assoc. Newsl.* 12(Oct.):7.

Smela, ME; Currier, SS; Bailey, EA; Essigmann, JM. 2001. "The chemistry and biology of aflatoxin B(1): From mutational spectrometry to carcinogenesis." *Carcinogenesis* 22(4):535-545.

Demple, B; Bailey, EA; Bennett, RAO; Masuda, Y; Wong, D; Xu, Y. 1998. *DNA Damage and Repair: Oxygen Radical Effects, Cellular Protection and Biological Consequences*. (Ed.: Dizdaroglu, M), Plenum Press, NY.

Bailey, EA; Iyer, RS; Stone, MP; Harris, TM; Essigmann, JM. 1996. "Mutational properties of the primary aflatoxin B1-DNA adduct." *Proc. Natl. Acad. Sci. USA* 93:1535-1539.

Bailey, EA; Iyer, RS; Harris, TM; Essigmann, JM. 1996. "A viral genome containing an unstable aflatoxin B1-N7 Gua adduct situated at a unique site." *Nucleic Acids Res.* 24:2821-2828.

Poster Presentations

Marchitti, SA; Bailey, LA. 2025. "Evaluation of US EPA's Proposed Rule for the Occupational Use of Carbon Tetrachloride and Proposal for a Revised Occupational Exposure Value." Abstract/Poster #4237/P751. Presented at the Society of Toxicology (SOT) 64th Annual Meeting and ToxExpo, Orlando, FL, March 16-20.
****Best Overall Abstract Award Winner, Risk Assessment Specialty Section**

Zu, K; Bailey, LA; Prueitt, RL; Beck, BD; Seeley, M. 2019. "Comparison of Lung Cancer Risks from Environmental Exposures to Arsenic and from Those Associated with Medical Monitoring Criteria for Smokers." Poster # 2776/P262. Presented at the Society of Toxicology (SOT) 58th Annual Meeting, Baltimore, MD, March 10-14.

Bailey, LA. 2019. "Evaluation of the Carcinogenic Mode of Action and Proposal for an Occupational Exposure Limit for Tetrachloroethylene." Poster # 1872/P255. Presented at the Society of Toxicology (SOT) 58th Annual Meeting, Baltimore, MD, March 10-14.

Bailey, LA; Rhomberg, LR. 2018. "Incorporating ToxCast Data into Naphthalene Human Health Risk Assessment." Poster # 2858/P381. Presented at the Society of Toxicology (SOT) 57th Annual Meeting, San Antonio, TX, March 11-15.

Bailey, LA; Lam, T; Peterson, MK; Beck, BD. 2017. "Does Hexavalent Chromium in Welding Fumes Cause Increased Lung Cancer Risk in Stainless Steel Welders?" Presented at the Society of Toxicology (SOT) 56th Annual Meeting, Baltimore, MD, March 12-16.

Bailey, L; Kerper, L; Goodman, J. 2016. "Occupational Exposure Level for Manganese in Welding Fumes Based on the Best Available Science." Presented at the Manganese 2016 Conference, New York, NY, September 25-28.

Bailey, L; Nascarella, M; Kerper, L; Rhomberg, L. 2015. "Hypothesis-based Weight-of-Evidence Evaluation and Risk Assessment for Naphthalene Carcinogenesis." Presented at the Society of Toxicology 54th Annual Meeting, San Diego, CA, March 22-26.

Bailey, L; Kerper, L; 2014. "Health-Protective Manganese Guideline for Welding and Other Occupations." Presented at the American Industrial Hygiene Association Fall Meeting, Washington, DC, October 20-21.

Bailey, L; Kerper, L; Beck, B. 2014. "Health-Protective Manganese Guideline for Welding and Other Occupations." Presented at the Society of Toxicology 53rd Annual Meeting, Phoenix, AZ, March 23-27.

Pemberton, M; Bailey, LA; Rhomberg, LR. 2013. "Weight-of-Evidence Evaluation of Methyl Methacrylate Olfactory Effects in Humans and Derivation of an Occupational Exposure Level." Presented at the Society of Toxicology 52nd Annual Meeting, San Antonio, TX. *Toxicologist* 132(1):473. Abstract No. 2216.

Prueitt, RL; Goodman, JE; Bailey, LA; Rhomberg, LR. 2012. "Hypothesis-Based Weight-of-Evidence Evaluation of the Neurodevelopmental Effects of Chlorpyrifos." Presented at the Society of Toxicology 51st Annual Meeting, San Francisco, CA. *Toxicologist* 126(1):309. Abstract No. 1430.

Bailey, LA; Goodman, JE; Beck, BD. 2012. "Revised Reference Concentration for Manganese Oxide Based on Recent Epidemiology and Pharmacokinetic Studies." Presented at the Society of Toxicology 51st Annual Meeting, San Francisco, CA, March. *Toxicologist* 126(1):213. Abstract No. 995.

Bailey, LA; Goodman, JE; Rhomberg, LR. 2011. "Hypothesis-based Weight-of-Evidence Evaluation of Naphthalene: Carcinogenic Hazard Assessment and Mode of Action." Presented at the Society of Environmental Toxicology and Chemistry (SETAC) North America 32nd Annual Meeting, Boston, MA, November 14, 1p.

Bailey, L; Hamade, AK; Rhomberg, LR. 2011. "Weight-of-Evidence Evaluation of a Plausible Mode of Action for Leukemogenesis from Inhalation Exposure to Formaldehyde." Presented at the Society of Toxicology 50th Annual Meeting, Washington, DC. *Toxicologist - Supplement to Toxicological Sciences* 120(Suppl. 2):417.

Peterson, MK; Bailey, L; Dodge, DG; Goodman, JE; Valberg, PA. 2011. "A weight-of-evidence evaluation of asbestos exposure and mesothelioma risk among electricians." *Toxicologist - Supplement to Toxicological Sciences* 120(Suppl. 2):414.

Hamade, AK; Bailey, L; Rhomberg, LR. 2011. "Does formaldehyde cause hematotoxicity? A weight-of-evidence evaluation of hematotoxicity studies in humans and animals in the context of leukemogenicity." *Toxicologist - Supplement to Toxicological Sciences* 120(Suppl. 2):417.

Goodman, JE; Mayfield, DB; Bailey, L; Rhomberg, LR. 2011. "Weight-of-evidence evaluation of formaldehyde exposure and leukemia risk." *Toxicologist - Supplement to Toxicological Sciences* 120(Suppl. 2):416.

Bailey, LA; Rhomberg, LR. 2009. "Hypothesis-Based Weight of Evidence (HBWoE) Evaluation of Naphthalene – Carcinogenic Hazard Assessment and Mode of Action." Presented at the 2009 Society for Risk Analysis Annual Meeting, Baltimore, MD, December 6-9.

Bailey, LA; Rhomberg, LR. 2009. "Hypothesis-Based Weight of Evidence – A Tool for Evaluating and Communicating Uncertainties and Apparent Contradictions in the Large Body of Evidence in Proposing a Potential Carcinogenic Mode of Action – Naphthalene as an Example." Presented at the 2009 Society of Toxicology Annual Meeting, Baltimore, MD, March 16-19.

Goodman, JE; Bailey, LA.; Beck, BD. 2008. "Recent Studies of the Health Effects of Manganese and the Implications for the Reference Concentration (RfC)." Presented at the 2008 Society of Toxicology Annual Meeting, Seattle, WA, March 16-20.

Bailey, L; Murray, D. 2006. "Comparison of EPA's Current Approach and a Proposed Approach to Evaluating Risk from Asbestos." Presented at the 2006 Brownfields Conference, Boston, MA, November 14.

Bailey, L; Murray, D. 2006. "Comparison of EPA's Current Approach and a Proposed Approach to Evaluating Risk from Asbestos." Presented at the 2006 University of Massachusetts at Amherst Conference on Soils, Sediments and Water, October 16-19.

Bailey, L; Lemay, J; Murray, D; Hunt, K. 2005. "Derivation of Soil Screening Values for the Vapor Intrusion Pathway – Is this a Valid Approach?" Presented at the 2005 University of Massachusetts at Amherst Conference on Soils, Sediments and Water, October 17-20.

Bailey, EA; Weil, M; Murray, D. 2004. "A Comparison of Risk Assessment Methods to Demonstrate Potential Risk from Exposure to Asbestos in Soil." Presented Poster at the 2004 University of Massachusetts at Amherst Conference on Soils, Sediments and Water, October 18-21.

Oral Public Comments, Presentations, and Testimony

Bailey, L. 2020. "Evaluation of Manganese Air Concentrations Related to the Ohio Manganese Research Program." Presented to US EPA, October 1.

Bailey, L. 2020. "Public Testimony Related to the Pennsylvania Department of Environmental Protection (PADEP) Proposed Rulemaking for 'Water Quality Standards for Manganese and Implementation.'" Pennsylvania Senate Environmental Resources & Energy Committee Hearing, September 9.

Bailey, L. 2020. "Public Testimony Related to the Pennsylvania Department of Environmental Protection (PADEP) Proposed Rulemaking for 'Water Quality Standards for Manganese and Implementation.'" PADEP Environmental Quality Board meeting, September 8.

Bailey, L. 2018. "Fenceline Air Monitoring: Interpretation and Risk Management." Presented at the Shale Insight Conference, October 23-25.

Bailey, L. 2018. "Review of Scientific Evidence Related to Potential Toxicity from Occupational Exposure to Manganese." Presented at the California Division of Occupation Safety and Health (DOSH) Health Advisory Committee (HEAC) Meeting, September 4.

Bailey, L. 2018. "Manganese Community Health Effects Studies: Interpretation and the Need to Consider Other Relevant Studies." Presented at the Air and Waste Management Association (A&WMA) Conference, Hartford, CT, June 28.

Bailey, L. 2017. Oral Comments Related to Potential Health Risks from Levels of Manganese, Benzene, Chromium, and Lead in Ambient Air in Lawrenceville, Pennsylvania. Presented at an Allegheny County Health Department (ACHD) Public Meeting, Lawrenceville, PA, December 4.

Bailey, L. 2017. Oral Comments Related to Potential Health Risks from Levels of Manganese in Ambient Air in East Liverpool, Ohio. Presented at an East Liverpool, Ohio, Public Meeting, October 24.

Bailey, L. 2017. "New Exposure Information Strategies for Chemical Risk Evaluation under the New TSCA." Presented as part of the Gradient Webinar Series, April 19.

Bailey, L. 2014. "Manganese Toxicity and Community Studies." Presented to the Manganese Interest Group Meeting, Washington, DC, October 16, 36p.

Bailey, L. 2014. Oral Public Comments Related to Naphthalene Toxicological Review at the US EPA Integrated Risk Information System (IRIS) Problem Formulation Meeting. September.

Bailey, L; Rhomberg, L. 2013. "Hypothesis-Based Weight of Evidence: An Approach to Assessing Causation and its Application to Regulatory Toxicology." Presented at the Society for Risk Analysis Annual Meeting, Arlington, VA, December 9.

Bailey, L. 2012. "Overview of Manganese Inhalation RfC and Oral RfD, and Implications for Interpretation of Community Studies." Presented to the Manganese Interest Group Meeting, Washington, DC, October 2, 41p.

Bailey, L. 2011. "Revised Manganese Reference Concentration – Implications for Interpretation of Recent Epidemiology Studies." Presented to the Manganese Interest Group Meeting, Washington, DC, June 15, 25p.

Appendix B

Testimony Experience of Lisa A. Bailey, Ph.D.

Lisa A. Bailey, Ph.D.
Last 4 Years of Expert Testimony Experience

Dr. Bailey has provided expert testimony as follows:

1. Steven Halvorsen *vs.* Union Pacific Railroad Company regarding a claim of causal association between occupational exposure to diesel exhaust, benzene, and herbicides and chronic lymphocytic leukemia. For defendant. Deposition on March 12, 2021.
2. Earl Neal *et al*. *vs*. Monsanto Company and Nathaniel Evans *vs*. Monsanto Company regarding claims of causal association between exposure to glyphosate-based herbicides and Non-Hodgkin Lymphoma. For defendant. Deposition on February 18, 2022.
3. Charles E. Adams, *et al*. *vs*. Adient US LLC regarding claims of exposure and health risks from TCE in indoor air and drinking water. For defendant. Deposition on September 10, 2024.
4. Charles A. Boggs *vs* BP Exploration and Production, Inc. and BP America Production Company related to the Deepwater Horizon spill and claims of respiratory health effects from exposure to particulate matter and benzene in ambient air. For defendant. October 2, 2024.

Appendix C

Materials Considered

Appendix D

Plaintiff Risk Calculations

Table D.1 Risk Calculations for the Baseline Daily Drinking Water and Shower Exposures for Terry Dyer

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Lifetime Average Daily Dose (LADD) or Exposure (LADE)[a]		Toxicity Reference Value		Excess Lifetime Cancer Risk[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units		Value	Units		
Central Tendency Exposure (CTE)															
CTE	Hadnot Point	Drinking water	Ingestion	Benzene	1.3E-05	mg/kg-day	2.6E-06	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	1.4E-07	5.5E-02	mg/kg-day	2.1E+04	N
				trans -1,2-Dichloroethylene	3.7E-05	mg/kg-day	7.6E-06	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	2.9E-04	mg/kg-day	6.1E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	3.9E-06	2.1E-01	mg/kg-day	3.5E+03	N
				Vinyl Chloride	1.4E-06	mg/kg-day	2.9E-07	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	2.1E-07	7.1E-02	mg/kg-day	2.5E+05	N
										Total for Hadnot Point Ingestion (CTE):	**4E-06**				
CTE	Tarawa Terrace	Drinking water	Ingestion	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	9.4E-05	mg/kg-day	1.9E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	6.6E-04	mg/kg-day	1.4E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	2.9E-07	5.0E+01	mg/kg-day	3.7E+05	N
				Trichloroethylene	2.7E-05	mg/kg-day	5.6E-06	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	3.6E-07	2.1E-01	mg/kg-day	3.8E+04	N
				Vinyl Chloride	5.3E-05	mg/kg-day	1.1E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	7.9E-06	7.1E-02	mg/kg-day	6.5E+03	N
										Total for Tarawa Terrace Ingestion (CTE):	**9E-06**				
CTE	Tarawa Terrace	Bath water	Dermal	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	1.4E-04	mg/kg-day	2.7E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	4.7E-03	mg/kg-day	9.0E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.9E-06	5.0E+01	mg/kg-day	5.5E+04	N
				Trichloroethylene	5.4E-05	mg/kg-day	1.0E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	6.8E-07	2.1E-01	mg/kg-day	2.0E+04	N
				Vinyl Chloride	4.6E-05	mg/kg-day	8.8E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	6.4E-06	7.1E-02	mg/kg-day	8.0E+03	N
										Total for Tarawa Terrace Dermal (CTE):	**9E-06**				
CTE	Tarawa Terrace	Indoor air	Inhalation	Benzene	NA	$\mu g/m^3$	NA	$\mu g/m^3$	7.8E-06	$(\mu g/m^3)^{-1}$	NA	3.8E+02	$\mu g/m^3$	NA	NA
				trans -1,2-Dichloroethylene	2.7E+00	$\mu g/m^3$	5.2E-01	$\mu g/m^3$	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.7E+01	$\mu g/m^3$	3.3E+00	$\mu g/m^3$	2.6E-07	$(\mu g/m^3)^{-1}$	8.5E-07	4.0E+05	$\mu g/m^3$	1.2E+05	N
				Trichloroethylene	8.7E-01	$\mu g/m^3$	1.7E-01	$\mu g/m^3$	4.1E-06	$(\mu g/m^3)^{-1}$	1.0E-06	2.4E+03	$\mu g/m^3$	1.5E+04	N
				Vinyl Chloride	1.8E+00	$\mu g/m^3$	3.5E-01	$\mu g/m^3$	4.4E-06	$(\mu g/m^3)^{-1}$	1.5E-06	1.3E+04	$\mu g/m^3$	3.6E+04	N
										Total for Tarawa Terrace Inhalation (CTE):	**3E-06**				
Reasonable Maximum Exposure (RME)															
RME	Hadnot Point	Drinking water	Ingestion	Benzene	3.5E-05	mg/kg-day	7.3E-06	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	4.0E-07	5.5E-02	mg/kg-day	7.5E+03	N
				trans -1,2-Dichloroethylene	1.0E-04	mg/kg-day	2.1E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	8.1E-04	mg/kg-day	1.7E-04	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.1E-05	2.1E-01	mg/kg-day	1.3E+03	N
				Vinyl Chloride	3.9E-06	mg/kg-day	8.2E-07	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	5.9E-07	7.1E-02	mg/kg-day	8.7E+04	N
										Total for Hadnot Point Ingestion (RME):	**1E-05**				

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Lifetime Average Daily Dose (LADD) or Exposure (LADE)[a]		Toxicity Reference Value		Excess Lifetime Cancer Risk[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units		Value	Units		
RME	Tarawa Terrace	Drinking water	Ingestion	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans-1,2-Dichloroethylene	2.6E-04	mg/kg-day	5.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.8E-03	mg/kg-day	3.7E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	7.8E-07	5.0E+01	mg/kg-day	1.3E+05	N
				Trichloroethylene	7.4E-05	mg/kg-day	1.5E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.0E-06	2.1E-01	mg/kg-day	1.4E+04	N
				Vinyl Chloride	1.4E-04	mg/kg-day	3.0E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	2.2E-05	7.1E-02	mg/kg-day	2.4E+03	N
										Total for Tarawa Terrace Ingestion (RME):	**2E-05**				
RME	Tarawa Terrace	Bath water	Dermal	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans-1,2-Dichloroethylene	2.2E-04	mg/kg-day	4.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	7.4E-03	mg/kg-day	1.4E-03	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	3.0E-06	5.0E+01	mg/kg-day	3.5E+04	N
				Trichloroethylene	8.5E-05	mg/kg-day	1.6E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.1E-06	2.1E-01	mg/kg-day	1.3E+04	N
				Vinyl Chloride	7.2E-05	mg/kg-day	1.4E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	1.0E-05	7.1E-02	mg/kg-day	5.1E+03	N
										Total for Tarawa Terrace Dermal (RME):	**1E-05**				
RME	Tarawa Terrace	Indoor air	Inhalation	Benzene	NA	$\mu g/m^3$	NA	$\mu g/m^3$	7.8E-06	$(\mu g/m^3)^{-1}$	NA	3.8E+02	$\mu g/m^3$	NA	NA
				trans-1,2-Dichloroethylene	8.1E+00	$\mu g/m^3$	1.6E+00	$\mu g/m^3$	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	5.3E+01	$\mu g/m^3$	1.0E+01	$\mu g/m^3$	2.6E-07	$(\mu g/m^3)^{-1}$	2.7E-06	4.0E+05	$\mu g/m^3$	3.9E+04	N
				Trichloroethylene	2.9E+00	$\mu g/m^3$	5.6E-01	$\mu g/m^3$	4.1E-06	$(\mu g/m^3)^{-1}$	3.4E-06	2.4E+03	$\mu g/m^3$	4.4E+03	N
				Vinyl Chloride	5.7E+00	$\mu g/m^3$	1.1E+00	$\mu g/m^3$	4.4E-06	$(\mu g/m^3)^{-1}$	4.8E-06	1.3E+04	$\mu g/m^3$	1.1E+04	N
										Total for Tarawa Terrace Inhalation (RME):	**1E-05**				
Custom High-End Exposure															
Custom	Hadnot Point	Drinking water	Ingestion	Benzene	3.9E-05	mg/kg-day	8.1E-06	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	4.4E-07	5.5E-02	mg/kg-day	6.8E+03	N
				trans-1,2-Dichloroethylene	1.1E-04	mg/kg-day	2.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	8.9E-04	mg/kg-day	1.8E-04	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.2E-05	2.1E-01	mg/kg-day	1.1E+03	N
				Vinyl Chloride	4.3E-06	mg/kg-day	9.0E-07	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	6.5E-07	7.1E-02	mg/kg-day	7.9E+04	N
										Total for Hadnot Point Ingestion (Custom):	**1E-05**				
Custom	Tarawa Terrace	Drinking water	Ingestion	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans-1,2-Dichloroethylene	2.8E-04	mg/kg-day	5.9E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	2.0E-03	mg/kg-day	4.2E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	8.8E-07	5.0E+01	mg/kg-day	1.2E+05	N
				Trichloroethylene	8.2E-05	mg/kg-day	1.7E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.1E-06	2.1E-01	mg/kg-day	1.2E+04	N
				Vinyl Chloride	1.6E-04	mg/kg-day	3.4E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	2.4E-05	7.1E-02	mg/kg-day	2.1E+03	N
										Total for Tarawa Terrace Ingestion (Custom):	**3E-05**				
Custom	Tarawa Terrace	Bath water	Dermal	Benzene	NA	mg/kg-day	NA	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	NA	5.5E-02	mg/kg-day	NA	NA
				trans-1,2-Dichloroethylene	2.2E-04	mg/kg-day	4.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	7.4E-03	mg/kg-day	1.4E-03	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	3.0E-06	5.0E+01	mg/kg-day	3.5E+04	N
				Trichloroethylene	8.5E-05	mg/kg-day	1.6E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.1E-06	2.1E-01	mg/kg-day	1.3E+04	N
				Vinyl Chloride	7.2E-05	mg/kg-day	1.4E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	1.0E-05	7.1E-02	mg/kg-day	5.1E+03	N
										Total for Tarawa Terrace Dermal (Custom):	**1E-05**				

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Lifetime Average Daily Dose (LADD) or Exposure (LADE)[a]		Toxicity Reference Value		Excess Lifetime Cancer Risk[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units		Value	Units		
Custom	Tarawa Terrace	Indoor air	Inhalation	Benzene	NA	$\mu g/m^3$	NA	$\mu g/m^3$	7.8E-06	$(\mu g/m^3)^{-1}$	NA	3.8E+02	$\mu g/m^3$	NA	NA
				trans -1,2-Dichloroethylene	8.1E+00	$\mu g/m^3$	1.6E+00	$\mu g/m^3$	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	5.3E+01	$\mu g/m^3$	1.0E+01	$\mu g/m^3$	2.6E-07	$(\mu g/m^3)^{-1}$	2.7E-06	4.0E+05	$\mu g/m^3$	3.9E+04	N
				Trichloroethylene	2.9E+00	$\mu g/m^3$	5.6E-01	$\mu g/m^3$	4.1E-06	$(\mu g/m^3)^{-1}$	3.4E-06	2.4E+03	$\mu g/m^3$	4.4E+03	N
				Vinyl Chloride	5.7E+00	$\mu g/m^3$	1.1E+00	$\mu g/m^3$	4.4E-06	$(\mu g/m^3)^{-1}$	4.8E-06	1.3E+04	$\mu g/m^3$	1.1E+04	N
										Total for Tarawa Terrace Inhalation (Custom):	**1E-05**				

Notes:

$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Micrograms per Cubic Meter; mg/kg-day = Milligrams per Kilogram Body Weight per Day; $(mg/kg\text{-}day)^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; N = No; NA = Not Applicable; POD = Point of Departure; Y = Yes.

(a) Lifetime average daily doses (LADDs), lifetime average daily exposures (LADEs), and excess lifetime cancer risks (ELCRs) are calculated using the following equations:

Ingestion and Dermal Contact:

$$LADD = \frac{DED \times EF \times ED}{AT}$$

$$ELCR = LADD \times CSF$$

Inhalation:

$$LADE = \frac{DEC \times EF \times ED}{AT}$$

$$ELCR = LADE \times IUR$$

where:

Variable	Definition	Units	Value	Source/Notes
LADD	Lifetime Average Daily Dose (Oral and Dermal)	mg/kg-day	Chemical specific	Calculated
LADE	Lifetime Average Daily Exposure (Inhalation)	$\mu g/m^3$	Chemical specific	Calculated
DED	Daily Exposure Dose	mg/kg-day	Chemical specific	LaKind (2025)
DEC	Daily Exposure Concentration	$\mu g/m^3$	Chemical specific	LaKind (2025)
EF	Exposure Frequency	days/year	365	Assumes daily exposure
ED	Exposure Duration	years	14.5	Total time spent on-base
AT	Averaging Time	days	25,550	70-year lifetime × 365 days/year
ELCR	Excess Lifetime Cancer Risk	unitless	Chemical specific	Calculated
CSF	Cancer Slope Factor	$(mg/kg\text{-}day)^{-1}$	Chemical specific	Section 5 of report
IUR	Inhalation Unit Risk	$(\mu g/m^3)^{-1}$	Chemical specific	Section 5 of report

As discussed in Section 5.2.1 of the report, the toxicity criteria for trichloroethylene (TCE) are based on multiple cancer endpoints, one of which (kidney cancer) is considered to have a mutagenic mode of action. Therefore, the LADDs and LADEs for TCE are adjusted prior to multiplying by the CSF or IUR using the equations below. Note that the $LADD_{TCE\ adj}$ and $LADE_{TCE\ adj}$ values are not presented in the table above.

Ingestion and Dermal Contact: $LADD_{TCE\ adj} = (LADD \times ADAF \times MAF_o) + (LADD \times CAF_o)$

Inhalation: $LADE_{TCE\ adj} = (LADE \times ADAF \times MAF_i) + (LADE \times CAF_i)$

where:

Variable	Definition	Units	Value	Source/Notes
ADAF	Age-Dependent Adjustment Factor for Mutagenic Compounds (2 to <16 years old)	unitless	3	US EPA (2024)
CAF_o	Carcinogenicity Adjustment Factor for the TCE Cancer Oral Slope Factor	unitless	0.804	US EPA (2024)
CAF_i	Carcinogenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.756	US EPA (2024)
MAF_o	Mutagenicity Adjustment Factor for the TCE Cancer Oral Slope Factor	unitless	0.202	US EPA (2024)
MAF_i	Mutagenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.244	US EPA (2024)

(b) The margins of exposures (MoEs) are calculated by dividing the POD by the LADD or the LADE.

Table D.2 Risk Calculations for the School Shower Exposures for Terry Dyer

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Lifetime Average Daily Dose (LADD) or Exposure (LADE)[a]		Toxicity Reference Value		Excess Lifetime Cancer Risk[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units		Value	Units		
Central Tendency Exposure (CTE)															
CTE	Hadnot Point	Shower water	Dermal	Benzene	3.2E-06	mg/kg-day	1.2E-07	mg/kg-day	5.5E-02	(mg/kg-day)$^{-1}$	6.8E-09	5.5E-02	mg/kg-day	4.5E+05	N
				trans -1,2-Dichloroethylene	1.2E-05	mg/kg-day	4.6E-07	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	(mg/kg-day)$^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	3.5E-05	mg/kg-day	1.3E-06	mg/kg-day	4.6E-02	(mg/kg-day)$^{-1}$	8.7E-08	2.1E-01	mg/kg-day	1.6E+05	N
				Vinyl Chloride	1.5E-07	mg/kg-day	5.8E-09	mg/kg-day	7.2E-01	(mg/kg-day)$^{-1}$	4.2E-09	7.1E-02	mg/kg-day	1.2E+07	N
										Total for Hadnot Point Dermal (CTE):	**1E-07**				
CTE	Hadnot Point	Indoor air	Inhalation	Benzene	9.9E-03	μg/m^3	3.8E-04	μg/m^3	7.8E-06	(μg/m^3)$^{-1}$	2.9E-09	3.8E+02	μg/m^3	1.0E+06	N
				trans -1,2-Dichloroethylene	4.8E-02	μg/m^3	1.8E-03	μg/m^3	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	μg/m^3	NA	μg/m^3	2.6E-07	(μg/m^3)$^{-1}$	NA	4.0E+05	μg/m^3	NA	NA
				Trichloroethylene	1.5E-01	μg/m^3	5.7E-03	μg/m^3	4.1E-06	(μg/m^3)$^{-1}$	3.5E-08	2.4E+03	μg/m^3	4.3E+05	N
				Vinyl Chloride	1.1E-03	μg/m^3	4.2E-05	μg/m^3	4.4E-06	(μg/m^3)$^{-1}$	1.8E-10	1.3E+04	μg/m^3	3.0E+08	N
										Total for Hadnot Point Inhalation (CTE):	**4E-08**				
Reasonable Maximum Exposure (RME)															
RME	Hadnot Point	Shower water	Dermal	Benzene	5.3E-06	mg/kg-day	2.0E-07	mg/kg-day	5.5E-02	(mg/kg-day)$^{-1}$	1.1E-08	5.5E-02	mg/kg-day	2.7E+05	N
				trans -1,2-Dichloroethylene	2.0E-05	mg/kg-day	7.5E-07	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	(mg/kg-day)$^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	5.7E-05	mg/kg-day	2.2E-06	mg/kg-day	4.6E-02	(mg/kg-day)$^{-1}$	1.4E-07	2.1E-01	mg/kg-day	9.7E+04	N
				Vinyl Chloride	2.5E-07	mg/kg-day	9.4E-09	mg/kg-day	7.2E-01	(mg/kg-day)$^{-1}$	6.8E-09	7.1E-02	mg/kg-day	7.5E+06	N
										Total for Hadnot Point Dermal (RME):	**2E-07**				
RME	Hadnot Point	Indoor air	Inhalation	Benzene	3.7E-02	μg/m^3	1.4E-03	μg/m^3	7.8E-06	(μg/m^3)$^{-1}$	1.1E-08	3.8E+02	μg/m^3	2.7E+05	N
				trans -1,2-Dichloroethylene	1.8E-01	μg/m^3	6.8E-03	μg/m^3	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	μg/m^3	NA	μg/m^3	2.6E-07	(μg/m^3)$^{-1}$	NA	4.0E+05	μg/m^3	NA	NA
				Trichloroethylene	5.7E-01	μg/m^3	2.2E-02	μg/m^3	4.1E-06	(μg/m^3)$^{-1}$	1.3E-07	2.4E+03	μg/m^3	1.1E+05	N
				Vinyl Chloride	4.3E-03	μg/m^3	1.6E-04	μg/m^3	4.4E-06	(μg/m^3)$^{-1}$	7.2E-10	1.3E+04	μg/m^3	7.6E+07	N
										Total for Hadnot Point Inhalation (RME):	**1E-07**				
Custom High-End Exposure															
Custom	Hadnot Point	Shower water	Dermal	Benzene	5.3E-06	mg/kg-day	2.0E-07	mg/kg-day	5.5E-02	(mg/kg-day)$^{-1}$	1.1E-08	5.5E-02	mg/kg-day	2.7E+05	N
				trans -1,2-Dichloroethylene	2.0E-05	mg/kg-day	7.5E-07	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	mg/kg-day	NA	mg/kg-day	2.1E-03	(mg/kg-day)$^{-1}$	NA	5.0E+01	mg/kg-day	NA	NA
				Trichloroethylene	5.7E-05	mg/kg-day	2.2E-06	mg/kg-day	4.6E-02	(mg/kg-day)$^{-1}$	1.4E-07	2.1E-01	mg/kg-day	9.7E+04	N
				Vinyl Chloride	2.5E-07	mg/kg-day	9.4E-09	mg/kg-day	7.2E-01	(mg/kg-day)$^{-1}$	6.8E-09	7.1E-02	mg/kg-day	7.5E+06	N
										Total for Hadnot Point Dermal (Custom):	**2E-07**				

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Lifetime Average Daily Dose (LADD) or Exposure (LADE)[a]		Toxicity Reference Value		Excess Lifetime Cancer Risk[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units		Value	Units		
Custom	Hadnot Point	Indoor air	Inhalation	Benzene	3.7E-02	$\mu g/m^3$	1.4E-03	$\mu g/m^3$	7.8E-06	$(\mu g/m^3)^{-1}$	1.1E-08	3.8E+02	$\mu g/m^3$	2.7E+05	N
				trans -1,2-Dichloroethylene	1.8E-01	$\mu g/m^3$	6.8E-03	$\mu g/m^3$	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	NA	$\mu g/m^3$	NA	$\mu g/m^3$	2.6E-07	$(\mu g/m^3)^{-1}$	NA	4.0E+05	$\mu g/m^3$	NA	NA
				Trichloroethylene	5.7E-01	$\mu g/m^3$	2.2E-02	$\mu g/m^3$	4.1E-06	$(\mu g/m^3)^{-1}$	1.3E-07	2.4E+03	$\mu g/m^3$	1.1E+05	N
				Vinyl Chloride	4.3E-03	$\mu g/m^3$	1.6E-04	$\mu g/m^3$	4.4E-06	$(\mu g/m^3)^{-1}$	7.2E-10	1.3E+04	$\mu g/m^3$	7.6E+07	N
									Total for Hadnot Point Inhalation (Custom):		**1E-07**				

Notes:

$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Micrograms per Cubic Meter; mg/kg-day = Milligrams per Kilogram Body Weight per Day; $(mg/kg\text{-}day)^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; N = No; NA = Not Applicable; POD = Point of Departure; Y = Yes.

(a) Lifetime average daily doses (LADDs), lifetime average daily exposures (LADEs), and excess lifetime cancer risks (ELCRs) are calculated using the following equations:

Dermal Contact:

$$LADD = \frac{DED \times EF \times ED}{AT}$$

$$ELCR = LADD \times CSF$$

Inhalation:

$$LADE = \frac{DEC \times EF \times ED}{AT}$$

$$ELCR = LADE \times IUR$$

where:

Variable	Definition	Units	Value	Source/Notes
LADD	Lifetime Average Daily Dose (Dermal)	mg/kg-day	Chemical specific	Calculated
LADE	Lifetime Average Daily Exposure (Inhalation)	$\mu g/m^3$	Chemical specific	Calculated
DED	Daily Exposure Dose	mg/kg-day	Chemical specific	LaKind (2025)
DEC	Daily Exposure Concentration	$\mu g/m^3$	Chemical specific	LaKind (2025)
EF	Exposure Frequency	days/year	221	Approximate number of school days per year
ED	Exposure Duration	years	4.4	Years attending middle and high school on-base
AT	Averaging Time	days	25,550	70-year lifetime × 365 days/year
ELCR	Excess Lifetime Cancer Risk	unitless	Chemical specific	Calculated
CSF	Cancer Slope Factor	$(mg/kg\text{-}day)^{-1}$	Chemical specific	Section 5 of report
IUR	Inhalation Unit Risk	$(\mu g/m^3)^{-1}$	Chemical specific	Section 5 of report

As discussed in Section 5.2.1 of the report, the toxicity criteria for trichloroethylene (TCE) are based on multiple cancer endpoints, one of which (kidney cancer) is considered to have a mutagenic mode of action. Therefore, the LADDs and LADEs for TCE are adjusted prior to multiplying by the CSF or IUR using the equations below. Note that the $LADD_{TCE\ adj}$ and $LADE_{TCE\ adj}$ values are not presented in the table above.

Dermal Contact: $LADD_{TCE\ adj} = (LADD \times ADAF \times MAF_o) + (LADD \times CAF_o)$

Inhalation: $LADE_{TCE\ adj} = (LADE \times ADAF \times MAF_i) + (LADE \times CAF_i)$

where:

Variable	Definition	Units	Value	Source/Notes
ADAF	Age-Dependent Adjustment Factor for Mutagenic Compounds (2 to <16 years old)	unitless	3	US EPA (2024)
CAF_o	Carcinogenicity Adjustment Factor for the TCE Cancer Oral Slope Factor	unitless	0.804	US EPA (2024)
CAF_i	Carcinogenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.756	US EPA (2024)
MAF_o	Mutagenicity Adjustment Factor for the TCE Cancer Oral Slope Factor	unitless	0.202	US EPA (2024)
MAF_i	Mutagenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.244	US EPA (2024)

(b) The margins of exposures (MoEs) are calculated by dividing the POD by the LADD or the LADE.

Table D.3 Risk Calculations for Swimming Exposures for Terry Dyer

Exposure Point	Analyte	Vapor Concentration (VC) in Pool Area ($\mu g/m^3$)	Daily Exposure Concentration (DEC)[a] ($\mu g/m^3$)	Lifetime Average Daily Exposure (LADE)[a] ($\mu g/m^3$)	IUR ($[\mu g/m^3]^{-1}$)	ELCR[a]	POD	MoE[b]	Exposure Exceeds POD? (Y/N)
Hadnot Point	Benzene	207	9	2.2E-01	7.8E-06	1.7E-06	3.8E+02	1.8E+03	N
	trans -1,2-Dichloroethylene	1,000	42	1.0E+00	NA	NA	NA	NA	NA
	Tetrachloroethylene	NA	NA	NA	2.6E-07	NA	4.0E+05	NA	NA
	Trichloroethylene	8,400	350	8.8E+00	4.1E-06	5.4E-05	2.4E+03	2.8E+02	N
	Vinyl Chloride	115	5	1.2E-01	4.4E-06	5.3E-07	1.3E+04	1.0E+05	N
					Total for Swimming Exposures – Hadnot Point:	**6E-05**			
Tarawa Terrace	Benzene	NA	NA	NA	7.8E-06	NA	3.8E+02	NA	NA
	trans -1,2-Dichloroethylene	2,540	106	2.7E+00	NA	NA	NA	NA	NA
	Tetrachloroethylene	34,100	1,421	3.6E+01	2.6E-07	9.3E-06	4.0E+05	1.1E+04	N
	Trichloroethylene	771	32	8.1E-01	4.1E-06	4.9E-06	2.4E+03	3.0E+03	N
	Vinyl Chloride	4,240	177	4.4E+00	4.4E-06	2.0E-05	1.3E+04	2.8E+03	N
					Total for Swimming Exposures – Tarawa Terrace:	**3E-05**			

Notes:

$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Micrograms per Cubic Meter; IUR = Inhalation Unit Risk; N = No; NA = Not Applicable; POD = Point of Departure; Y = Yes.

(a) Daily exposure concentrations (DECs), lifetime average daily exposures (LADEs), and excess lifetime cancer risks (ELCRs) are calculated using the following equations:

$$DEC = \frac{VC \times ET}{24 \text{ hours/day}}$$

$$LADE = \frac{DEC \times EF \times EV}{AT}$$

$$ELCR = LADE \times IUR$$

where:

Variable	Definition	Units	Value	Source/Notes
VC	Vapor Concentration in Pool Area	$\mu g/m^3$	Chemical specific	LaKind (2025)
DEC	Daily Exposure Concentration	$\mu g/m^3$	Chemical specific	Calculated
LADE	Lifetime Average Daily Exposure	$\mu g/m^3$	Chemical specific	Calculated
ET	Exposure Time	hours/day	1	Professional judgment
EF	Exposure Frequency	days/event	1	Professional judgment
EV	Events During Exposure Duration	number of events	642	Professional judgment
AT	Averaging Time	days	25,550	70-year lifetime × 365 days/year
ELCR	Excess Lifetime Cancer Risk	unitless	Chemical specific	Calculated
IUR	Inhalation Unit Risk	$(\mu g/m^3)^{-1}$	Chemical specific	Section 5 of report

As discussed in Section 5.2.1 of the report, the toxicity criteria for trichloroethylene (TCE) are based on multiple cancer endpoints, one of which (kidney cancer) is considered to have a mutagenic mode of action. Therefore, the LADEs for TCE are adjusted prior to multiplying by the IUR using the equation below. Note that the $LADE_{TCE\ adj}$ values are not presented in the table above.

$$LADE_{TCE\ adj} = (LADE \times ADAF \times MAF_i) + (LADE \times CAF_i)$$

where:

Variable	Definition	Units	Value	Source/Notes
ADAF	Age-Dependent Adjustment Factor for Mutagenic Compounds (2 to <16 years old)	unitless	3	US EPA (2024)
CAF_i	Carcinogenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.756	US EPA (2024)
MAF_i	Mutagenicity Adjustment Factor for the TCE Inhalation Unit Risk Value	unitless	0.244	US EPA (2024)

(b) The margins of exposures (MoEs) are calculated by dividing the POD by the LADE.

Table D.4 Summary of Risks by Exposure Pathway (Baseline + Additional) for Terry Dyer

Exposure Scenario	Exposure Point	Analyte	Baseline Exposure Pathways: Ingestion (Drinking Water) ELCR	%	Dermal (Shower) ELCR	%	Inhalation (Indoor Air) ELCR	%	Additional Pathways: Dermal (School Shower) ELCR	%	Inhalation (School Shower Air) ELCR	%	Inhalation (Swimming Pool) ELCR	%	Total ELCR
Central Tendency Exposure (CTE)															
CTE	Hadnot Point: Drinking Water,	Benzene	1.4E-07	3%	NA	–	NA	–	6.8E-09	7%	2.9E-09	8%	1.7E-06	3%	1.8E-06
	School Shower Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	NA	–	1.9E-06	21%	8.5E-07	25%	NA	–	NA	–	NA	–	2.7E-06
		Trichloroethylene	3.9E-06	92%	6.8E-07	8%	1.0E-06	30%	8.7E-08	89%	3.5E-08	92%	5.4E-05	96%	5.9E-05
	Tarawa Terrace: Home Bath	Vinyl Chloride	2.1E-07	5%	6.4E-06	71%	1.5E-06	45%	4.2E-09	4%	1.8E-10	0.5%	5.3E-07	0.9%	8.6E-06
	Exposures	**Pathway-Specific Total:**	**4E-06**		**9E-06**		**3E-06**		**1E-07**		**4E-08**		**6E-05**		**7E-05**
CTE	Tarawa Terrace: Drinking Water,	Benzene	NA	–	NA	–	NA	–	6.8E-09	7%	2.9E-09	8%	NA	–	9.7E-09
	Home Bath Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	2.9E-07	3%	1.9E-06	21%	8.5E-07	25%	NA	–	NA	–	9.3E-06	28%	1.2E-05
		Trichloroethylene	3.6E-07	4%	6.8E-07	8%	1.0E-06	30%	8.7E-08	89%	3.5E-08	92%	4.9E-06	15%	7.1E-06
	Hadnot Point: School Shower	Vinyl Chloride	7.9E-06	92%	6.4E-06	71%	1.5E-06	45%	4.2E-09	4%	1.8E-10	0.5%	2.0E-05	58%	3.5E-05
	Exposures	**Pathway-Specific Total:**	**9E-06**		**9E-06**		**3E-06**		**1E-07**		**4E-08**		**3E-05**		**5E-05**
		Exposure Point with Highest Pathway-Specific Risk:	***9E-06***		***9E-06***		***3E-06***		***1E-07***		***4E-08***		***6E-05***		***8E-05***
Reasonable Maximum Exposure (RME)															
RME	Hadnot Point: Drinking Water,	Benzene	4.0E-07	3%	NA	–	NA	–	1.1E-08	7%	1.1E-08	8%	1.7E-06	3%	2.1E-06
	School Shower Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	NA	–	3.0E-06	21%	2.7E-06	24%	NA	–	NA	–	NA	–	5.7E-06
		Trichloroethylene	1.1E-05	92%	1.1E-06	8%	3.4E-06	31%	1.4E-07	89%	1.3E-07	92%	5.4E-05	96%	6.9E-05
	Tarawa Terrace: Home Bath	Vinyl Chloride	5.9E-07	5%	1.0E-05	71%	4.8E-06	44%	6.8E-09	4%	7.2E-10	0.5%	5.3E-07	0.9%	1.6E-05
	Exposures	**Pathway-Specific Total:**	**1E-05**		**1E-05**		**1E-05**		**2E-07**		**1E-07**		**6E-05**		**9E-05**
RME	Tarawa Terrace: Drinking Water,	Benzene	NA	–	NA	–	NA	–	1.1E-08	7%	1.1E-08	8%	NA	–	2.2E-08
	Home Bath Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	7.8E-07	3%	3.0E-06	21%	2.7E-06	24%	NA	–	NA	–	9.3E-06	28%	1.6E-05
		Trichloroethylene	1.0E-06	4%	1.1E-06	8%	3.4E-06	31%	1.4E-07	89%	1.3E-07	92%	4.9E-06	15%	1.1E-05
	Hadnot Point: School Shower	Vinyl Chloride	2.2E-05	92%	1.0E-05	71%	4.8E-06	44%	6.8E-09	4%	7.2E-10	0.5%	2.0E-05	58%	5.6E-05
	Exposures	**Pathway-Specific Total:**	**2E-05**		**1E-05**		**1E-05**		**2E-07**		**1E-07**		**3E-05**		**8E-05**
		Exposure Point with Highest Pathway-Specific Risk:	***2E-05***		***1E-05***		***1E-05***		***2E-07***		***1E-07***		***6E-05***		***1E-04***
Custom High-End Exposure															
Custom	Hadnot Point: Drinking Water,	Benzene	4.4E-07	3%	NA	–	NA	–	1.1E-08	7%	1.1E-08	8%	1.7E-06	3%	2.2E-06
	School Shower Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	NA	–	3.0E-06	21%	2.7E-06	24%	NA	–	NA	–	NA	–	5.7E-06
		Trichloroethylene	1.2E-05	92%	1.1E-06	8%	3.4E-06	31%	1.4E-07	89%	1.3E-07	92%	5.4E-05	96%	7.0E-05
	Tarawa Terrace: Home Bath	Vinyl Chloride	6.5E-07	5%	1.0E-05	71%	4.8E-06	44%	6.8E-09	4%	7.2E-10	0.5%	5.3E-07	0.9%	1.6E-05
	Exposures	**Pathway-Specific Total:**	**1E-05**		**1E-05**		**1E-05**		**2E-07**		**1E-07**		**6E-05**		**9E-05**
Custom	Tarawa Terrace: Drinking Water,	Benzene	NA	–	NA	–	NA	–	1.1E-08	7%	1.1E-08	8%	NA	–	2.2E-08
	Home Bath Exposures, and	*trans* -1,2-Dichloroethylene	NA	–	NA	–	NA	–	NA	–	NA	–	NA	–	NA
	Swimming	Tetrachloroethylene	8.8E-07	3%	3.0E-06	21%	2.7E-06	24%	NA	–	NA	–	9.3E-06	28%	1.6E-05
		Trichloroethylene	1.1E-06	4%	1.1E-06	8%	3.4E-06	31%	1.4E-07	89%	1.3E-07	92%	4.9E-06	15%	1.1E-05
	Hadnot Point: School Shower	Vinyl Chloride	2.4E-05	92%	1.0E-05	71%	4.8E-06	44%	6.8E-09	4%	7.2E-10	0.5%	2.0E-05	58%	5.9E-05
	Exposures	**Pathway-Specific Total:**	**3E-05**		**1E-05**		**1E-05**		**2E-07**		**1E-07**		**3E-05**		**9E-05**
		Exposure Point with Highest Pathway-Specific Risk:	***3E-05***		***1E-05***		***1E-05***		***2E-07***		***1E-07***		***6E-05***		***1E-04***

Notes:

ELCR = Excess Lifetime Cancer Risk; NA = Not Applicable.

Appendix E

Point of Departure (POD) Derivations for Trichloroethylene (TCE) and Vinyl Chloride

Because the United States Environmental Protection Agency (US EPA) does not present points of departure (PODs) for trichloroethylene (TCE) or vinyl chloride for several endpoints and exposure pathways for which toxicity criteria are available, as described in this appendix, I have estimated the PODs based on the oral and inhalation toxicity criteria for these chemicals and pathways. These PODs are used in the margin of exposure (MoE) calculations discussed in Section 7.

E.1 Trichloroethylene (TCE)

PODs can be estimated from cancer toxicity criteria based on the fact that the cancer slope factor (CSF) or inhalation unit risk (IUR) values are expressed in terms of a specific risk per milligrams per kilogram body weight per day (*i.e.*, $[\text{mg/kg-day}]^{-1}$) or per micrograms per cubic meter (*i.e.*, $[\mu\text{g/m}^3]^{-1}$), respectively.

For TCE, the PODs that US EPA provides for some of the CSFs and IURs, and that are used to extrapolate to other cancer toxicity criteria, are based on a cancer risk of 1% (*i.e.*, the lower confidence limit of the exposure dose or concentration at an extra risk level of 1% [LED_{01} or LEC_{01}]) (US EPA, 2011a). Assuming that the TCE CSFs and IURs for which PODs were not provided would also be equivalent to a 1% cancer risk, the following equations can be used to calculate PODs from those CSFs and IURs.

To estimate PODs (LED_{01} values) from CSFs:

$$\text{POD (mg/kg-day)} = 1\% \div \text{CSF}\left([\text{mg/kg-day}]^{-1}\right)$$

To estimate PODs (LEC_{01} values) from IURs:

$$\text{POD}\left(\mu\text{g/m}^3\right) = 1\% \div \text{IUR}\left([\mu\text{g/m}^3]^{-1}\right)$$

Tables E.1 and E.2 summarize the PODs for TCE.

Table E.1 US EPA TCE Oral Cancer Toxicity Values (Cancer Slope Factors [CSFs]) and Points of Departure (PODs)

Chemical	Oral CSF[a] ($[\text{mg/kg-day}]^{-1}$)	POD[a] (mg/kg-day)	Cancer Type	Sources
TCE	4.6×10^{-2}	$LED_{01} = 0.21$	Renal cell carcinoma, NHL, and liver cancer	US EPA (2011a,b)
	9.33×10^{-3}	$LED_{01} = 1.07^{b}$	Renal cell carcinoma	
	2.16×10^{-2}	$LED_{01} = 0.46^{b}$	NHL	
	1.55×10^{-2}	$LED_{01} = 0.65^{b}$	Liver cancer	

Notes:

LED_{01} = Lower Confidence Limit of the Exposure Dose at an Extra Risk Level of 1%; mg/kg-day = Milligrams per Kilogram Body Weight per Day; $(\text{mg/kg-day})^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; NHL = Non-Hodgkin's Lymphoma; TCE = Trichloroethylene; US EPA = United States Environmental Protection Agency.

(a) US EPA (2011b) calculated the oral CSFs for renal cell carcinoma, NHL, and liver cancer individually and the LED_{01} for the three cancers combined as described in Section 5 and Table 5.1.

(b) PODs (LED_{01} values) for renal cell carcinoma, NHL, and liver cancer are calculated based on the equation described above. For example, for the renal cell carcinoma LED_{01}, the calculation is as follows: $1\% \div 0.00933\ (\text{mg/kg-day})^{-1} = 1.07$ mg/kg-day.

Table E.2 US EPA TCE Inhalation Cancer Toxicity Values (Inhalation Unit Risks [IURs]) and Points of Departure (PODs)

Chemical	IUR[a] ($[\mu g/m^3]^{-1}$; $[ppm]^{-1}$)	POD[a] ($\mu g/m^3$ [ppb])	Cancer Type	Sources
TCE	4.1×10^{-6} $(\mu g/m^3)^{-1}$; 2.2×10^{-2} $(ppm)^{-1}$	LEC_{01} = 2,445 (455)	Renal cell carcinoma, NHL, and liver cancer	Charbotel *et al.* (2006); Raaschou-Nielsen *et al.* (2003); US EPA (2011a,b)
	1.0×10^{-6} $(\mu g/m^3)^{-1}$; 5.5×10^{-3} $(ppm)^{-1}$	LEC_{01} = 9,781 (1,820)	Renal cell carcinoma	
	2.0×10^{-6} $(\mu g/m^3)^{-1}$ 1.1×10^{-2} $(ppm)^{-1}$	LEC_{01} = 4,890[b] (910)	NHL	
	1.0×10^{-6} $(\mu g/m^3)^{-1}$; 5.5×10^{-3} $(ppm)^{-1}$	LEC_{01} = 9,781[b] (1,820)	Liver cancer	

Notes:

$\mu g/m^3$ = Microgram per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Microgram per Cubic Meter; LEC_{01} = Lower Confidence Limit of the Exposure Concentration at an Extra Risk Level of 1%; ppb = Parts per Billion; ppm = Parts per Million; $(ppm)^{-1}$ = Per Parts per Million; NHL = Non-Hodgkin's Lymphoma; TCE = Trichloroethylene; US EPA = United States Environmental Protection Agency.

(a) US EPA (2011b) calculated the individual IURs and the LEC_{01} values for renal cell carcinoma, NHL, and liver cancer combined and renal cell carcinoma individually as described in Section 5 and Table 5.2.

(b) PODs (LEC_{01} values) for NHL and liver cancer are calculated based on the equation described above. For example, for the NHL LEC_{01}, the calculation is as follows: $1\% \div 0.000002\ (\mu g/m^3)^{-1} = 4{,}890\ \mu g/m^3$. Note that rounding the IURs changes the PODs slightly.

E.2 Vinyl Chloride

Similar calculations were conducted for vinyl chloride. As described by US EPA (2003), one set of oral and inhalation cancer toxicity criteria for vinyl chloride (that are essentially identical to the other set of toxicity criteria calculated by the agency) are based on a cancer risk of 10% (*i.e.*, the lower confidence limit of the exposure dose or concentration at an extra risk level of 10% [LED_{10} or LEC_{10}]). Therefore, the following equations can be used to calculate PODs from these CSFs and IURs.

To estimate PODs (LED_{10} values) from CSFs:

$$\text{POD (mg/kg-day)} = 10\% \div \text{CSF}\left([\text{mg/kg-day}]^{-1}\right)$$

To estimate PODs (LEC_{10} values) from IURs:

$$\text{POD}\left(\mu g/m^3\right) = 10\% \div \text{IUR}\left([\mu g/m^3]^{-1}\right)$$

Tables E.3 and E.4 summarize the PODs for vinyl chloride.

Table E.3 US EPA Vinyl Chloride Oral Cancer Toxicity Values (Cancer Slope Factors [CSFs]) and Points of Departure (PODs)

Chemical	Oral CSF[a] ([mg/kg-day]$^{-1}$)	POD[b] (mg/kg-day)	Cancer Type (Sex/Species)	Sources
Vinyl Chloride	**Continuous Lifetime Exposure During Adulthood**			
	7.2×10^{-1}; 7.5×10^{-1}	LED_{10} = 0.133	Liver angiosarcomas, hepatocellular carcinomas, and neoplastic liver nodules (female rat)	Feron *et al.* (1981); US EPA (2000, 2003)
	Continuous Lifetime Exposure from Birth			
	1.4; 1.5	LED_{10} = 0.067	Liver angiosarcomas, hepatocellular carcinomas, and neoplastic liver nodules (female rat)	Feron *et al.* (1981); US EPA (2000, 2003)

Notes:

LED_{10} = Lower Confidence Limit of the Exposure Dose at an Extra Risk Level of 10%; mg/kg-day = Milligrams per Kilogram Body Weight per Day; $(mg/kg\text{-}day)^{-1}$ = Per Milligrams per Kilogram Body Weight per Day; US EPA = United States Environmental Protection Agency.

(a) US EPA (2003) calculated the individual oral CSFs as described in Section 5 and Table 5.10.

(b) PODs (LED_{10} values) are calculated based on the equation described above. For example, for the continuous lifetime exposure during adulthood LED_{10}, the calculation is as follows: 10% ÷ 0.75 $(mg/kg\text{-}day)^{-1}$ = 0.133 mg/kg-day.

Table E.4 US EPA Vinyl Chloride Inhalation Cancer Toxicity Values (Inhalation Unit Risks [IURs]) and Points of Departure (PODs)

Chemical	IUR[a] ([μg/m^3]$^{-1}$)	POD[b] (μg/m^3 [ppb])	Cancer Type (Sex/Species)	Sources
Vinyl Chloride	**Continuous Lifetime Exposure During Adulthood**			
	4.4×10^{-6}	LEC_{10} = 22,727 (8,900)	Liver angiosarcomas, angiomas, hepatomas, and neoplastic liver nodules (female rat)	Popper *et al.* (1981); Maltoni *et al.* (1984); US EPA (2000, 2003)
	Continuous Lifetime Exposure from Birth			
	8.8×10^{-6}	LEC_{10} = 11,364 (4,445)	Liver angiosarcomas, angiomas, hepatomas, and neoplastic liver nodules (female rat)	Popper *et al.* (1981); Maltoni *et al.* (1984); US EPA (2000, 2003)

Notes:

μg/m^3 = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = Per Microgram per Cubic Meter; LEC_{10} = Lower Confidence Limit of the Exposure Concentration at an Extra Risk Level of 10%; ppb = Parts per Billion; US EPA = United States Environmental Protection Agency.

(a) US EPA (2003) calculated the individual IURs as described in Section 5 and Table 5.11.

(b) PODs (LEC_{10} values) are calculated based on the equation described above. For example, for the continuous lifetime exposure during adulthood LEC_{10}, the calculation is as follows: 10% ÷ 0.0000044 $(\mu g/m^3)^{-1}$ = 22,727 μg/m^3.

References

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(8):777-787. doi: 10.1093/annhyg/mel039.

Feron, VJ; Hendriksen, CFM; Speek, AJ; Til, HP; Spit, BJ. 1981. "Lifespan oral toxicity study of vinyl chloride in rats." *Food Cosmet. Toxicol.* 19:317-333.

Maltoni, C; Lefemine, G; Ciliberti, A; eds. 1984. *Experimental Research on Vinyl Chloride Carcinogenesis. Part I: Review of Available Data on General Pathology and Carcinogenicity*. Princeton Archives of Research on Industrial Carcinogenesis: Volume 2. Scientific Publishing, Co., Princeton, NJ. p1-35.

Popper, H; Maltoni, C; Selikoff, IJ. 1981. "Vinyl chloride-induced hepatic lesions in man and rodents. A comparison." *Liver* 1:7-20.

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(12):1182-1192.

US EPA. 2000. "Toxicological Review of Vinyl Chloride (CAS No. 75-01-4)." EPA/635/R-00/004. 197p., May.

US EPA. 2003. "IRIS Chemical Assessment Summary for Vinyl Chloride (CAS No. 75-01-4)." 63p., October 28. Accessed at https://www.epa.gov/iris.

US EPA. 2011a. "IRIS Chemical Assessment Summary for Trichloroethylene (CAS No. 79-01-6)." 65p., September 28. Accessed at https://www.epa.gov/iris.

US EPA. 2011b. "Toxicological Review of Trichloroethylene (CAS No. 79-01-6) in Support of Summary Information on the Integrated Risk Information System (IRIS) (Final)." EPA/635/R-09/011F, 2469p., September.