Exhibit 469

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

In the Case of:
Vivian Connard (as Representative of Stephen Connard) *v.* United States

Prepared by

Lisa A. Bailey, Ph.D.

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

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

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Abbreviations

µg/m^3	Micrograms per Cubic Meter of Air
(µg/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
ALL	Acute Lymphocytic Leukemia
AML	Acute Myeloid Leukemia
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BMDL	Lower Confidence Limit on the Benchmark Dose
CLL	Chronic Lymphocytic Leukemia
CML	Chronic Myeloid Leukemia
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-day)$^{-1}$	Per Milligrams per Kilogram Body Weight per Day
NHL	Non-Hodgkin's Lymphoma
NRC	National Research Council
NTP	National Toxicology Program
PBPK	Physiologically Based Pharmacokinetic
PCE	Tetrachloroethylene/Perchloroethylene
POD	Point of Departure
ppb	Parts per Billion
ppm	Parts per Million
RAGS	Risk Assessment Guidance for Superfund
RME	Reasonable Maximum Exposure
SD	Standard Deviation
SDWA	Safe Drinking Water Act

TCE	Trichloroethylene
TT	Tarawa Terrace
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 *Vivian Connard (as representative of Stephen Connard) 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 leukemia (acute myeloid leukemia [AML]) 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 $[mg/kg\text{-}day]^{-1}$). 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 $(mg/kg\text{-}day)^{-1}$ 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 ($\mu g/m^3$)

    DEC = Daily Exposure Concentration ($\mu g/m^3$)

    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, 2024). 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 ($[mg/kg\text{-}day]^{-1}$)
- 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 $(mg/kg\text{-}day)^{-1}$ to an LADD of 0.005 mg/kg-day would result in the following risk calculation: 0.005 mg/kg-day × 0.01 $(mg/kg\text{-}day)^{-1}$ = 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 benzene exposure and all types of leukemia (including acute myeloid leukemia [AML], acute lymphocytic leukemia [ALL], chronic myeloid leukemia [CML], and chronic lymphocytic leukemia [CLL]), "equipoise and above evidence for causation" for all types of leukemia for TCE, and "below equipoise evidence for causation" for exposure to PCE or vinyl chloride and leukemia. ATSDR (2017b) provided no comment on whether there is a causal association between 1,2-tDCE exposure and leukemia. Overall, US EPA, the International Agency for Research on Cancer (IARC), and the National Toxicology Program (NTP) have concluded that benzene can cause leukemia in humans at some doses, based predominantly on observations of associations with AML, with limited evidence of associations with ALL and CLL, but these agencies do not conclude that there is a known association between exposure to PCE, TCE, vinyl chloride, or 1,2-tDCE and leukemia (see Section 5). Dr. Goodman (2025) concluded that, while the scientific evidence supports a causal

[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 that 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).

association between benzene exposures and AML at exposures ≥40-75 ppm-years, the epidemiology studies do not provide consistent or compelling evidence that benzene exposure is associated with ALL, CLL, or CML. Dr. Goodman also concluded that, overall, the scientific evidence does not support a causal association between TCE, PCE, vinyl chloride, or 1,2-tDCE and leukemia.

Section 5 also summarizes the US EPA toxicity criteria used in the cancer risk evaluation for the plaintiff. The benzene toxicity criteria are based on leukemia as the most sensitive endpoint. ATSDR (2017b) concludes for TCE that there is "equipoise and above evidence for causation for all types of leukemia." Because the scientific evidence does not support, or only provides weak support for, an association between leukemia and exposure to TCE, PCE, and vinyl chloride,[2] the cancer toxicity criteria for these chemicals are also not based on leukemia. Therefore, cancer risk calculations for TCE, PCE, and vinyl chloride are not predictive and are overly conservative of leukemia 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 Mr. Connard. Mr. Connard was stationed at Camp Lejeune from October 1977 to February 1979 and again from March 1980 to July 1981. While he was on-base, Mr. Connard either lived or worked at Mainside Barracks, Hadnot Point, or Hospital Point, which were all serviced by the Hadnot Point [HP] water system. For my risk calculations, I used TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE exposure estimates for Mr. Connard 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 (Hadnot Point [HP] and Tarawa Terrace [TT]). I combined this information with the regulatory toxicity criteria summarized in Section 5, to conduct a conservative regulatory risk evaluation for Mr. Connard. Risks were calculated for the following exposure pathways and scenarios for the exposure period of concern (approximately 3 years) for Mr. Connard:

- **Baseline Exposure Pathways:**
  - Drinking Water Ingestion – For this exposure pathway, it is not clear that the plaintiff's water ingestion occurred from only one of the two WTPs, I evaluated three scenarios for both the HP and TT WTPs: (1) central tendency exposure (CTE), which assumes ingestion of 1.3 liter (L) of tap water per day; (2) reasonable maximum exposure (RME), which assumes ingestion of 3.3 L of tap water per day; and (3) military high-end exposure, which assumes ingestion of 6 L of tap water per day.
  - Dermal and Inhalation Exposures from Showering – For these exposure pathways, I calculated risks based on the CTE (50th percentile) and RME (95th percentile) dermal dose and inhalation concentration outputs from a communal showering facility exposure model (ATSDR, 2024a), and based on Mr. Connard's location of residence during his time at Camp LeJeune (HP WTP). The inhalation concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Exposures from the communal shower facility model are estimated based on a mean daily shower duration of 20 minutes, and a range of shower durations that includes 30 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.
- **Exposure Scenarios Evaluated for Mr. Connard:**

[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.

- The CTE exposure scenario, which includes the following exposure pathways: CTE drinking water ingestion (TT and HP WTPs), and CTE dermal and inhalation exposures from showering (HP WTP).
- The RME exposure scenario, which includes the following exposure pathways: RME drinking water ingestion (TT and HP WTPs), and RME dermal and inhalation exposures from showering (HP WTP).
- The military high-end exposure scenario, which includes the following exposure pathways: military high-end water ingestion (TT and HP WTPs), and RME dermal and inhalation exposures from showering (HP WTP).

Based on standard risk assessment methodology, which includes overly health-protective assumptions about exposure and risk, the maximum risk estimate calculated for Mr. Connard's estimated exposures (1×10^{-4}, or 1 cancer case 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%). However, the cancer risk estimate is for all cancer types for all chemical exposures evaluated and is driven predominantly by TCE and vinyl chloride in drinking water (see Appendix D, Table D.2). As discussed above and in Section 5, none of the regulatory agencies concluded that vinyl chloride exposure is a known cause of leukemia in humans. US EPA's cancer toxicity criteria are based on liver cancer for vinyl chloride. In addition, the TCE cancer toxicity values are based on NHL, kidney cancer, and liver cancer combined, and are summed across all of those endpoints. Therefore, the cancer risk estimates for TCE and vinyl chloride are overly conservative estimates of risk for leukemia (AML) for Mr. Connard and should not be interpreted to suggest there is an excess AML risk of 1×10^{-4}. As shown in Appendix D, the maximum estimated cancer risk for benzene (the only chemical toxicity value that is based on leukemia) is 1×10^{-6} (1 leukemia case in 1,000,000 exposed people, or 0.0001% increased risk of leukemia) from the military high-end exposure scenario at HP. This risk estimate is equal to the lower end of US EPA's acceptable target risk range, providing support that Mr. Connard's exposures would not have been expected to lead to his leukemia (AML).

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 Mr. Connard's exposures, the MoEs range from 190 to 22,000,000, all of which are well above 1, providing additional support that Mr. Connard's exposures would not have been expected to lead to his leukemia (AML).

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

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 Mr. Connard's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 3 years that he was stationed at the Camp Lejeune are causally associated with his leukemia (AML).

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 Dr. Hennet (2024) and Spiliotopoulos (2024) regarding groundwater modeling for Camp Lejeune;
  - Expert reports submitted on behalf of Mr. Connard by Drs. Reynolds (2025a) and Gondek (2025);
  - Plaintiff's (or plaintiff's representative's) deposition; and
  - Other plaintiff materials, if available, as cited within (*e.g.*, declaration, military and/or employment 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 (or plaintiff's representative's) deposition and other relevant plaintiff materials;
- 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 leukemia 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 leukemia, 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%.

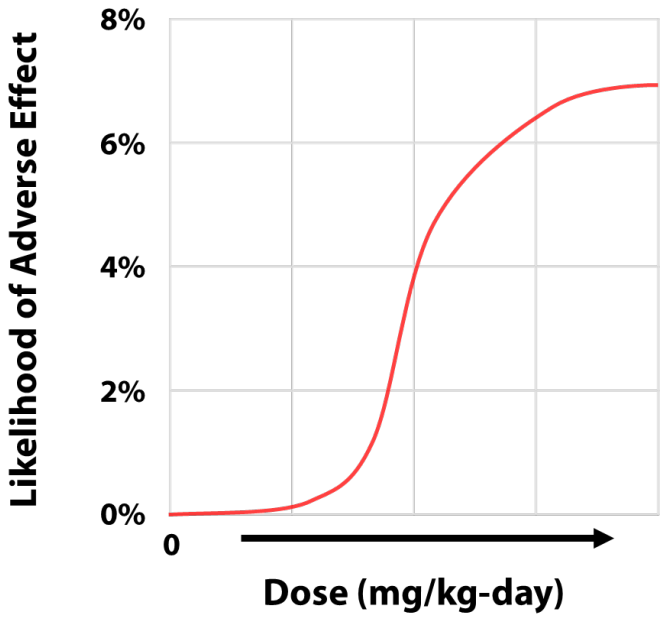

Figure 3.1 Dose-Response Curve

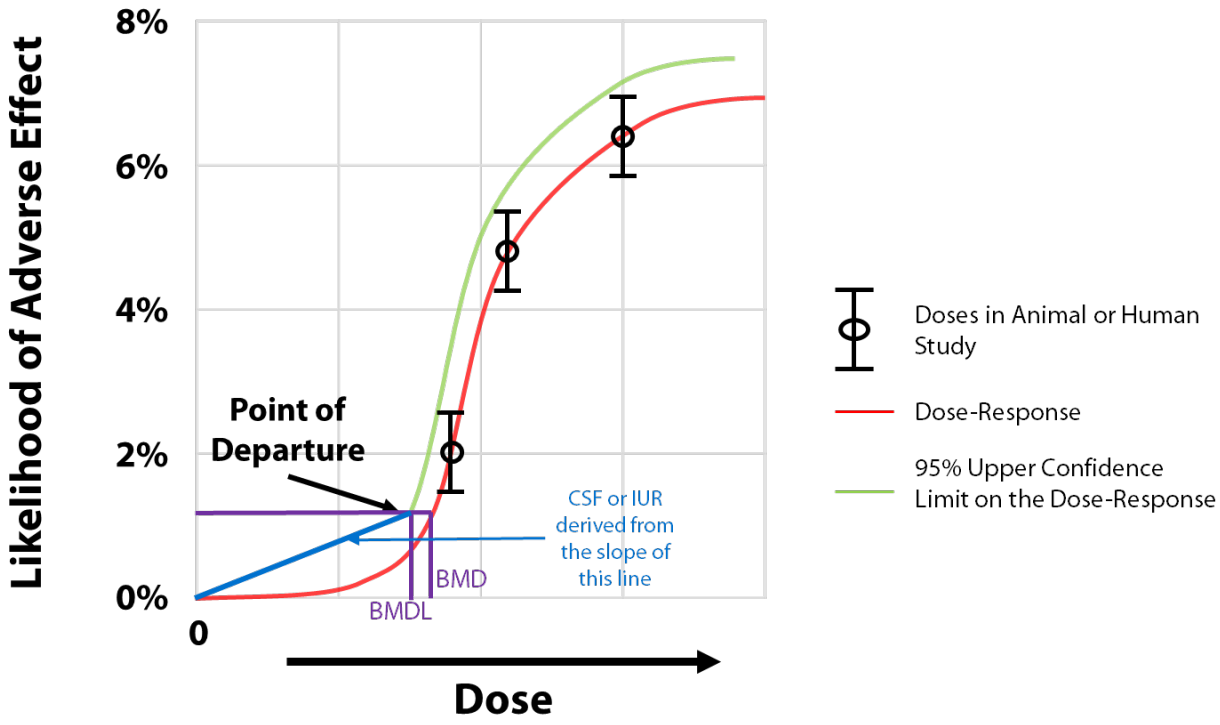

Figure 3.2 Approach for Cancer Slope Factor (CSF) or Inhalation Unit Risk (IUR) Development.
BMD = Benchmark Dose; BMDL = Lower Confidence Limit on the Benchmark Dose.

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.*, $[mg/kg\text{-}day]^{-1}$). For example, a CSF of 0.01 $(mg/kg\text{-}day)^{-1}$ 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:

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

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:

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

where:

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

See Appendix D for more details on these calculations.

3.3.3 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, 2024). As described by ACS (2024), the lifetime probability of developing leukemia is 1.9% for men and 1.3% 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, 2024; 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 leukemia) 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.

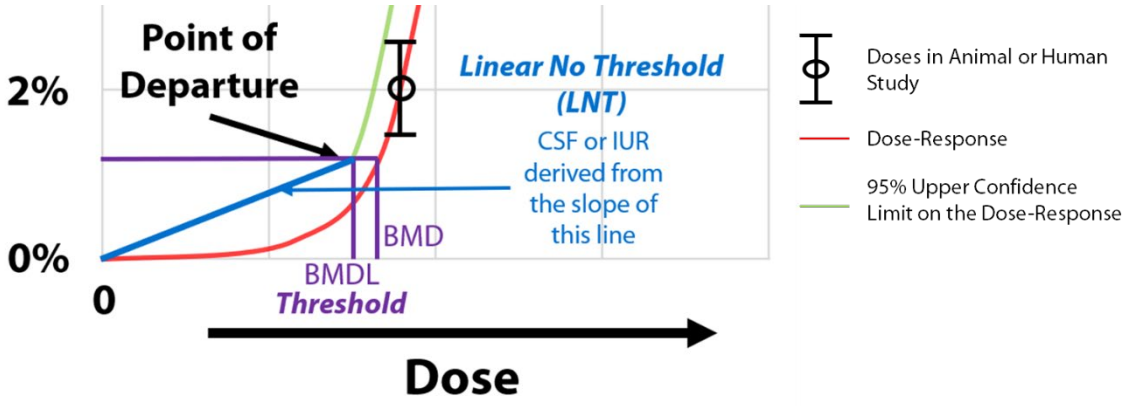

Figure 3.3 Linear No-Threshold (LNT) *vs*. Threshold Models. CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk.

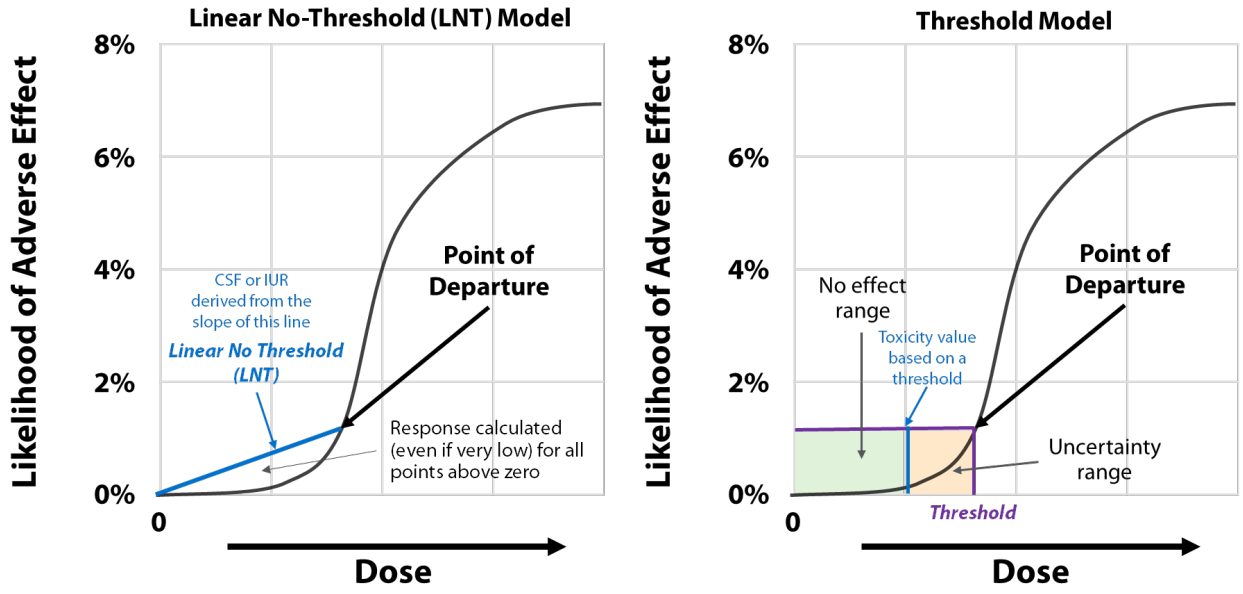

Figure 3.4 Linear No-Threshold (LNT) *vs*. Threshold Model Extrapolations from the Point of Departure (POD)

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 New River, Onslow Beach, Courthouse Bay, Camp Geiger, Rifle Range, and Montford Point/Camp Johnson – were not contaminated with solvents (Hennet, 2024). 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 leukemia. 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 leukemia, 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), IARC (2014), ATSDR (2019a), and NTP (2015) do not conclude that TCE exposure is a known cause of leukemia in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, and somewhat inconsistent with its more recent toxicological profile for TCE (ATSDR, 2019a), ATSDR (2017b) concluded that there was "equipoise and above evidence for causation for all types of leukemia, including AML, ALL, CML and CLL" for TCE exposure. ATSDR (2017b) stated that its conclusion was based on "not strong but nevertheless sufficient" evidence from occupational and human drinking water studies, and from animal and human evidence of "immune disorders that have been linked to leukemia."

Based on the available epidemiology studies and agency reviews that evaluated TCE exposure and leukemia, Dr. Goodman concluded, "Given the lack of a consistent association between TCE and leukemia (overall), AML, or CML and the high likelihood of exposure measurement error across studies, I conclude that the epidemiology evidence does not support a causal association between TCE exposure and leukemia" (Goodman, 2025). Dr. Goodman also concluded that the animal studies "[do] not provide compelling evidence that TCE can cause leukemia" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that have evaluated potential associations between TCE exposure and leukemia concluded that, overall, the scientific evidence "does not support a causal association between TCE exposure and leukemia" (Goodman, 2025).

5.1.2 Tetrachloroethylene (PCE)

To understand the potential association between PCE exposure and leukemia, 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) and IARC (2014); none of the agency documents concluded that PCE exposure is a known cause of leukemia in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that the evidence for causation is "below equipoise" for exposure to PCE and leukemia (including CLL and AML) based on limited and inconsistent epidemiology evidence and uncertainties regarding the relevance of the finding of mononuclear-cell leukemia in rats.

Based on the available epidemiology studies and agency reviews that evaluated PCE exposure and leukemia, Dr. Goodman concluded, "Given that most analyses do not provide evidence of associations between PCE exposure and leukemia or leukemia subtypes, and the methodological limitations of these epidemiology studies, particularly the high likelihood of exposure measurement error, I conclude that the epidemiology evidence does not support a causal association between PCE exposure and leukemia, ALL, AML, or CML" (Goodman, 2025). Dr. Goodman also concluded that, "There is no animal evidence indicating PCE can cause leukemia in humans" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between PCE exposure and leukemia concluded that, overall, the scientific evidence "does not support a causal association between PCE and leukemia" (Goodman, 2025).

5.1.3 Benzene

To understand the potential association between benzene exposure and leukemia, 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) conclude that there is scientific evidence that exposure to benzene can cause leukemia in humans at some doses, based predominantly on observations of associations with acute myeloid leukemia (AML), with limited evidence of associations with acute lymphocytic leukemia (ALL) and chronic lymphocytic leukemia (CLL). In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that there is "sufficient evidence for causation for benzene and all leukemia types, *i.e.*, ALL, CLL, AML, and CML." ATSDR stated that is conclusion is based on "results of the meta-analyses" and "recent cohort studies and the finding that occupational benzene exposure is associated with reductions in both lymphoid and myeloid cell types."

Based on the available epidemiology studies and agency reviews that evaluated benzene exposure and leukemia, Dr. Goodman concluded that "epidemiology evidence supports an association between benzene exposures above 40 to 75 ppm-years and AML. The evidence is strongest for exposures that occurred within 10 to 15 years before diagnosis. The evidence also supports an association with MDS at exposures above 40 ppm-years. The evidence does not support a causal association between benzene and leukemia overall or ALL or CML" (Goodman, 2025). Dr. Goodman also concluded that "With the exception of leukemias and lymphomas combined in one study, no increases in leukemia incidence were reported in any chronic inhalation bioassay, and results from subchronic bioassays were inconsistent. Overall, animal bioassay findings do not support benzene as a cause of leukemia in humans" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between benzene exposure and leukemia concluded that, "the scientific evidence supports a causal association between benzene exposures and AML at exposures greater than 40 to 75 ppm-years, particularly for exposures within 10 to 15 years of diagnosis. Evidence also supports an association with MDS at exposures greater than 40 ppm-years. Epidemiology studies do not provide consistent or compelling evidence that benzene exposure is associated with ALL or CML" (Goodman, 2025).

5.1.4 Vinyl Chloride

To understand the potential association between vinyl chloride exposure and leukemia, 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 leukemia in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) concluded that the

evidence for causation is "below equipoise" for exposure to vinyl chloride and leukemia based predominantly on an epidemiology meta-analysis that observed no elevated risk.

Based on the available epidemiology studies and agency reviews that evaluated vinyl chloride exposure and leukemia, Dr. Goodman concluded that "Given the lack of a consistent or strong association between vinyl chloride exposure and leukemia, AML, and MDS and the methodological limitations of most epidemiology studies, particularly the high likelihood of exposure measurement error, I conclude that the epidemiology evidence does not support a causal association between vinyl chloride exposure and leukemia, AML, or MDS" (Goodman, 2025). Dr. Goodman also concluded, with regard to animal studies, that "[o]verall, treatment-related leukemias were not reported in any chronic vinyl chloride bioassay" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between vinyl chloride exposure and leukemia concluded that overall, the "evidence does not support a causal association between vinyl chloride and leukemia" (Goodman, 2025).

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

To understand the potential association between 1,2-tDCE exposure and leukemia, 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 leukemia (Goodman, 2025). Overall, US EPA (2010a,b) and ATSDR (2023) have not concluded that 1,2-tDCE is associated with increased leukemia risk in humans. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR (2017b) did not comment on whether *cis*-1,2-dichloroethylene (1,2-cDCE), 1,2-tDCE, or their mixtures are carcinogenic.

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 leukemia, increased leukemia

risk is not expected from TCE exposure, and TCE cancer toxicity values specific to leukemia 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 leukemia risk.

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

Chemical	Oral CSF[a,b] ($[mg/kg\text{-}day]^{-1}$)	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; $(mg/kg\text{-}day)^{-1}$ = 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} $(mg/kg\text{-}day)^{-1}$. NHL: 1.10×10^{-2} $(ppm)^{-1}$ × 1.97 ppm per mg/kg-day = 2.16×10^{-2} $(mg/kg\text{-}day)^{-1}$. Liver Cancer: 5.49×10^{-3} $(ppm)^{-1}$ × 2.82 ppm per mg/kg-day = 1.55×10^{-2} $(mg/kg\text{-}day)^{-1}$.
(b) The CSF for the three cancer types combined was derived by US EPA (2011a) as follows: $(5.49 \times 10^{-3}$ $[ppm]^{-1}$ × 1.7 ppm per mg/kg-day) × 5 = 4.6×10^{-2} $(mg/kg\text{-}day)^{-1}$. The factor of 5 is equal to the total risks summed across all three endpoints (4.6×10^{-2} $[mg/kg\text{-}day]^{-1}$) divided the by the kidney cancer risk (9.33×10^{-3} $[mg/kg\text{-}day]^{-1}$).
(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 ($[\mu g/m^3]^{-1}$; $[ppm]^{-1}$)	POD ($\mu g/m^3$ [ppb])	Cancer Type	Sources
TCE	4.1×10^{-6} $(\mu g/m^3)^{-1}$; 2.2×10^{-2} $(ppm)^{-1}$	LEC_{01} = 2,445[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} $(\mu g/m^3)^{-1}$; 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:
$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = 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 $\mu g/m^3$). The factor of 4 is equal to the total risks summed across all three endpoints (4.1×10^{-6} $[\mu g/m^3]^{-1}$) divided by the kidney cancer risk (1.0×10^{-6} $[\mu g/m^3]^{-1}$).
(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 cell carcinoma, NHL, and liver cancer	$4..6 \times 10^{-2}$ $(mg/kg\text{-}day)^{-1}$
	IUR	Renal cell carcinoma, NHL, and liver cancer	4.1×10^{-6} $(\mu g/m^3)^{-1}$

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; TCE = Trichloroethylene.
Source: US EPA (2011b).

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 weak for an association between PCE exposure and leukemia, increased leukemia risk is not expected from PCE exposure, and PCE cancer toxicity values specific to leukemia are not available. Further, using US EPA's PCE cancer toxicity criteria that are based on liver cancer is not predictive of leukemia 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 $([mg/kg\text{-}day]^{-1})$	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; $(mg/kg\text{-}day)^{-1}$ = 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 $([\mu g/m^3]^{-1}; [ppm]^{-1})$	POD $(\mu g/m^3$ [ppb])	Cancer Type (Sex/Species)	Source
PCE	2.6×10^{-7} $(\mu g/m^3)^{-1}$; 1.8×10^{-3} $(ppm)^{-1}$	390,000 (60,000)	Hepatocellular adenomas or carcinomas (male mice)	JISA (1993); US EPA (2012b,c)

Notes:
$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = 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} $(mg/kg\text{-}day)^{-1}$
	IUR		2.6×10^{-7} $(\mu g/m^3)^{-1}$

Notes:
$(\mu g/m^3)^{-1}$ = Per Micrograms per Cubic Meter; CSF = Cancer Slope Factor; IUR = Inhalation Unit Risk; $(mg/kg\text{-}day)^{-1}$ = 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.

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

Chemical	Oral CSF $([mg/kg\text{-}day]^{-1})$	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; $(mg/kg\text{-}day)^{-1}$ = 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 $([\mu g/m^3]^{-1}; [ppm]^{-1})$	POD $(\mu g/m^3$ [ppb])	Cancer Type	Sources
Benzene	2.2×10^{-6} to 7.8×10^{-6} $(\mu g/m^3)^{-1}$; 7.1×10^{-3} to 2.5×10^{-2} $(ppm)^{-1}$	383 (120)	Leukemia	Rinsky *et al.* (1981, 1987); US EPA (1998, 2003a)

Notes:
$\mu g/m^3$ = Micrograms per Cubic Meter; $(\mu g/m^3)^{-1}$ = 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} $(mg/kg\text{-}day)^{-1}$
	IUR		7.8×10^{-6} $(\mu g/m^3)^{-1}$

Notes:
$(\mu g/m^3)^{-1}$ = Per Micrograms 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 (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 is weak for an association between vinyl chloride exposure and leukemia, increased leukemia risk is not expected from vinyl chloride exposure, and vinyl chloride cancer toxicity values specific to leukemia are not available. Further, using US EPA's vinyl chloride cancer toxicity criteria that are based on liver cancer is not predictive of leukemia 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 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, 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 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, 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} $(mg/kg\text{-}day)^{-1}$ (adult continuous)
	IUR		4.4×10^{-6} $(\mu g/m^3)^{-1}$ (adult continuous)

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 Mr. Connard's residential and employment history, including the duration of time he was stationed at Camp Lejeune, and a risk evaluation for him based on his 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

Based on Mr. Connard's military records, he was stationed at Camp Lejeune from October 1977 to February 1979, and then again from March 1980 to July 1981 (Connard, 2024) (~ 34 months, or 2.8 years). Based on Mrs. Vivian Connard's deposition testimony, Mr. Connard lived or worked at Hadnot Point, Hospital Point, or Mainside Barracks while stationed at Camp Lejeune, which were serviced by the Hadnot Point Water Treatment Plant (HP WTP) (Connard, 2024). During his time at Camp Lejeune, Mr. Connard was assigned to heavy artillery (Connard, 2024). Mrs. Connard was unable to provide details regarding Mr. Connard's day-to-day activities, but with respect to water consumption, she stated in her deposition that, "he would tell me he'd be out in the field and he would drink a lot of water" and stated that Mr. Connard said he drank "gallons" of water (Connard, 2024). No information was provided regarding the frequency of field activities. Mrs. Connard was also unable to provide information regarding Mr. Connard's shower activity (*i.e.*, frequency or duration) while he was at Camp Lejeune.

Mrs. Connard is claiming that her husband's exposure to the water at Camp Lejeune was the cause of his acute myeloid leukemia (AML), which he was diagnosed with on March 24, 2001 (Connard, 2024). According to Mrs. Connard, her husband passed away from cardiac arrest on May 14, 2010 (Connard, 2024).

6.2 Plaintiff Exposure Estimates

Exposure estimates for Mr. Connard 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 Mr. Connard'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 their time on-base. Since Mr. Connard lived at the Mainside Barracks (*i.e.*, the main part of Camp Lejeune served by the Hadnot Point water system), I evaluated shower exposures for Mr. Connard from the HP WTP only. Mr. Connard's 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). Risks were calculated for the

following baseline exposure pathways and scenarios for the exposure period of concern (approximately 3 years) for Mr. Connard:

- Drinking Water Ingestion – For this exposure pathway, it is not clear that the plaintiff's water ingestion occurred from only one of the two WTPs, I evaluated three scenarios for both the HP and TT WTPs: (1) central tendency exposure (CTE), which assumes ingestion of 1.3 liter (L) of tap water per day; (2) reasonable maximum exposure (RME), which assumes ingestion of 3.3 L of tap water per day; and (3) military high-end exposure, which assumes ingestion of 6 L of tap water per day.
    - There is limited information about Mr. Connard's typical activities and how that would have impacted water consumption patterns. However, since Mr. Connard did engage in some field activity, according to Mrs. Connard's deposition, I evaluate all three scenarios, including the military high-end exposure scenario.
- Dermal and inhalation Exposures from Showering – 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 a communal showering facility exposure model (ATSDR, 2024a), and based on Mr. Connard's location of residence during his time at Camp LeJeune (HP WTP). The inhalation concentrations from the shower model were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Both the CTE and RME exposures from the communal shower facility model are estimated based on a mean daily shower duration of 20 minutes, with a standard deviation (SD) of 5.3 minutes (the SD is the model's default value) (LaKind, 2025). As described in Dr. LaKind's report (LaKind, 2025), using these inputs to the communal shower model results in a range of shower durations including 30 minutes for 95% of the people being modeled. Note that the communal shower model accounts for additional water uses, including sinks and toilets.
    - No information was provided regarding the frequency and duration or Mr. Connard's showers; therefore, I assume that Mr. Connard took one 20-minute shower per day while on-base, which is consistent with the mean duration used in the shower model.

Based on the above exposure pathways, the following exposure scenarios are evaluated for Mr. Connard:

- The CTE exposure scenario, which includes the following exposure pathways: CTE drinking water ingestion (TT and HP WTPs), and CTE dermal and inhalation exposures from showering (HP WTP).
- The RME exposure scenario, which includes the following exposure pathways: RME drinking water ingestion (TT and HP WTPs), and RME dermal and inhalation exposures from showering (HP WTP).
- The military high-end exposure scenario, which includes the following exposure pathways: military high-end drinking water ingestion (TT and HP WTPs), and RME dermal and inhalation exposures from showering (HP WTP).

6.3 Regulatory Risk Calculations

Risk calculations for Mr. Connard 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 Mr. Connard (approximately 3 years) 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 Mr. Connard'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 Excess Lifetime Cancer Risks (ELCRs) by Exposure Pathway for Mr. Connard[a]

Exposure Pathway	Water Source	Excess Lifetime Cancer Risks		
		Central Tendency	Reasonable Maximum	Military High-End
Baseline Exposure Pathways				
Ingestion of Drinking Water	HP WTP	2×10^{-5}	5×10^{-5}	1×10^{-4}
	TT WTP	3×10^{-6}	7×10^{-6}	1×10^{-5}
Dermal Contact from Showering in the Barracks	HP WTP	2×10^{-6}	3×10^{-6}	3×10^{-6}
Inhalation from Showering in the Barracks	HP WTP	3×10^{-6}	6×10^{-6}	6×10^{-6}
Total ELCRs (All Pathways)				
Assuming Drinking Water Comes from HP WTP		3×10^{-5}	6×10^{-5}	1×10^{-4}
Assuming Drinking Water Comes from TT WTP		8×10^{-6}	2×10^{-5}	2×10^{-5}

Notes:
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 Mr. Connard's estimated exposures (*i.e.*, military high-end exposure scenario at Hadnot Point [HP]) 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 target excess cancer risk range. Note that this cancer risk estimate is for all cancer types for all chemical exposures evaluated and is driven predominantly by TCE and vinyl chloride in drinking water (see Appendix D, Table D.2). As discussed in Section 5, none of the regulatory agency documents concluded that vinyl chloride exposure is a known cause of leukemia in humans. US EPA's cancer toxicity criteria are based on liver cancer for vinyl chloride. Thus, cancer risk estimates for vinyl chloride are overly protective of leukemia risks. As discussed in Section 5, the TCE cancer toxicity values are based on NHL, kidney cancer, and liver cancer combined, and are summed across all of those endpoints; therefore, the cancer risk estimates from TCE also result in overly conservative estimates of risk for leukemia (AML) for the plaintiff. Therefore, the main drivers of the highest risk estimate for Mr. Connard's exposures (TCE and vinyl chloride) are not predictive of leukemia (AML) risks and should not be interpreted to suggest there is an excess AML risk of 1×10^{-4}. As shown in Appendix D, the estimated cancer risks for benzene (the only chemical toxicity value that is based on leukemia) are equal to the lower end of US EPA's target risk range, with a maximum ELCR of 1×10^{-6}, or 1 leukemia case in 1,000,000 exposed people, or 0.0001% increased risk of leukemia from the military high-end exposure scenario at HP.

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). Therefore, they are based on conservative assumptions about exposures and health effects, and, consequently, are likely overestimates for healthy 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 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 do not support the conclusion that Mr. Connard's AML was a result of being 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 exposure for Mr. Connard, and applying conservative, health-protective assumptions, Mr. Connard's exposures to chemicals in the Camp Lejeune drinking water did not increase his overall cancer risk by more than 0.01% (*i.e.*, 1×10^{-4}, or one cancer case in 10,000 exposed people) over his background cancer risk. It is notable that half of this risk is from vinyl chloride, a chemical for which no public health agency has concluded is causally associated with leukemia (see Section 5). The cancer risk from the only chemical for which ATSDR concludes there is "sufficient evidence" of an association between exposure and leukemia – benzene (*i.e.*, $1 \times 10^{-6,}$ or 0.0001%) – is equal to the lower end of US EPA's acceptable risk range. Therefore, one cannot reasonably conclude that Mr. Connard's exposures to chemicals in Camp Lejeune water are causally associated with his AML.

Because the cancer risks presented in Table 6.1 are overestimated for healthy individuals in a population (like Mr. Connard during the time of his alleged exposure), in Section 7, I have also conducted margin of exposure (MoE) comparisons between 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 leukemia.

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:

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

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.

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 Table D.1, the MoEs for the plaintiff range from 190 to 22,000,000. For benzene exposure (*i.e.*, the only chemical for which the toxicity values are based on leukemia) *via* all exposure pathways, the MoEs range from 3,300 to 100,000. 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 Leukemia

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 leukemia risk (Goodman, 2025).

Although Dr. Goodman reviewed epidemiology studies that evaluated potential correlations between chemical exposures and leukemia 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 on base and leukemia or leukemia subtypes. Although some studies reported a few statistically significant risk estimates, they were not consistent across analyses of the Camp Lejeune population, and several were <1. (Goodman, 2025)

8.1 Trichloroethylene (TCE)

As summarized in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" (ATSDR, 2017b), concluded that for TCE there is "equipoise and above evidence for causation for all types of leukemia, including AML, ALL, CML and CLL." US EPA (2011a, 2020a), IARC (2014), NTP (2015), and ATSDR (2019a) (inconsistent with its Camp Lejeune evidence assessment) do not conclude that TCE exposure is a known cause of leukemia in humans. Dr. Goodman concluded that, overall, the scientific evidence does not support a causal association between TCE and leukemia (Goodman, 2025).

Based on my review of Dr. Goodman's report (Goodman, 2025), the only epidemiology (occupational) study that also reported exposure information for TCE – Talibov *et al*. (2014) – reported no association between TCE exposure and leukemia at concentrations as high as 121 ppm-years. I converted this exposure estimate from an occupational exposure to a continuous daily exposure for a resident (121 ppm-years × 5/7 days × 8/24 hours = 29 ppm-years). This exposure estimate is orders of magnitude above (1,600-fold higher than) those estimated for Mr. Connard (0.018 ppm-years). I calculated Mr. Connard's cumulative TCE exposure in ppm-years by converting the maximum TCE daily inhalation concentration (34 µg/m^3) to units of ppm (0.0063 ppm)[7] and multiplying by the number of years that Mr. Connard was at Camp Lejeune (2.8 years), resulting in an exposure estimate of 0.018 ppm-years.

Mr. Connard'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

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

leukemia in chronic animal bioassays at TCE oral doses up to 2,339 mg/kg-d. This dose is orders of magnitude higher than Mr. Connard's maximum estimated TCE oral LADD of 0.001 mg/kg-day.

See Appendix D for Mr. Connard's inhalation exposure and oral dose estimates.

8.2 Tetrachloroethylene (PCE)

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 causation is "below equipoise" for exposure to PCE and leukemia. ATSDR (2019b), US EPA (2012b, 2020b), and IARC (2014) do not conclude that PCE exposure is a known cause of leukemia in humans. Dr. Goodman also concluded that the scientific evidence does not support a causal association between PCE and leukemia (Goodman, 2025).

Based on my review of Dr. Goodman's report (Goodman, 2025), the only epidemiology (occupational) study that also reported exposure information for PCE – Talibov *et al*. (2014) – reported no association between PCE exposure and leukemia at concentrations as high as 106 ppm-years. I converted this exposure estimate from an occupational exposure to a continuous daily exposure for a resident (106 ppm-years × 5/7 days × 8/24 hours = 25 ppm-years). This exposure estimate is orders of magnitude above (60,000-fold higher than) those estimated for Mr. Connard (0.0004 ppm-years). I calculated Mr. Connard's cumulative PCE exposure in ppm-years by converting the maximum PCE daily inhalation concentration (1 $\mu g/m^3$) to units of ppm (0.00015 ppm)[8] and multiplying by the number of years that Mr. Connard was at Camp Lejeune (2.8 years), resulting in an exposure estimate of 0.0004 ppm-years.

Mr. Connard'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 leukemia in chronic animal bioassays at PCE oral doses up to 1,072 mg/kg-d. This dose is orders of magnitude higher than Mr. Connard's maximum estimated PCE oral LADD of 0.00024 mg/kg-day.

See Appendix D for Mr. Connard'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 there is "sufficient evidence for causation for benzene and all leukemia types, *i.e.*, ALL, CLL, AML, and CML." US EPA (2003a), ATSDR (2007a, 2015), and IARC (2018) conclude that there is scientific evidence that high exposure to benzene can cause leukemia in humans. Dr. Goodman concluded that the scientific evidence supports a causal association between benzene exposures and AML at cumulative exposures ≥40-75 ppm-years (Goodman, 2025).

For my exposure comparisons, I relied on the leukemia epidemiology studies conducted by Rinsky *et al*. (1981, 1987) that are considered by US EPA to be the most reliable studies from which to quantify a potential association between benzene exposure and leukemia and from which to derive benzene cancer toxicity criteria (US EPA, 2003a) (see Section 5). A comparison of the exposure information from this study to the plaintiff's exposure estimates is described in Section 7; *i.e.*, Mr. Connard's benzene exposure

[8] 1 ppm PCE = 6.78 mg/m^3 (CDC, 2019b).

estimates (over all exposure pathways) are 3,300 to 100,000 times lower than the leukemia PODs for benzene that are based on the Rinsky *et al*. (1981, 1987) studies.

Further, Mr. Connard's cumulative benzene inhalation exposure estimate (0.0003 ppm-years) is orders of magnitude below the occupational cumulative benzene exposure estimate (≥40-75 ppm-years) that Dr. Goodman discusses, based on the most current scientific information, as the benzene exposure concentrations above which there is evidence of an association with leukemia (Goodman, 2025). Adjusting the 75 ppm-year occupational exposure estimate to a continuous exposure estimate for the general population results in a benzene exposure estimate of 18 ppm-years (75 ppm-years × 5/7 days per week × 8/24 hours per day = 18 ppm-years), which is ~53,000-fold higher than Mr. Connard's estimated benzene exposure during the time he was stationed at Camp Lejeune (0.00033 ppm-years). I calculated Mr. Connard's cumulative benzene exposure in ppm-years by converting the maximum benzene daily inhalation concentration (0.38 µg/m^3) to units of ppm (0.00012 ppm)[9] and multiplying by the number of years that Mr. Connard was stationed at Camp Lejeune (2.8 years), resulting in an exposure estimate of 0.00033 ppm-years. Based on a similar calculation, Mr. Connard's estimated benzene exposures would be approximately 30,000-fold lower than a 40 ppm-year benzene exposure estimate (after adjusting for continuous exposures).

Mr. Connard's oral 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 leukemia in chronic animal bioassays at benzene oral doses up to 250 mg/kg-d. This dose is orders of magnitude higher than Mr. Connard's maximum estimated benzene oral LADD of 0.000017 mg/kg-day.

See Appendix D for Mr. Connard's inhalation exposure and oral dose estimates.

8.4 Vinyl Chloride

As summarized in Section 5, ATSDR, in its "Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune" (ATSDR, 2017b), concluded that that the evidence for causation is "below equipoise" for exposure to vinyl chloride and leukemia. ATSDR (2024b) and US EPA (2003b) did not conclude that vinyl chloride exposure is a known cause of leukemia in humans. Dr. Goodman concluded that the evidence does not support a causal association between vinyl chloride and leukemia (Goodman, 2025).

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

Mr. Connard's vinyl chloride exposure estimates are well below those reported in the animal bioassays discussed by Dr. Goodman (2025). Dr. Goodman's report indicates that there are no significant increases or trends in leukemia in 2-year chronic animal bioassays at vinyl chloride oral doses up to 300 mg/kg-d and vinyl chloride daily inhalation concentrations as high as 30,000 ppm (Goodman, 2025). These exposures are orders of magnitude higher than Mr. Connard's maximum estimated vinyl chloride oral LADD of 0.000069 mg/kg-day and inhalation LADE of 0.000029 ppm (0.073 µg/m^3).[10]

[9] 1 ppm benzene = 3,190 µg/m^3 (CDC, 2019c).
[10] 1 ppm vinyl chloride = 2,560 µg/m^3 (CDC, 2019d).

See Appendix D for Mr. Connard's inhalation exposure and oral dose estimates.

8.5 *trans*-1,2-Dichloroethylene (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 leukemia (Goodman, 2025). ATSDR (2017b) provided no comment on whether there is a causal association between 1,2-tDCE exposure and leukemia. US EPA and ATSDR do not conclude that there is an association between exposure to 1,2-tDCE and leukemia (see Section 5). Therefore, exposure comparisons cannot be made for 1,2-tDCE.

8.6 Conclusions from Epidemiology and Toxicology Studies

As described above, Mr. Connard's exposures to TCE, PCE, benzene, and vinyl chloride were well below levels of concern for leukemia (benzene), or well below exposure levels where significant increases in leukemia were not observed (PCE, benzene, vinyl chloride). Therefore, these results provide additional support that Mr. Connard's estimated exposures while stationed at Camp Lejeune would not have been expected to lead to his leukemia.

9 Rebuttal of the Plaintiff's Experts' Reports

I reviewed the reports of the plaintiff's experts: Dr. Kelly Reynolds (2025a), who provided exposure estimates for the plaintiff, and Dr. Lukasz P. Gondek (2025), who provided opinions on specific causation for Mr. Connard. Below, I note the methodological flaws in their analyses, with respect to risk assessment.

9.1 Dr. Reynolds

Dr. Reynolds' report (Reynolds, 2025a) does not provide reliable estimates of TCE, PCE, vinyl chloride, and benzene exposures for Mr. Connard from 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 (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). 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).

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 was stationed 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, 2025b). 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 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 units of microgram of a chemical per cubic meter of air (µg/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 total ingested amount of 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 comparison to exposure estimates in most reliable animal or epidemiology studies. Doses (mg/kg-day) or inhalation concentrations (µg/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 Dr. Gondek

Dr. Gondek's report does not provide a robust analysis of specific causation for leukemia risk with regard to Mr. Connard's alleged exposures. Dr. Gondek concludes that the evidence demonstrates that Mr. Connard's TCE, PCE, and benzene exposures during his time at Camp Lejeune are "more likely than not" the cause of his AML (Gondek, 2025). However, Dr. Gondek's reliance on Dr. Reynolds' exposure estimates is seriously flawed and not consistent with US EPA risk assessment guideline. Therefore, Dr. Gondek cannot use these exposure estimates to determine Mr. Connard's risks. Below, I describe several flaws in his analysis:

- Dr. Gondek's risk evaluation is 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 (discussed in Section 3).
- Dr. Gondek relies on Dr. Reynolds' exposure charts to support his conclusion that Mr. Connard's exposures were "significant" and "substantial." However, as discussed in the previous section, in regard to Dr. Reynolds' report, 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 months will, undoubtedly, result in a very large value. But it is an incorrect value for the purpose of risk evaluation. Therefore, Dr. Gondek's conclusions based on estimates of total chemical mass exposure for Mr. Connard are meaningless and misleading and cannot be relied upon for risk evaluation for Mr. Connard.
- Further, Dr. Gondek incorrectly refers to the total masses from Dr. Reynolds' report (in µg) as a cumulative drinking water concentration (ppb or µg/L). Dr. Gondek states that Mr. Connard would have ingested "1,574,286 µg/L of TCE." However, the value of 1,574,286 discussed by Dr. Reynolds *is a total mass of TCE (in µg) calculated for the entire time spent at Camp Lejeune, based on water concentrations and ATSDR drinking water exposure assumptions, and not a concentration (ppb or µg/L) in drinking water*. According to Dr. Reynolds' report, the total concentration of TCE in the water that Mr. Connard is assumed to have been exposed to during his time at Camp Lejeune ranged from 69-546 µg/L, orders of magnitude lower than what Dr. Gondek incorrectly states. Therefore, Dr. Gondek's statement that Mr. Connard would have been exposed to 1,574,286 µg/L TCE in drinking water is grossly incorrect and misleading. Dr. Gondek incorrectly makes similar statements for the total mass of PCE and benzene, referring to both as µg/L.
- Dr. Gondek's comparison to US EPA MCLs for allowable chemical concentrations in drinking water is not a reliable risk evaluation method.
    - US EPA does not use MCLs to evaluate potential risks to human health.

- MCLs are derived to be acceptable (health-protective) daily drinking water concentrations over a lifetime of exposure (~70 years) (US EPA, 2024b), which is much longer than Mr. Connard's 3 years of exposure during his time at Camp Lejeune.
- Per US EPA guidance for cancer risk assessment, it is the cumulative dose averaged over a lifetime that is critical for evaluating cancer risk. As shown in equations in Section 3, estimated doses are averaged over a 70-year lifetime before calculating risks. Therefore, once averaged over a lifetime, it is possible to have a dose based on a higher exposure concentration for a shorter exposure duration (which is the case for some of the plaintiffs at Camp Lejeune) not exceed a dose based on a lower exposure concentration for a longer exposure duration (like ingesting water at the MCL every day for a lifetime). In both cases, risks would be considered acceptable under US EPA guidelines. Therefore, a simple comparison of drinking water concentrations to MCLs, without considering exposure duration, is not consistent with standard risk assessment practice and is misleading.

- Dr. Gondek refers to a study by Cohn *et al*. (1994) to provide support for his conclusions regarding comparison to the TCE MCL. However, as discussed by Dr. Goodman (2025), Cohn *et al*. (1994) was considered by US EPA in its toxicological profile for TCE (US EPA, 2011a), within which US EPA concluded that the evidence "was not robust or conclusive" for an association between TCE exposure and childhood leukemia.
- Dr. Gondek also refers to exposure information from several Camp Lejeune studies to support his 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 on base and leukemia or leukemia subtypes. Although some studies reported a few statistically significant risk estimates, they were not consistent across analyses of the Camp Lejeune population, and several were <1. (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, overall, the scientific evidence does not support a causal association between TCE, PCE, vinyl chloride, or 1,2-tDCE exposure and leukemia, and only supports a causal association between benzene exposures ≥40-75 ppm-years (much higher than Mr. Connard's) and AML.

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 estimated for Mr. Connard's exposures do not exceed US EPA's acceptable cancer risk range.

Therefore, Dr. Reynolds' and Dr. Gondek's expert reports do not change my opinions, as discussed in my report and summarized in Section 10, regarding Mrs. Connard's claim that exposures from Camp Lejeune are the cause of Mr. Connard's AML.

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 AML epidemiology studies and toxicology studies discussed in Section 8, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Mr. Connard's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 3 years that he was stationed at Camp Lejeune are causally associated with his AML.

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Appendix A

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

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

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

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Poster Presentations

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****Best Overall Abstract Award Winner, Risk Assessment Specialty Section**

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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.

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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.

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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.

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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 Stephen Connard

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	9.0E-05	mg/kg-day	3.6E-06	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	2.0E-07	5.5E-02	mg/kg-day	1.5E+04	N
				trans -1,2-Dichloroethylene	2.5E-03	mg/kg-day	1.0E-04	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	2.6E-04	mg/kg-day	1.1E-05	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	2.2E-08	5.0E+01	mg/kg-day	4.8E+06	N
				Trichloroethylene	5.9E-03	mg/kg-day	2.4E-04	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.1E-05	2.1E-01	mg/kg-day	8.8E+02	N
				Vinyl Chloride	3.7E-04	mg/kg-day	1.5E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	1.1E-05	1.3E-01	mg/kg-day	8.9E+03	N
										Total for Hadnot Point Ingestion (CTE):	**2E-05**				
CTE	Hadnot Point	Shower water	Dermal	Benzene	1.3E-05	mg/kg-day	5.3E-07	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	2.9E-08	5.5E-02	mg/kg-day	1.0E+05	N
				trans -1,2-Dichloroethylene	3.1E-04	mg/kg-day	1.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.6E-04	mg/kg-day	6.5E-06	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.4E-08	5.0E+01	mg/kg-day	7.7E+06	N
				Trichloroethylene	8.6E-04	mg/kg-day	3.5E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.6E-06	2.1E-01	mg/kg-day	6.0E+03	N
				Vinyl Chloride	2.6E-05	mg/kg-day	1.1E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	7.6E-07	1.3E-01	mg/kg-day	1.3E+05	N
										Total for Hadnot Point Dermal (CTE):	**2E-06**				
CTE	Hadnot Point	Indoor air	Inhalation	Benzene	1.7E-01	$\mu g/m^3$	6.9E-03	$\mu g/m^3$	7.8E-06	$(\mu g/m^3)^{-1}$	5.4E-08	3.8E+02	$\mu g/m^3$	5.6E+04	N
				trans -1,2-Dichloroethylene	5.1E+00	$\mu g/m^3$	2.1E-01	$\mu g/m^3$	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	4.5E-01	$\mu g/m^3$	1.8E-02	$\mu g/m^3$	2.6E-07	$(\mu g/m^3)^{-1}$	4.7E-09	4.0E+05	$\mu g/m^3$	2.2E+07	N
				Trichloroethylene	1.5E+01	$\mu g/m^3$	6.1E-01	$\mu g/m^3$	4.1E-06	$(\mu g/m^3)^{-1}$	2.5E-06	2.4E+03	$\mu g/m^3$	4.0E+03	N
				Vinyl Chloride	8.2E-01	$\mu g/m^3$	3.3E-02	$\mu g/m^3$	4.4E-06	$(\mu g/m^3)^{-1}$	1.5E-07	2.3E+04	$\mu g/m^3$	6.9E+05	N
										Total for Hadnot Point Inhalation (CTE):	**3E-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	1.8E-04	mg/kg-day	7.3E-06	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.3E-03	mg/kg-day	5.3E-05	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.1E-07	5.0E+01	mg/kg-day	9.5E+05	N
				Trichloroethylene	5.4E-05	mg/kg-day	2.2E-06	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	1.0E-07	2.1E-01	mg/kg-day	9.6E+04	N
				Vinyl Chloride	9.8E-05	mg/kg-day	4.0E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	2.9E-06	1.3E-01	mg/kg-day	3.4E+04	N
										Total for Tarawa Terrace Ingestion (CTE):	**3E-06**				
Reasonable Maximum Exposure (RME)															
RME	Hadnot Point	Drinking water	Ingestion	Benzene	2.2E-04	mg/kg-day	8.9E-06	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	4.9E-07	5.5E-02	mg/kg-day	6.2E+03	N
				trans -1,2-Dichloroethylene	6.2E-03	mg/kg-day	2.5E-04	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	6.5E-04	mg/kg-day	2.6E-05	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	5.5E-08	5.0E+01	mg/kg-day	1.9E+06	N
				Trichloroethylene	1.5E-02	mg/kg-day	6.1E-04	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	2.8E-05	2.1E-01	mg/kg-day	3.5E+02	N
				Vinyl Chloride	9.1E-04	mg/kg-day	3.7E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	2.6E-05	1.3E-01	mg/kg-day	3.6E+03	N
										Total for Hadnot Point Ingestion (RME):	**5E-05**				
RME	Hadnot Point	Shower water	Dermal	Benzene	1.6E-05	mg/kg-day	6.5E-07	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	3.6E-08	5.5E-02	mg/kg-day	8.5E+04	N
				trans -1,2-Dichloroethylene	3.8E-04	mg/kg-day	1.5E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	2.0E-04	mg/kg-day	8.1E-06	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.7E-08	5.0E+01	mg/kg-day	6.2E+06	N
				Trichloroethylene	1.1E-03	mg/kg-day	4.4E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	2.0E-06	2.1E-01	mg/kg-day	4.7E+03	N
				Vinyl Chloride	3.2E-05	mg/kg-day	1.3E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	9.3E-07	1.3E-01	mg/kg-day	1.0E+05	N
										Total for Hadnot Point Dermal (RME):	**3E-06**				

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	Hadnot Point	Indoor air	Inhalation	Benzene	3.8E-01	µg/m3	1.5E-02	µg/m3	7.8E-06	$(\mu g/m^3)^{-1}$	1.2E-07	3.8E+02	$\mu g/m^3$	2.5E+04	N
				trans -1,2-Dichloroethylene	1.1E+01	µg/m3	4.4E-01	µg/m3	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.0E+00	µg/m3	4.0E-02	µg/m3	2.6E-07	$(\mu g/m^3)^{-1}$	1.1E-08	4.0E+05	$\mu g/m^3$	9.9E+06	N
				Trichloroethylene	3.4E+01	µg/m3	1.4E+00	µg/m3	4.1E-06	$(\mu g/m^3)^{-1}$	5.6E-06	2.4E+03	$\mu g/m^3$	1.8E+03	N
				Vinyl Chloride	1.8E+00	µg/m3	7.3E-02	µg/m3	4.4E-06	$(\mu g/m^3)^{-1}$	3.2E-07	2.3E+04	$\mu g/m^3$	3.1E+05	N
										Total for Hadnot Point Inhalation (RME):	**6E-06**				
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	4.4E-04	mg/kg-day	1.8E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	3.2E-03	mg/kg-day	1.3E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	2.7E-07	5.0E+01	mg/kg-day	3.9E+05	N
				Trichloroethylene	1.3E-04	mg/kg-day	5.3E-06	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	2.4E-07	2.1E-01	mg/kg-day	4.0E+04	N
				Vinyl Chloride	2.4E-04	mg/kg-day	9.7E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	7.0E-06	1.3E-01	mg/kg-day	1.4E+04	N
										Total for Tarawa Terrace Ingestion (RME):	**7E-06**				
Military High-End Exposure															
Military	Hadnot Point	Drinking water	Ingestion	Benzene	4.1E-04	mg/kg-day	1.7E-05	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	9.1E-07	5.5E-02	mg/kg-day	3.3E+03	N
				trans -1,2-Dichloroethylene	1.2E-02	mg/kg-day	4.8E-04	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.2E-03	mg/kg-day	4.8E-05	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.0E-07	5.0E+01	mg/kg-day	1.0E+06	N
				Trichloroethylene	2.7E-02	mg/kg-day	1.1E-03	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	5.0E-05	2.1E-01	mg/kg-day	1.9E+02	N
				Vinyl Chloride	1.7E-03	mg/kg-day	6.9E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	4.9E-05	1.3E-01	mg/kg-day	1.9E+03	N
										Total for Hadnot Point Ingestion (Military):	**1E-04**				
Military	Hadnot Point	Shower water	Dermal	Benzene	1.6E-05	mg/kg-day	6.5E-07	mg/kg-day	5.5E-02	$(mg/kg\text{-}day)^{-1}$	3.6E-08	5.5E-02	mg/kg-day	8.5E+04	N
				trans -1,2-Dichloroethylene	3.8E-04	mg/kg-day	1.5E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	2.0E-04	mg/kg-day	8.1E-06	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	1.7E-08	5.0E+01	mg/kg-day	6.2E+06	N
				Trichloroethylene	1.1E-03	mg/kg-day	4.4E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	2.0E-06	2.1E-01	mg/kg-day	4.7E+03	N
				Vinyl Chloride	3.2E-05	mg/kg-day	1.3E-06	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	9.3E-07	1.3E-01	mg/kg-day	1.0E+05	N
										Total for Hadnot Point Dermal (Military):	**3E-06**				
Military	Hadnot Point	Indoor air	Inhalation	Benzene	3.8E-01	µg/m3	1.5E-02	µg/m3	7.8E-06	$(\mu g/m^3)^{-1}$	1.2E-07	3.8E+02	$\mu g/m^3$	2.5E+04	N
				trans -1,2-Dichloroethylene	1.1E+01	µg/m3	4.4E-01	µg/m3	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	1.0E+00	µg/m3	4.0E-02	µg/m3	2.6E-07	$(\mu g/m^3)^{-1}$	1.1E-08	4.0E+05	$\mu g/m^3$	9.9E+06	N
				Trichloroethylene	3.4E+01	µg/m3	1.4E+00	µg/m3	4.1E-06	$(\mu g/m^3)^{-1}$	5.6E-06	2.4E+03	$\mu g/m^3$	1.8E+03	N
				Vinyl Chloride	1.8E+00	µg/m3	7.3E-02	µg/m3	4.4E-06	$(\mu g/m^3)^{-1}$	3.2E-07	2.3E+04	$\mu g/m^3$	3.1E+05	N
										Total for Hadnot Point Inhalation (Military):	**6E-06**				
Military	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	8.1E-04	mg/kg-day	3.3E-05	mg/kg-day	NA	NA	NA	NA	NA	NA	NA
				Tetrachloroethylene	6.0E-03	mg/kg-day	2.4E-04	mg/kg-day	2.1E-03	$(mg/kg\text{-}day)^{-1}$	5.1E-07	5.0E+01	mg/kg-day	2.1E+05	N
				Trichloroethylene	2.5E-04	mg/kg-day	1.0E-05	mg/kg-day	4.6E-02	$(mg/kg\text{-}day)^{-1}$	4.6E-07	2.1E-01	mg/kg-day	2.1E+04	N
				Vinyl Chloride	4.5E-04	mg/kg-day	1.8E-05	mg/kg-day	7.2E-01	$(mg/kg\text{-}day)^{-1}$	1.3E-05	1.3E-01	mg/kg-day	7.3E+03	N
										Total for Tarawa Terrace Ingestion (Military):	**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	2.83	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

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

Table D.2 Summary of Risks By Exposure Pathway for Stephen Connard

Exposure Scenario	Exposure Point	Analyte	Baseline Exposure Pathways						Total ELCR
			Ingestion (Drinking Water)		Dermal (Shower)		Inhalation (Indoor Air)		
			ELCR	%	ELCR	%	ELCR	%	
Central Tendency Exposure (CTE)									
CTE	Hadnot Point: All Exposure Pathways	Benzene	2.0E-07	0.9%	2.9E-08	1%	5.4E-08	2%	2.8E-07
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	2.2E-08	0.1%	1.4E-08	0.6%	4.7E-09	0.2%	4.0E-08
		Trichloroethylene	1.1E-05	50%	1.6E-06	67%	2.5E-06	92%	1.5E-05
		Vinyl Chloride	1.1E-05	49%	7.6E-07	32%	1.5E-07	5%	1.2E-05
		Pathway-Specific Total:	**2E-05**		**2E-06**		**3E-06**		**3E-05**
CTE	Tarawa Terrace: Drinking Water Hadnot Point: Dermal and Inhalation from Showering	Benzene	NA	--	2.9E-08	1%	5.4E-08	2%	8.2E-08
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	1.1E-07	4%	1.4E-08	0.6%	4.7E-09	0.2%	1.3E-07
		Trichloroethylene	1.0E-07	3%	1.6E-06	67%	2.5E-06	92%	4.2E-06
		Vinyl Chloride	2.9E-06	93%	7.6E-07	32%	1.5E-07	5%	3.8E-06
		Pathway-Specific Total:	**3E-06**		**2E-06**		**3E-06**		**8E-06**
Reasonable Maximum Exposure (RME)									
RME	Hadnot Point: All Exposure Pathways	Benzene	4.9E-07	0.9%	3.6E-08	1%	1.2E-07	2%	6.4E-07
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	5.5E-08	0.1%	1.7E-08	0.6%	1.1E-08	0.2%	8.3E-08
		Trichloroethylene	2.8E-05	51%	2.0E-06	68%	5.6E-06	93%	3.6E-05
		Vinyl Chloride	2.6E-05	48%	9.3E-07	31%	3.2E-07	5%	2.8E-05
		Pathway-Specific Total:	**5E-05**		**3E-06**		**6E-06**		**6E-05**
RME	Tarawa Terrace: Drinking Water Hadnot Point: Dermal and Inhalation from Showering	Benzene	NA	--	3.6E-08	1%	1.2E-07	2%	1.6E-07
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	2.7E-07	4%	1.7E-08	0.6%	1.1E-08	0.2%	3.0E-07
		Trichloroethylene	2.4E-07	3%	2.0E-06	68%	5.6E-06	93%	7.9E-06
		Vinyl Chloride	7.0E-06	93%	9.3E-07	31%	3.2E-07	5%	8.2E-06
		Pathway-Specific Total:	**7E-06**		**3E-06**		**6E-06**		**2E-05**
Military High-End Exposure									
Military	Hadnot Point: All Exposure Pathways	Benzene	9.1E-07	0.9%	3.6E-08	1%	1.2E-07	2%	1.1E-06
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	1.0E-07	0.1%	1.7E-08	0.6%	1.1E-08	0.2%	1.3E-07
		Trichloroethylene	5.0E-05	50%	2.0E-06	68%	5.6E-06	93%	5.8E-05
		Vinyl Chloride	4.9E-05	49%	9.3E-07	31%	3.2E-07	5%	5.1E-05
		Pathway-Specific Total:	**1E-04**		**3E-06**		**6E-06**		**1E-04**
Military	Tarawa Terrace: Drinking Water Hadnot Point: Dermal and Inhalation from Showering	Benzene	NA	--	3.6E-08	1%	1.2E-07	2%	1.6E-07
		trans -1,2-Dichloroethylene	NA	--	NA	--	NA	--	NA
		Tetrachloroethylene	5.1E-07	4%	1.7E-08	0.6%	1.1E-08	0.2%	5.4E-07
		Trichloroethylene	4.6E-07	3%	2.0E-06	68%	5.6E-06	93%	8.1E-06
		Vinyl Chloride	1.3E-05	93%	9.3E-07	31%	3.2E-07	5%	1.4E-05
		Pathway-Specific Total:	**1E-05**		**3E-06**		**6E-06**		**2E-05**

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] ($[mg/kg\text{-}day]^{-1}$)	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: $10\% \div 0.75\ (mg/kg\text{-}day)^{-1} = 0.133$ 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] ($[\mu g/m^3]^{-1}$)	POD[b] ($\mu g/m^3$ [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:
$\mu g/m^3$ = 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: $10\% \div 0.0000044\ (\mu g/m^3)^{-1} = 22{,}727\ \mu g/m^3$.

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