Exhibit 545

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

In the Case of:
Diane Rothchild *v.* United States

Prepared by

Lisa Bailey

Lisa A. Bailey, Ph.D.

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

May 8, 2025

GRADIENT

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

Table of Contents

Page

List of Tables

List of Figures

Abbreviations

$\mu g/m^3$	Micrograms per Cubic Meter
1,2-tDCE	*trans*-1,2-Dichloroethylene
ADD	Average Daily Dose
ADE	Average Daily Exposure
AFC	Antibody-Forming Cell
ALC	Absolute Lymphocyte Count
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BMDL	Lower Confidence Limit on the Benchmark Dose
CTE	Central Tendency Exposure
DEC	Daily Exposure Concentration
DED	Daily Exposure Dose
ED	Exposure Duration
EF	Exposure Frequency
EU	European Union
HB	Holcomb Boulevard
HI	Hazard Index
HP	Hadnot Point
HQ	Hazard Quotient
L	Liter
LOAEL	Lowest Observed Adverse Effect Level
MCL	Maximum Contaminant Level
mg/kg-day	Milligram per Kilogram Body Weight-Day
MoE	Margin of Exposure
MRL	Minimal Risk Level
NOAEL	No Observed Adverse Effect Level
OEL	Occupational Exposure Limit
p-RfC	Provisional Reference Concentration
PBPK	Physiologically-Based Pharmacokinetic
PCE	Perchloroethylene / Tetrachloroethylene
PD	Parkinson's Disease
PHA	Public Health Assessment
POD	Point of Departure
ppb	Parts per Billion
ppm	Parts per Million
RAGS	US EPA's Risk Assessment Guidance for Superfund
RfC	Reference Concentration
RfD	Reference Dose
RME	Reasonable Maximum Exposure
SD	Standard Deviation
SDWA	Safe Drinking Water Act
sRBC	Sheep Red Blood Cells

TCE	Trichloroethylene
TT	Tarawa Terrace
TTD	Target Organ Toxicity Dose
UF_A	Interspecies Uncertainty Factor
UF_D	Database Uncertainty Factor
UF_H	Human Variability Uncertainty Factor
UF_L	Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor
UF_{Schr}	Subchronic to Chronic Uncertainty Factor
UF	Uncertainty Factor
US DOJ	United States Department of Justice
US EPA	United States Environmental Protection Agency
VC	Vapor Concentration
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 on 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, or ingestion) to a particular substance may be associated with potential human health risk, both hazard and exposure (including level, duration, and frequency) 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 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 of *Diane Rothchild v. US* 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 [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 Parkinson's Disease (PD) 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 non-cancer toxicity criteria derived by US EPA are referred to as oral reference doses (RfDs) or inhalation reference concentrations (RfCs). ATSDR's toxicity criteria for evaluating potential non-cancer hazards, derived similarly to US EPA's RfDs/RfCs are referred to as minimal risk levels (MRLs). Non-cancer toxicity criteria are doses or concentrations at or below which adverse health effects are not expected. These criteria are derived based on the most sensitive cancer endpoint evaluated in the available studies, and then are further adjusted to lower doses or concentrations based on uncertainty factors (UFs), such as a UF for use of an animal study instead of a human study, or a UF to account for sensitive individuals in the population (*i.e.*, children or the elderly). RfDs or oral MRLs are described as doses in milligrams per kilogram body weight per day (or mg/kg-day). RfCs and inhalation MRLs are described as inhalation concentrations of chemicals in microgram per cubic meter of air (or $\mu g/m^3$). For example:
    - An RfD (or oral MRL) of 0.01 mg/kg-day is the dose in milligram per kilogram body weight per day (mg/kg-day) of a chemical that is not expected to lead to adverse health effects.
    - An RfC (or inhalation MRL) of 0.01 $\mu g/m^3$ is the inhalation exposure concentration in microgram per cubic meter of air ($\mu g/m^3$) of a chemical that is not expected to lead to adverse health effects.
- 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 μg/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 (AT; equal to the exposure duration for non-cancer risk evaluations), I calculated the plaintiff's average daily doses (ADDs) for oral and dermal chemical exposures and the average daily exposures (ADEs) for inhalation chemical exposures for the plaintiff.
  - I calculated the plaintiff's ADDs as follows:
    - ADD = (DED × EF × ED) ÷ AT, where:

      ADD = 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 (days)
  - I calculated the plaintiff's ADEs as follows:
    - ADE = (DEC × EF × ED) ÷ AT, where:

      ADE = Average Daily Exposure (μg/m^3)

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

      EF = Exposure Frequency (days/year)

      ED = Exposure Duration (years)

      AT = Averaging Time (days)
- In the **risk characterization** step in the risk assessment, the estimated human exposure levels of concern (ADD or ADE, as described above) are combined with the dose-response assessment (toxicity criteria [*e.g.*, RfD or RfC]) for each chemical to calculate non-cancer risk estimates for each chemical and exposure pathway (*i.e.*, ingestion, dermal contact, or inhalation).
  - Non-cancer toxicity criteria are conservative toxicity values used in regulatory risk evaluations as reference doses or exposure concentrations against which a specific chemical dose or exposure concentration can be compared. If the dose or exposure concentration of concern for a particular chemical is equal to or lower than the non-cancer toxicity criterion for that chemical, adverse health effects are not expected (US EPA, 1989).
      - Non-cancer risks (or "hazard quotients" or "HQs") from oral or dermal exposures to a chemical are calculated by dividing the oral or dermal dose of that chemical (ADD) by the chemical-specific RfD (or MRL), as follows:
    - HQ from Oral or Dermal Exposure = ADD (mg/kg-day) ÷ RfD (mg/kg-day)
  - Similarly, non-cancer risks (HQs) from inhalation exposure to a chemical are calculated by dividing the inhalation exposure concentration of that chemical (ADE) by the chemical-specific RfC (or MRL), as follows:

- ▶ HQ from Inhalation Exposure = ADE (μg/m^3) ÷ RfC (μg/m^3)

- After calculating non-cancer HQs from exposure to chemicals *via* each relevant exposure pathway, the hazard index (HI) is derived by summing the HQs across chemicals and exposure pathways. If an HI is less than or equal to US EPA's target HI of 1, adverse health effects are not expected, and there is no need for further evaluation (US EPA, 1989). If an HI is greater than 1, the *potential* for non-cancer health effects from the evaluated exposures requires further evaluation. However, because of the conservative nature of regulatory toxicity criteria, the exceedance of a health-protective RfD or RfC does not mean that adverse health effects will occur or are even likely to occur.

- As an example risk calculation, applying an RfD of 0.01 mg/kg-day to an ADD of 0.005 mg/kg-day would result in the following risk calculation: 0.005 mg/kg-day ÷ 0.01 mg/kg-day = an HQ of 0.5. This HQ is then added to other HQs for other chemicals and pathways to calculate the HI. If the HI falls at or below US EPA's target non-cancer HI of 1, adverse health effects are not expected.

    - If an HI is greater than 1, US EPA recommends segregating the HIs over target organs (US EPA, 1989). This approach is particularly applicable to risk evaluations focused on a specific non-cancer health effect. Since the health effect of concern in this case is PD, which is a neurological endpoint, I have segregated the HIs to estimate a neurological HI summed over all exposure pathways and chemicals of concern.

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 "equipoise and above evidence for causation for TCE and Parkinson disease," and that the evidence for causation was "below equipoise" for exposure to PCE and PD. ATSDR (2017b) provided no comment on whether there is a causal association between benzene, vinyl chloride, or 1,2-tDCE exposure and PD. As discussed in Section 5, US EPA did not conclude that there is an association between exposure to TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE and PD. Dr. Goodman's report concluded that, overall, the currently available toxicology and epidemiology evidence do not support a causal association between TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE exposure and PD (Goodman, 2025).

---

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

Section 5 also summarizes the US EPA toxicity criteria used in the non-cancer risk evaluation for the plaintiff. The TCE toxicity criteria are based on several non-cancer health effects as the most sensitive endpoints. Because the scientific evidence does not support an association between PD and exposure to TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE, the non-cancer toxicity criteria for these chemicals are not based on PD, and therefore are not predictive, and are overly conservative, of PD risk. However, I conservatively apply the criteria for these chemicals to estimate the plaintiff's overall non-cancer risk. In addition, I have calculated non-cancer risks based only on the most sensitive neurological endpoints for each chemical, some of which would not be considered related to PD (*e.g.*, color vision changes). Overall, the toxicity criteria derived to be protective of the most sensitive neurological effects for TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE are considered protective of all neurological effects evaluated for these chemicals, including effects related to PD.

In Section 6, I calculate non-cancer risks based on exposure estimates for Ms. Rothchild. Ms. Rothchild worked as a teacher at Camp Lejeune from August 1972 through December 1974 while living off-base. During the first school year (August 1972-May 1973), Ms. Rothchild taught at the New River Air Station, but starting in August 1973, she taught at Tarawa Terrace. During this time period, she also visited the base for social and recreational activities outside of her teaching hours. For my risk calculations, I used TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE exposure estimates for Ms. Rothchild from tap water (*via* ingestion of drinking water, and *via* dermal and inhalation exposure to school bathroom water while hand washing as well as to vapors while swimming) 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 Ms. Rothchild. Risks were calculated for the following scenarios for the exposure period of concern (approximately 2.4 years) for Ms. Rothchild:

- **Baseline Exposure Pathways:**
  - Drinking Water Ingestion – For this exposure pathway, because it is not clear that the plaintiff's water ingestion occurred from only one of the two water treatment systems, I evaluated two 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; and (2) reasonable maximum exposure (RME), which assumes ingestion of 3.3 L of tap water per day.
  - Dermal and Inhalation Exposures from Hand Washing (TT WTP) – 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 school bathroom facility exposure model (ATSDR, 2024a), and based on the plaintiff's location of work during her time at Camp Lejeune (TT WTP). The dermal doses and inhalation concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Exposures from the school bathroom facility model are estimated based on a mean hand washing duration of 0.61 minutes and a standard deviation of 0.57 minutes, which are model default values (LaKind, 2025).
- **Additional Exposure Pathways:**
  - Inhalation Exposures from Swimming (HP and TT WTPs) – Ms. Rothchild testified that she used a pool while at Camp Lejeune, including while taking sailing lessons. Ms. Rothchild did not specify the location of the pool, or whether the pool was indoors or outdoors. I conservatively evaluated the swimming pathway assuming an indoor pool scenario at both HP and TT WTPs, using air exposure concentrations provided by Dr. LaKind and described in her report (LaKind, 2025).
- **Exposure Scenarios Evaluated for Ms. Rothchild:**

- The CTE exposure scenario includes the following exposure pathways: CTE drinking water ingestion (HP and TT WTPs), CTE dermal and inhalation exposures from handwashing (TT WTP), and inhalation from swimming (HP and TT WTPs).
- The RME exposure scenario includes the following exposure pathways: RME drinking water ingestion (HP and TT WTPs), RME dermal and inhalation exposures from handwashing (TT WTPs), and inhalation from swimming (HP and TT WTPs).

Based on standard risk assessment methodology, which includes overly health-protective assumptions about exposure and risk, risk estimates for Ms. Rothchild do not exceed US EPA's target non-cancer hazard index (HI) of 1 for even the highest exposure assumptions. Further, the maximum total HI (0.3) for TCE (the only chemical for which ATSDR [2017b] considers the evidence to be "equipoise and above for causation" for PD) is well below 1.

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, responses from animal or human studies) before uncertainty factors are applied to derive the toxicity criteria. These comparisons are called margins of exposure (MoE), and are equal to the doses or exposure concentrations that are the basis of the toxicity criteria divided by the plaintiff-specific doses or exposure concentrations. MoEs above 1 provide support that adverse health effects would not be expected for the individual. Based on these comparisons for Ms. Rothchild's exposures, the MoEs range from 200 to 14,000,000,000; all of which are well above 1, providing additional support that Ms. Rothchild's exposures would not have been expected to lead to her PD.

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

Based on the results of my analysis described above, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Ms. Rothchild's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 2.4 years that she worked and spent time at Camp Lejeune are causally associated with her Parkinson's disease.

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 addresses general causation information regarding exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE;
  - Expert report of Dr. LaKind (2025) regarding exposure information for the plaintiff;
  - Expert reports of Drs. Hennet (2024) and Spiliotopoulos (2024) regarding groundwater modeling for Camp Lejeune;
  - Expert reports submitted on behalf of Ms. Rothchild by Drs. Reynolds (2025a) and Andruska (2025);
  - Plaintiff's deposition; and
  - Other Plaintiff materials, if available, as cited within (*e.g.*, declaration, military 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 deposition, employment history, and other materials relevant to the plaintiff's exposure;
- 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 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 *vs*. risk evaluation to assess potential causation.

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 the occurrence of a health effect.
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; assess the magnitude of uncertainty in the 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 PD is a non-cancer health effect, in this section, I have focused the dose-response and risk characterization methodology discussions on non-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 and conservative nature of non-cancer toxicity criteria used in regulatory risk assessments.

3.3.1.1 Derivation of Non-Cancer Toxicity Criteria

Regulatory toxicity criteria for cancer and non-cancer 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 non-cancer toxicity criteria are typically derived. Because the plaintiff was diagnosed with PD, the process for derivation of regulatory non-cancer toxicity criteria is described below.

The non-cancer toxicity criteria used by US EPA are referred to as oral reference doses (RfDs) or inhalation reference concentrations (RfCs). ATSDR's toxicity criteria for evaluating potential non-cancer hazards are referred to as minimal risk levels (MRLs). As described further below, non-cancer toxicity criteria are doses or concentrations at or below which adverse health effects are not expected.

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. RfDs or RfCs (or MRLs) are typically derived by identifying the POD (the dose associated with no, or a very low, response in animal or human studies) on the dose-response curve and applying adjustment factors to the POD to account for potential uncertainties; these adjustment factors are referred to as "uncertainty factors" (or UFs).

US EPA often uses a benchmark dose (BMD) modeling approach (US EPA, 2012a) to develop dose-response curves and PODs for 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 derivation of a non-cancer toxicity criterion (*e.g.*, RfD or RfC) from a POD, based on a BMD/BMDL and application of uncertainty factors. For non-cancer toxicity criteria, the BMDL values are typically associated with a response in the range of 5-10%.

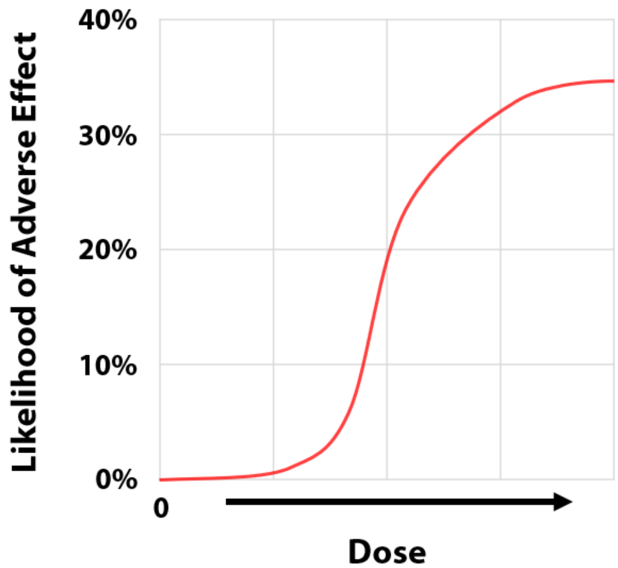

Figure 3.1 Dose Response Curve

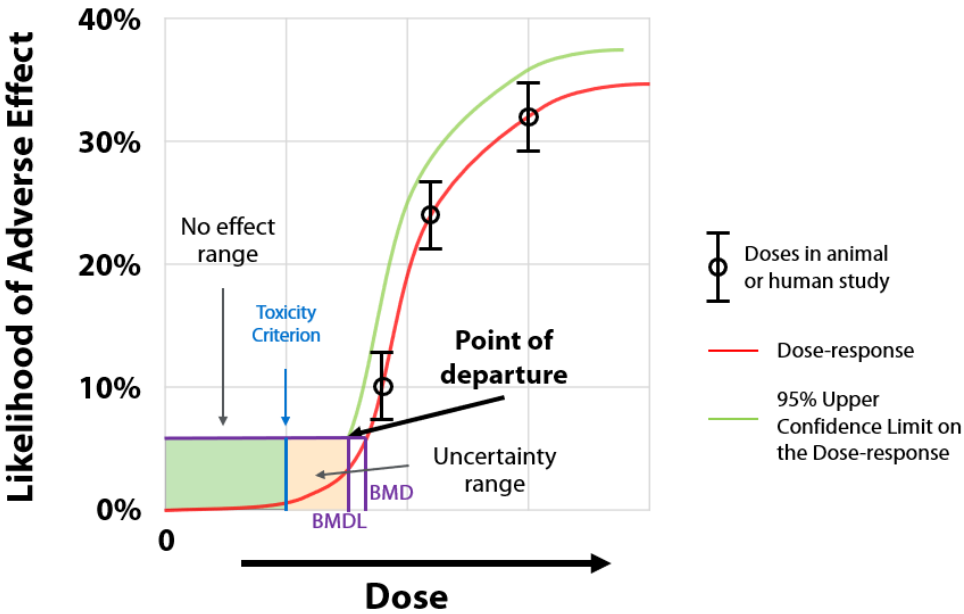

Figure 3.2 Approach for Non-Cancer Toxicity Criterion Development

As described further below, if the dose or exposure concentration of concern for a particular chemical is at or below the non-cancer toxicity criterion for that chemical, adverse health effects would not be expected.

Because regulatory toxicity criteria are derived to be protective of the most sensitive individuals in a population, for non-cancer health effects, regulatory agencies typically apply several UFs to the POD to derive toxicity criteria protective of sensitive individuals (*i.e.*, the POD is divided by the product of all of the UFs combined). For example, if the POD is based on a lowest observed adverse effect level (LOAEL), as opposed to a no observed adverse effect level (NOAEL), regulatory agencies will often apply a UF of 10 to adjust the POD to a value that is considered closer to a NOAEL (UF_L). UFs can also be applied for use of an animal study instead of a human study (interspecies uncertainty factor [UF_A]), and for database uncertainties (database uncertainty factor [UF_D]).

For almost all toxicity criteria derived to be protective of the general population, an additional UF of 3 to 10 is applied to the POD to adjust to a value estimated to be protective of sensitive individuals (*e.g.*, pregnant women and children) (human variability uncertainty factor [UF_H]). Typically, a UF of 10 is applied as a UF_H for the general population. A UF_H of <10 is often used for derivation of occupational exposure limits (OELs) because workers are considered a less sensitive population overall than a population that includes children and elderly. For example, as summarized in a recent review by Schneider *et al*. (2022), occupational regulatory agencies within the European Union (EU) consider worker and general population differences, and apply a UF of 5 or less for human-to-human variability for derivation of occupational exposure limits. Dankovic *et al*. (2015) describes the EU approach for OELs, and several studies that provide support for a 2-fold higher adjustment factor for elderly and diseased individuals compared to a healthy population, providing support for use of a 5-fold, instead of a 10-fold, UF_H for healthy workers.

There are also differences in the exposure duration for health effect studies that need to be considered in derivation of toxicity criteria. Chronic studies are considered to be exposures of >7 years (>10% of a lifetime) to a lifetime (70 years) (US EPA, 2021). Subchronic studies are considered to be 10% or less of a lifetime of exposure (≤7 years) (US EPA, 2021). If a subchronic study is used as the basis of derivation of a chronic toxicity criterion, regulatory agencies will often apply a UF of 10 to estimate an exposure concentration protective of a chronic exposure duration (subchronic to chronic uncertainty factor [UF_{Schr}]). When subchronic exposures are of concern, it is important to consider whether the toxicity criterion applied in the risk calculation includes a 10-fold adjustment for a longer chronic exposure.

Typically, regulatory agencies will not apply a total UF of more than 3000 for derivation of toxicity criteria (US EPA, 2002a). However, given that a total UF can be this high, to provide adequate perspective on potential causal relationships, any risk calculation that is used to estimate potential health risks for an individual should consider the total UF and how much lower the resulting toxicity criterion is compared to the POD from the original human or animal study. For example, let us assume that a NOAEL of 10 mg/kg-day from a subchronic human study has been chosen as the POD for derivation of a toxicity criterion. The POD would then be divided by the product of several UFs, including a UF_H of 10 for protection of sensitive individuals, a UF_{Schr} of 10 for use of a subchronic study for derivation of a chronic value, and possibly a UF_D of 10 for uncertainties in the database, for a total UF of 1000. The resulting toxicity value would be 0.01 mg/kg-day (10 mg/kg-day ÷ 1000 = 0.01 mg/kg-day). In a risk calculation, if the exposure of concern is 0.1 mg/kg-day, that dose will exceed the toxicity criterion by 10-fold, but will still be 100-fold lower than the NOAEL that was observed in the study. Therefore, when interpreting risk estimates that exceed regulatory risk limits, it is important to consider how the exposure of concern compares to the exposure estimates from the study that is the basis of the toxicity values. It is important to also consider if the exposure of concern may have been subchronic and whether the exposures were in a healthy population; if these are true, the UF_H and the UF_{Schr} are overly protective and result in a toxicity value that may be orders of magnitude lower than what is protective of the population (or individual) that is being evaluated.

Further, for some chemicals for which there is only reliable observational information (*i.e.*, a human or animal study) to derive either an RfD or RfC, US EPA might conduct what is called a "route-to-route extrapolation" and derive an RfC from and RfD, 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. 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 for non-cancer risk evaluations is equal to the exposure duration (US EPA, 1989).

For evaluating oral and dermal exposures for non-cancer risk estimates, the relevant dose metric is the average daily dose (ADD), which is defined as the amount of a chemical taken into the body *via* oral or dermal exposure during the exposure duration, averaged over the exposure period. Using the DED estimates from Dr. LaKind, I calculate ADDs for oral and dermal exposures to the chemicals of interest as follows:

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

where:

ADD	=	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 (days)

For evaluating inhalation exposures for non-cancer risk estimates, the relevant dose metric is the average daily exposure (ADE), which is defined as the amount of chemical that someone is exposed to *via* inhalation during the exposure duration, averaged over the exposure period. Using the DEC estimates from Dr. LaKind, I calculate ADEs for inhalation exposures to the chemicals of interest as follows:

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

where:

ADE	=	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 (days)

3.3.2.1 Calculations for the Indoor Swimming Inhalation Exposure Pathway

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

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

where:

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

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

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

where:

ADE	=	Average Exposure Concentration (μg/m^3)
DEC	=	Daily Exposure Concentration (μg/m^3)
EF	=	Exposure Frequency (days/event)
EV	=	Events During Exposure Duration (number of events)
AT	=	Averaging Time (days)

See Appendix D for details on this calculation.

3.3.3 Risk Characterization for Non-Cancer Health Effects

Per US EPA (1989) guidance, non-cancer risk (or the "hazard quotient" or "HQ") from oral exposure to a chemical is calculated by dividing the oral dose of that chemical by the chemical-specific RfD (or oral MRL), as follows:

$$\text{Hazard quotient} = ADD\ (\frac{mg}{kg-d})\ \div RfD\ (\frac{mg}{kg-d})$$

Similarly, per US EPA (1989), the HQ from inhalation exposure to a chemical is calculated by dividing the inhalation concentration of that chemical by the chemical-specific RfC (or inhalation MRL), as follows:

$$\text{Hazard quotient} = ADE\ \left(\frac{\mu g}{m^3}\right)\ \div RfC\ \left(\frac{\mu g}{m^3}\right)$$

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).

After calculating non-cancer HQs from exposure to chemicals *via* each relevant exposure pathway, the hazard index (HI) is derived by summing the HQs across chemicals and exposure pathways. If an HI is less than or equal to US EPA's target HI of 1, adverse health effects are not expected, and there is no need for further evaluation. If an HI is greater than 1, the *potential* for non-cancer health effects from the evaluated exposures requires further evaluation. However, because of the conservative nature of regulatory toxicity criteria, the exceedance of a health-protective RfD or RfC does not mean that adverse health effects will occur or are even likely to occur. See further discussion below.

If the HI is greater than 1, US EPA recommends segregating the HIs over target organs (US EPA, 1989). This approach is particularly applicable to risk evaluations that are focused on a specific health effect. Since the health effect of concern in this case is PD, which is a neurological endpoint, I have segregated the HIs to estimate a neurological HI summed over all exposure pathways and chemicals of concern. As described further in Section 5, there are toxicity criteria based on neurological effects for some of the chemicals (TCE, PCE, benzene, and 1,2-tDCE). Because exposure to the majority of these chemicals has not been found to be related to PD, the neurological toxicity criteria for most of these chemicals are not related to PD (*e.g.*, the TCE inhalation toxicity criterion is based on wakefulness). However, since toxicity criteria are derived based on the most sensitive endpoint, the neurological toxicity criteria are considered protective of other potential neurological health effects, including PD.

3.3.3.1 Interpretation of Hazard Quotients

Given the conservative nature of these toxicity criteria (often at least an order of magnitude lower than the exposure concentration in the animal or human studies that are used as the basis of the toxicity criteria), an exceedance of a chronic toxicity value such as a chronic RfC, RfD, or MRL (or an HQ greater than 1), does not indicate that any one individual is at elevated risk, as described in the example above. That is to say, these chronic toxicity values include uncertainty factors and assumptions of continuous exposures, which result in concentrations often well below those where adverse effects have been observed, and therefore, are not intended to be a strict level above which toxic effects will definitely occur and below which no effects will occur. However, given the highly conservative nature of the UFs that are applied in derivation of toxicity criteria, and that the toxicity values are derived to be protective of the most sensitive populations, health effects are unlikely to occur at exposure concentrations equal to or below these toxicity criteria.

ATSDR emphasizes this point when describing its MRLs:

> An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. These substance specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. **It is important to note that MRLs are not intended to define cleanup or action levels for ATSDR or other Agencies…** (ATSDR, 2018a [**emphasis in original**]).

ATSDR also states with respect to MRLs derived from animal studies that:

> "the resulting MRL may be as much as a hundredfold below levels shown to be nontoxic in laboratory animals" (ATSDR, 2018a).

And, further that:

> "**If someone is exposed to an amount above the MRLs, it does not mean that health problems will happen**. When health assessors find exposures higher than the MRLs, it means that they may want to look more closely at a site" (ATSDR, 2018b [**emphasis added**]).

3.4 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 over-prediction 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). 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, 2002a; 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 limit, 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.

3.5 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 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 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:

$$\text{MoE} = \frac{\text{POD for the Non-Cancer Toxicity Value}}{\text{Individual ADD or ADE}}$$

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 systems (ATSDR, 2013a).

Operations at Camp Lejeune started in late 1941. Multiple water treatment plants (WTPs)[2] 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.*,

[2] 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).[3]

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).[4] 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).[5]

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).

---

[3] 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).

[4] 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).

[5] 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 PD. In addition, I summarize the toxicity criteria for TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE that are applied in the Plaintiff-specific risk evaluation (Section 6). The toxicity criteria I describe are: (1) those derived by US EPA and ATSDR that are based on the most sensitive non-cancer endpoint, and (2) neurological toxicity criteria derived by ATSDR in its Public Health Assessment (PHA) for Camp Lejeune drinking water (ATSDR, 2017a). Note that none of the endpoints for the toxicity criteria are for PD.

5.1 Hazard Assessments

5.1.1 Trichloroethylene (TCE)

To understand the potential association between TCE exposure and PD, I reviewed the expert report prepared by Dr. Goodman (2025). In addition, I reviewed the overall conclusions from ATSDR (2019a) and US EPA (2011a, 2020a) TCE toxicological reports; none of the regulatory agency documents concluded that TCE exposure is a known cause of PD. In its assessment of the evidence regarding drinking water contaminants at Camp Lejeune, ATSDR concluded that there is "equipoise and above evidence for causation for TCE and Parkinson disease" (ATSDR, 2017b).

Based on the available epidemiology studies and agency reviews that evaluated TCE exposure and PD, Dr. Goodman concluded that, "currently available epidemiology evidence does not support a causal association between TCE exposure and PD" (Goodman, 2025). Based on animal bioassays, Dr. Goodman concluded that, "as a whole, evidence from experimental animal studies does not support a causal relationship between TCE exposure and PD in humans" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology and toxicology studies that evaluated potential associations between TCE exposure and PD concluded that overall, "as a whole, the currently available evidence does not support a causal association between TCE exposure and PD" (Goodman, 2025).

5.1.2 Tetrachloroethylene (PCE)

To understand the potential association between PCE exposure and PD, I reviewed the expert report prepared by Dr. Goodman (Goodman, 2025). In addition, I reviewed the overall conclusions from US EPA (2012b, 2020b) and ATSDR (2017b, 2019b) PCE toxicological reports; none of the regulatory agency documents concluded that PCE exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) concluded "below equipoise evidence for causation" for exposure to PCE and PD. ATSDR noted that their conclusion was based on "very limited" epidemiology evidence and no supportive mechanistic evidence for PCE (ATSDR, 2017b).

Based on the available epidemiology studies and agency reviews that evaluated PCE exposure and PD, Dr. Goodman concluded that, "currently available epidemiology evidence does not support a causal association between PCE exposure and PD" (Goodman, 2025). Dr. Goodman noted that PCE exposure and PD has not been evaluated in animal studies, and concluded that, "suggestions that a common metabolite of PCE and TCE may cause PD are not supported by the available evidence" (Goodman, 2025). In summary, Dr. Goodman's review of the epidemiology studies that evaluated potential associations between PCE exposure and PD concluded that, "as a whole, the currently available evidence does not support a causal association between PCE exposure and PD" (Goodman, 2025).

5.1.3 Benzene

To understand the potential association between benzene exposure and PD, I reviewed the expert report prepared by Dr. Goodman (Goodman, 2025). In addition, I reviewed the overall conclusions from agency benzene toxicological reports (ATSDR, 2007a, 2015, 2017b; US EPA, 2002b). None of the regulatory agency documents concluded that benzene exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) did not comment on whether there is a causal association between exposure to benzene and PD.

Based on the available epidemiology studies and agency reviews that evaluated benzene exposure and PD, Dr. Goodman concluded that, "epidemiology evidence does not support a causal association between benzene exposure and PD" (Goodman, 2025). Dr. Goodman noted that benzene exposure and PD has not been evaluated in animal studies. In summary, Dr. Goodman's review of the epidemiology studies that evaluated potential associations between benzene exposure and PD concluded that, "as a whole, the currently available evidence does not support a causal association between benzene exposure and PD" (Goodman, 2025).

5.1.4 Vinyl Chloride

To understand the potential association between vinyl chloride exposure and PD, I reviewed the expert report prepared by Dr. Goodman (Goodman, 2025). In addition, I reviewed the overall conclusions from US EPA (2003) and ATSDR (2024b) vinyl chloride toxicological reports; neither agency concluded that vinyl chloride exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) did not comment on whether there is an association between vinyl chloride exposure and PD.

Based on the available epidemiology studies and agency reviews that evaluated benzene exposure and vinyl chloride, Dr. Goodman concluded that, "currently available epidemiology evidence does not support a causal association between vinyl chloride exposure and PD" (Goodman, 2025). Dr. Goodman noted that vinyl chloride exposure and PD has not been evaluated in animal studies. In summary, Dr. Goodman's review of the epidemiology studies that evaluated potential associations between vinyl chloride exposure and PD concluded that "as a whole, the currently available evidence does not support a causal association between vinyl chloride exposure and PD" (Goodman, 2025).

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

To understand the potential association between 1,2-tDCE exposure and PD, I reviewed the expert report prepared by Dr. Goodman (2025). Dr. Goodman concluded that, "currently available scientific evidence is too limited to address whether there is a causal association between trans-1,2-DCE exposure and PD" (Goodman, 2025). In addition, I reviewed the overall conclusions from US EPA (2010b) and ATSDR (2023) 1,2-tDCE toxicological reports; neither agency concluded that 1,2-tDCE exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) did not comment on whether there is an association between exposure to 1,2-tDCE and PD.

5.2 Toxicity Criteria

This section summarizes the non-cancer toxicity criteria that US EPA derived for TCE, PCE, benzene, vinyl chloride, and 1,2-t-DCE based on the methodology described in Section 3, and US EPA's hazard assessment of these chemicals as described in the documents cited below. As described in Section 3, non-cancer toxicity criteria are derived based on the most sensitive endpoint identified by the regulatory agency deriving the value; none of these toxicity criteria are directly relevant to PD. In its PHA for Camp Lejeune, ATSDR also derived non-cancer TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE toxicity criteria for several health effects other than the most sensitive, including for neurological effects (ATSDR, 2017b); these values are also described below.

5.2.1 Trichloroethylene (TCE)

5.2.1.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.1 summarizes the points of departure (PODs), total uncertainty factors (UFs), candidate RfDs and effects associated with each, and the final RfD US EPA derived for TCE (US EPA, 2011b). Table 5.2 summarizes the PODs, UFs, candidate RfCs and effects associated with each, and the final RfC US EPA derived for TCE (US EPA, 2011b).

Based on the hazard assessment for TCE, US EPA derived three candidate RfDs from three rodent TCE drinking water studies (US EPA, 2011b); one based on fetal heart malformations in rats (Johnson *et al*., 2003); one based on altered immune system endpoints in mice (Peden-Adams *et al*., 2006); and one based on decreased thymus weight in mice (Keil *et al*., 2009). US EPA used the average of these candidate RfDs as the final RfD (Table 5.1). US EPA then applied a TCE physiologically-based pharmacokinetic (PBPK) model to conduct a route-to-route (oral-to-inhalation) extrapolation to derive the TCE RfCs from two studies used for the TCE RfD derivations (US EPA, 2011b) (Table 5.2).

Table 5.1 US EPA TCE Non-Cancer Chronic Oral Toxicity Values

Chemical	POD mg/kg-d	UF	Candidate RfD (mg/kg-d)	RfD (mg/kg-d)	Effect/Source
TCE	0.0051	10[a]	0.00051	0.0005	Fetal cardiac abnormalities in rats (Johnson *et al.*, 2003).
	0.37	1000[b]	0.00037		Altered immune system in mice (Peden-Adams *et al.*, 2006).
	0.048	100[c]	0.00048		Decreased thymus weight in adult female mice (Keil *et al.*, 2009).

Notes:

LOAEL = Lowest Observed Adverse Effect Level; mg/kg-d = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; TCE = Trichloroethylene; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.

(a) A UF_A of 3 was applied to account for interspecies toxicodynamic differences, and a UF of 3 was applied to account for differences in sensitive populations, for a total UF of 10.

(b) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 10 was applied to account for interspecies toxicokinetic and toxicodynamic differences, and a UF of 10 was applied to account for differences in sensitive populations, for a total UF of 1,000.

(c) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 3 was applied to account for interspecies toxicodynamic uncertainty, and a UF_H of 3 was applied to account for differences in sensitive populations, for a total UF of 100.

Source: US EPA (2011a,b).

Table 5.2 US EPA TCE Non-Cancer Chronic Inhalation Toxicity Values

Chemical	POD mg/m³ (ppm)	UF	Candidate RfC µg/m³ (ppb)	RfC µg/m³ (ppb)	Effect/Source
TCE	0.021 (0.0037)	10[a]	2.1 (0.37)	2 (0.4)	Increased fetal cardiac malformations in rats (Johnson *et al.*, 2003)
	0.19 (0.033)	100[b]	1.9 (0.33)		Decreased thymus weight in adult female mice (Keil *et al.*, 2009)

Notes:

LOAEL = Lowest Observed Adverse Effect Level; $\mu g/m^3$ = Microgram per Meter Cubed; mg/m^3 = Milligram per Meter Cubed; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; RfC = Reference Concentration; TCE = Trichloroethylene; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.

(a) A UF_A of 3 was applied to account for interspecies toxicodynamic differences, and a UF of 3 was applied to account for differences in sensitive populations, for a total UF of 10.

(b) A UF_L of 10 was applied for the use of a LOAEL, a UF_A of 3 was applied to account for interspecies toxicodynamic uncertainty, and a UF_H of 3 was applied to account for differences in sensitive populations, for a total UF of 100.

Source: US EPA (2011a,b).

5.2.1.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for TCE (ATSDR, 2019a) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for TCE and the TCE toxicity values applied in the PHA for evaluation of human health risk. In its 2019 toxicological profile for TCE, and in the PHA (ATSDR, 2017a), ATSDR adopted the US EPA chronic RfD and values for TCE (described above) as the MRLs for oral and inhalation exposures, respectively, for both chronic and subchronic (intermediate) exposure durations.

In addition to the RfD, RfC, and MRL values for TCE, the ATSDR PHA also derived specific target organ toxicity doses (TTDs) for several endpoints, including neurological effects, *via* oral and inhalation routes of exposure (ATSDR, 2017a). The oral and inhalation TTDs for neurological effects are summarized in Table 5.3. Although the PHA (ATSDR, 2017a) provides information on the effect, it does not specifically describe the derivation of the TTDs nor does it report the underlying studies that provide the basis of the values. However, based on my review of the ATSDR toxicological profile for TCE (2019a) and US EPA's toxicological review for TCE (US EPA, 2011a), it is likely that the ATSDR PHA relied on the studies of Gash *et al*. (2008) and Arito *et al*. (1994) to determine TTDs for neurological effects of TCE *via* oral and inhalation routes of exposure (Table 5.3).

Table 5.3 ATSDR TCE TTDs for Neurological Effects *via* the Oral and Inhalation Routes of Exposure

Chemical	POD mg/kg-d	POD μg/m³	UF	TTD_{neuro}	Effect / *Likely* Source
TCE	1000	-	1,000[a]	1 mg/kg-d	Subchronic oral study observed decreased dopaminergic neurons in rats (Gash *et al.*, 2008).
	-	63,930[b]	1,000[b]	64 μg/m³ (11.9 ppb)	Subchronic oral study observed decreased wakefulness in rats (Arito *et al.*, 1994).[b]

Notes:

ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; μg/m³ = Microgram per Meter Cubed; mg/kg-d = Milligram per Kilogram Body Weight per Day; mg/m³ = Milligram per Meter Cubed; PHA = Public Health Assessment; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; TCE = Trichloroethylene; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.

(a) Although not discussed in the ATSDR PHA (ATSDR, 2017a), ATSDR (2019a) provides a LOAEL for Gash *et al.* (2008) of 1,000 mg/kg-d. The UFs are not discussed in either document.

(b) Although not discussed in the ATSDR PHA (ATSDR, 2017a), ATSDR (2019a) and US EPA (2011a) provide a LOAEL for Arito *et al.* (1994) of 50 ppm which is equivalent to 269 mg/m³ (1 ppm TCE = 5.37 mg/m³). Based on an exposure of 8 hours per day, 5 days/week for 6 weeks, US EPA calculated a continuous human equivalent concentration of ~64,000 μg/m³. The composite UF of 1000 described by US EPA (2011a) is as follows: UF_{Schr} = 3 for subchronic to chronic uncertainty; UF_A = 3 for animal to human uncertainty; UF_H = 10 for human variability; UF_L = 10 for use of a LOAEL).

Source: ATSDR (2017a).

For the toxicity value of 64 μg/m³, also described by US EPA in its toxicological review for TCE, US EPA (2011a) included a UF of 3 to adjust from a subchronic study to a chronic exposure duration. Therefore, I have removed that UF to adjust the value to reflect a subchronic exposure duration (64 μg/m³ × 3), resulting in subchronic TCE toxicity criterion of 192 μg/m³ (0.036 parts per million [ppm]) that can be applied for subchronic exposure durations (*i.e.*, less than 7 years of exposure per US EPA guidelines) for neurological effects.

5.2.1.3 TCE Toxicity Criteria Applied in the Risk Calculations

Since US EPA's and ATSDR's toxicity values for TCE (that are based on the most sensitive endpoints) are not based on neurological effects, I applied ATSDR's neurological TTDs for TCE risk calculations in this report. It is notable that although the endpoints are neurological, they are not necessarily related to PD (*e.g.*, the inhalation value is based on wakefulness). However, since toxicity criteria are derived to be protective of the most sensitive endpoint, the neurological effect toxicity criteria are considered protective of all neurological effects evaluated for TCE, including effects related to PD. The toxicity criteria used in the TCE risk calculations are summarized in Table 5.4.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

Table 5.4 TCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
TCE	Oral TTD	Chronic/Subchronic	Neurological	1 mg/kg-d
	Inhalation TTD	Chronic	Neurological	64 µg/m³ (11.9 ppb)
	Inhalation TTD	Subchronic	Neurological	192 µg/m³ (36 ppb)

Notes:
µg/m³ = Microgram per Meter Cubed; mg/kg-d = Milligram per Kilogram Body Weight per Day; ppb = Parts per Billion; TCE = Trichloroethylene; TTD = Target Organ Toxicity Dose.

5.2.2 Tetrachloroethylene (PCE)

5.2.2.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.5 summarizes the PODs, total UFs, candidate RfDs and effects associated with each, and the final RfD US EPA derived for PCE (US EPA, 2012b). Table 5.6 summarizes the PODs, UFs, candidate RfCs and effects associated with each, and the final RfC US EPA derived for PCE (US EPA, 2012b).

Based on the hazard assessment for PCE, US EPA first derived two candidate RfCs from two human PCE studies (US EPA, 2012b); one based on reaction time and cognitive effects from worker inhalation exposures (Echeverria *et al*., 1995), and one based on color vision changes from worker inhalation exposures (Cavalleri *et al*., 1994) (Table 5.6). US EPA then applied a PCE PBPK model to conduct a route-to-route (inhalation-to-oral) extrapolation to derive the PCE RfDs from the same studies used for the PCE RfC derivations (Table 5.5).

Table 5.5 US EPA PCE Non-Cancer Chronic Oral Toxicity Values

Chemical	POD (mg/kg-d)	UF	Candidate RfD (mg/kg-d)	RfD (mg/kg-d)	Effect/Source
PCE	9.7	1,000[a]	0.0097	0.006	Reaction time, cognitive effects in occupationally-exposed adults (Echeverria *et al*., 1995).
	2.6	1,000[a]	0.0026		Color vision changes in occupationally exposed adults (Cavalleri *et al*., 1994).

Notes:
LOAEL = Lowest Observed Adverse Effect Level; mg/kg-d = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
(a) A total UF of 1,000 was used for both candidate RfDs and comprised a UF_H of 10 for human variability, a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, and a UF_D of 10 for database uncertainty.
Source: US EPA (2012b).

Table 5.6 US EPA PCE Non-Cancer Chronic Inhalation Toxicity Values

Chemical	POD (mg/m^3)	UF	Candidate RfC µg/m^3 (ppb)	RfC µg/m^3 (ppb)	Effect/Source
PCE	56	1,000[a]	56 (8)	40 (6)	Reaction time, cognitive effects in occupationally-exposed adults (Echeverria *et al.*, 1995).
	15	1,000[a]	15 (2)		Color vision changes in occupationally exposed adults (Cavalleri *et al.*, 1994).

Notes:
LOAEL = Lowest Observed Adverse Effect Level; µg/m^3 = Microgram per Meter Cubed; mg/m^3 = Milligram per Meter Cubed; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; US EPA = United States Environmental Protection Agency.
(a) A total UF of 1,000 was used for both candidate RfCs and comprised a UF_H of 10 for human variability, a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, and a UF_D of 10 for database uncertainty.
Source: US EPA (2012b).

5.2.2.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for PCE (ATSDR, 2019b) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for PCE and the PCE toxicity values applied in the PHA for evaluation of human health risk. In its 2019 toxicological profile for PCE, and in the PHA (ATSDR, 2017a), ATSDR derived 0.008 mg/kg-day and 0.04 mg/m^3 (0.006 ppm) values for PCE as the MRLs for oral and inhalation exposures, respectively, for both chronic and subchronic (intermediate) exposure durations, based on the same study that US EPA used (Cavelleri *et al*., 1994). The oral MRL is similar to US EPA's RfD and the inhalation MRL is the same as US EPA's RfC. Table 5.7 summarizes the PCE oral MRL.

Table 5.7 ATSDR PCE Non-Cancer Oral Toxicity Values

Chemical	Exposure Duration	POD (mg/kg-d)	Combined UF	MRL (mg/kg-d)	Effect/Source
PCE	Intermediate	2.3	300[a]	0.008	Color vision changes in occupationally exposed adults (Cavalleri *et al.*, 1994).
	Chronic				

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; PCE = Tetrachloroethylene; POD = Point of Departure; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor.
Source: ATSDR (2019b).
(a) ATSDR applied a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, a UF_H of 10 for human variability, and a UF_D of 3 for database uncertainty.
Source: ATSDR (2019b).

Note that ATSDR reports neurological effect TTDs for PCE in its PHA for Camp Lejeune (ATSDR, 2017a) that are equal to the US EPA RfD and RfC for PCE.

5.2.2.3 PCE Toxicity Criteria Applied in the Risk Calculations

The toxicity criteria used in the PCE risk calculations are summarized in Table 5.8. It is notable that although the endpoints are neurological, they are not necessarily related to PD (*e.g.*, one of the oral values is based on color vision changes). However, since the endpoints are based on the most sensitive endpoints from the most reliable animal and human studies, they are considered protective of other health effects, including PD.

Table 5.8 PCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
PCE	Oral RfD	Chronic/Subchronic	Neurological	0.006 mg/kg-d
	Inhalation RfC	Chronic/Subchronic	Neurological	40 μg/m^3 (6 ppb)

Notes:

μg/m^3 = Microgram per Meter Cubed; mg/kg-d = Milligram per Kilogram Body Weight per Day; PCE = Tetrachloroethylene; ppb = Parts per Billion; RfC = Reference Concentration; RfD = Reference Dose.

5.2.3 Benzene

5.2.3.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.9 summarizes the POD, total UF, associated health effects, and the final RfD US EPA derived for benzene (US EPA, 2002b). Table 5.10 summarizes the POD, total UF, and the final RfC US EPA derived for benzene (US EPA, 2002b). Based on the hazard assessment for benzene, US EPA (2002b) derived an RfC for benzene based on decreased absolute lymphocyte count (ALC) in workers following chronic inhalation exposure to benzene (Rothman *et al*., 1996) (Table 5.10). US EPA then conducted an inhalation-to-oral extrapolation to derive a benzene RfD from the same study used to derive the benzene RfC (Table 5.9).

Table 5.9 US EPA Benzene Non-Cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-d)	UF	RfD (mg/kg-d)	Effect/Source
Benzene	1.2	300[a]	0.004	Decreased absolute lymphocyte count (ALC) in a human occupational study (Rothman *et al.*, 1996)

Notes:

LOAEL = Lowest Observed Adverse Effect Level; mg/kg-day = Milligram per Kilogram Body Weight per Day; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.

(a) US EPA applied a UF_L of 3 for extrapolation from a LOAEL to NOAEL, a UF_H of 10 for human variability, a UF_{Schr} of 3 for subchronic to chronic exposure, and a UF_D of 3 for database uncertainty.

Source: US EPA (2002b).

Table 5.10 US EPA Benzene Non-Cancer Chronic Inhalation Toxicity Value

Chemical	POD µg/m³ (ppb)	UF	RfC µg/m³(ppb)	Effect/Source
Benzene	8,200 (2,600)	300[a]	30 (9)	Decreased absolute lymphocyte (ALC) count in a human occupational study (Rothman *et al.*, 1996)

Notes:
µg/m³ = Microgram per Cubic Meter; LOAEL = Lowest Observed Adverse Effect Level; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Source: US EPA (2002b).
(a) US EPA applied a UF_L of 3 for extrapolation from a LOAEL to NOAEL, a UF_H of 10 for human variability, a UF_{Schr} of 3 for subchronic to chronic exposure, and a UF_D of 3 for database uncertainty.

5.2.3.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for benzene (ATSDR, 2007a) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for benzene and the benzene toxicity values applied in the PHA for evaluation of human health risk. Tables 5.11 and 5.12 summarize the PODs, total UFs, and MRLs derive by ATSDR for benzene.

ATSDR (2007a) derived a chronic oral MRL for benzene based on the chronic occupational inhalation study conducted by Lan *et al*. (2004) (see discussion below) and conducting an inhalation-to-oral extrapolation (Table 5.11). ATSDR (2007a) derived an intermediate inhalation MRL for benzene based on delayed immune response effects in mice following inhalation exposure to benzene (Rosenthal and Snyder, 1987), and derived a chronic inhalation MRL for benzene based on decreased B cell counts in a study of occupationally exposed workers (Lan *et al*., 2004) (Table 5.12).

Table 5.11 ATSDR Benzene Non-Cancer Oral Toxicity Value

Chemical	Exposure Duration	POD (mg/kg-d)	UF	MRL (mg/kg-d)	Effect/Source
Benzene	Chronic	0.014	30[a]	0.0005	B-cell count in occupationally exposed workers (Lan *et al.*, 2004)

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-d = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; POD = Point of Departure; UF = Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.
Source: ATSDR (2007a).
(a) ATSDR (2007a) applied a UF_H of 10 for human variability and a UF_D of 3 for the uncertainty in route-to-route extrapolation, for a total UF of 30.

Table 5.12 ATSDR Benzene Non-Cancer Inhalation Toxicity Values

Chemical	Exposure Duration	POD mg/m³; (ppb)	UF	MRL µg/m³ (ppb)	Effect/Source
Benzene	Intermediate	5.8 (1.8)	300[a]	20 (6)	Immune effects in mice (Rosenthal and Snyder, 1987)
	Chronic	0.096 (0.03)	10[b]	9.6 (3)	B-cell counts in occupationally exposed workers (Lan *et al.*, 2004)

Notes:
µg/m³ = Microgram per Cubic Meter; ATSDR = Agency for Toxic Substances and Disease Registry; LOAEL = Lowest Observed Adverse Effect Level; mg/m³ = Milligram per Cubic Meter; MRL = Minimal Risk Level; NOAEL = No Observed Adverse Effect Level; POD = Point of Departure; ppb = Parts per Billion; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_L = Lowest Observed Adverse Effect Level to No Observed Adverse Effect Level Uncertainty Factor.
Source: ATSDR (2007a).
(a) ATSDR (2007a) applied a UF_L of 10 for extrapolation from a LOAEL to a NOAEL, a UF_A of 3 for extrapolation from animals to humans, and a UF_H of 10 for human variability.
(b) ATSDR (2007a) applied a UF_H of 10 for human variability.

In addition to the RfD, RfC, and MRL values for benzene, in its PHA for Camp Lejeune drinking water, ATSDR derived a TTD for neurological effects *via* the oral route of exposure (ATSDR, 2017a). The POD, total UF, and oral TTD for neurological effects for benzene is summarized in Table 5.13. The PHA (ATSDR, 2017a), however, does not specifically describe the derivation of the TTD, nor does it report the underlying study that provides the basis for this value. However, the POD is generally consistent with the lowest intermediate exposure NOAEL (no observed adverse effect level) (8 mg/kg-day) and LOAEL (lowest adverse effect level) (8 mg/kg-day) for neurological effects reported in mice in the benzene toxicological profile (ATSDR, 2007a [Table 3-2]). ATSDR did not derive an inhalation TTD for neurological effects for benzene.

Table 5.13 ATSDR Benzene TTD for Neurological Effects *via* the Oral Route of Exposure

Chemical	POD (mg/kg-d)	UF	TTD_{neuro} (mg/kg-d)	Effect	Source
Benzene	15	100[a]	0.15	Not provided	Not provided

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-d = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor.
(a) ATSDR did not discuss the basis of the UFs.
Source: ATSDR (2017a).

5.2.3.3 Benzene Toxicity Criteria Applied in the Risk Calculations for the Plaintiff

Since US EPA's and ATSDR's toxicity values for benzene (that are based on the most sensitive endpoints) are not based on neurological effects, I applied ATSDR's neurological TTD for benzene risk calculations in this report (*i.e.*, for the oral route of exposure only since an inhalation value was not provided). Although the endpoint is reported to be neurological, based on the discussion in Section 5.1.3, it is unlikely to be related to PD. However, the value should be considered protective of other neurological health effects, including PD. The toxicity criteria used in the benzene non-cancer risk calculations are summarized in Table 5.14.

Table 5.14 Benzene Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
Benzene	Oral TTD	Chronic/Subchronic	Neurological	0.15 mg/kg-d
	Inhalation MRL	Chronic	B-cell counts	9.6 µg/m^3
	Inhalation MRL	Subchronic	Immune effects	20 µg/m^3

Notes:
µg/m^3 = Microgram per Meter Cubed; mg/kg-d = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; TTD = Target Organ Toxicity Dose.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

5.2.4 Vinyl Chloride

5.2.4.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.15 summarizes the PODs, total UF, associated health effects, and the final RfD US EPA derived for vinyl chloride (US EPA, 2003). Table 5.16 summarizes the PODs, total UFs, associated health effects, and the final RfC US EPA derived for vinyl chloride (US EPA, 2003).

Based on the hazard assessment for vinyl chloride, US EPA (2003) derived an RfD for vinyl chloride based on liver polymorphism effects in rats following chronic exposure to vinyl chloride in the diet (Til *et al*., 1982; US EPA, 2003) (Table 5.15). US EPA then applied a vinyl chloride PBPK model to conduct a route-to-route (oral-to-inhalation) extrapolation to derive the vinyl chloride RfC from the same study used for the vinyl chloride RfC derivation (US EPA, 2003) (Table 5.16).

Table 5.15 US EPA Vinyl Chloride Non-Cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-d)	UF	RfD (mg/kg-d)	Effect/Source
Vinyl chloride	0.09	30[a]	0.003	Liver cell polymorphism in rat chronic feed study (Til *et al.*, 1982, US EPA, 200)

Notes:
mg/kg-d = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; US EPA = United States Environmental Protection Agency.
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.
Source: US EPA (2003).

Table 5.16 US EPA Vinyl Chloride Non-Cancer Chronic Inhalation Toxicity Value

Chemical	POD µg/m³ (ppb)	UF	RfC µg/m³ (ppb)	Effect/Source
Vinyl chloride	2,500 (~100)	30[a]	100 (~39)	Liver cell polymorphism in rat chronic feed study (Til *et al.*, 1982; US EPA, 2003)

Notes:
µg/m³ = Microgram per Meter Cubed; POD = Point of Departure; ppb = Parts per Billion; RfC = Reference Concentration; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; US EPA = United States Environmental Protection Agency.
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.
Source: US EPA (2003).

5.2.4.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for vinyl chloride (ATSDR, 2024b) and its PHA for Camp Lejeune drinking water (ATSDR, 2017a) to identify the current ATSDR MRLs for vinyl chloride and the vinyl chloride toxicity values applied in the PHA for evaluation of human health risk. In its 2024 toxicological profile for vinyl chloride, ATSDR (2024b) concluded that there were insufficient data for vinyl chloride for the derivation of an intermediate-duration oral MRL or for derivation of a chronic-duration inhalation MRL for vinyl chloride. ATSDR (2024b) derived a chronic-duration oral MRL for vinyl chloride, equal to the US EPA RfD, and derived the same way as the US EPA RfD, from the same studies (see above).

ATSDR (2024b) also derived an intermediate-duration inhalation MRL for vinyl chloride based on increased incidence of centrilobular hypertrophy of the liver in female rat offspring following inhalation exposure to vinyl chloride during gestation and lactation (Thornton *et al.*, 2002). The MRL derivation is summarized in Table 5.17.

Table 5.17 ATSDR Vinyl Chloride Non-Cancer Inhalation Toxicity Value

Chemical	Exposure Duration	POD µg/m³ (ppb)	UF	MRL µg/m³ (ppb)	Effect/Source
Vinyl chloride	Intermediate	1,500 (512.5)	30[a]	50 (20)	Increased incidence of centrilobular hypertrophy of the liver in rats (Thornton *et al.*, 2002).

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; µg/m³ = Microgram per Meter Cubed; MRL = Minimal Risk Level; POD = Point of Departure; ppb = Parts per Billion; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.
(a) A UF_H of 10 was applied for human variability and a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species.
Source: ATSDR (2024b).

ATSDR did not derive neurological TTDs for vinyl chloride in its PHA for Camp Lejeune (ATSDR, 2017a).

5.2.4.3 Vinyl Chloride Toxicity Criteria Applied in the Risk Calculations

The toxicity criteria used in the vinyl chloride non-cancer risk calculations (protective of the most sensitive endpoints, including neurological effects) are summarized in Table 5.18.

Table 5.18 Vinyl Chloride Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
Vinyl chloride	Oral RfD	Chronic/Subchronic	Liver polymorphism	0.003 mg/kg-d
	Inhalation MRL	Chronic/Subchronic	Centrilobular hypertrophy of the liver	50 μg/m^3

Notes:
mg/kg-d = Milligram per Kilogram Body Weight per Day; μg/m^3 = Microgram per Meter Cubed; MRL = Minimal Risk Level; RfD = Reference Dose.

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

5.2.5.1 US EPA Toxicity Criteria (RfDs and RfCs)

Table 5.19 summarizes the POD, total UF, associated health effects, and the final RfD US EPA derived for 1,2-tDCE (US EPA, 2010a,b). Table 5.20 summarizes the PODs, total UFs, associated health effects, and the final RfC US EPA derived for 1,2-tDCE (US EPA, 2010a,b).

Based on the hazard assessment for 1,2-tDCE, US EPA (2010a,b) derived an RfD for 1,2-tDCE based on a decreased number of antibody-forming cells (AFCs) against sheep red blood cells (sRBCs) in male mice following exposure to 1,2-tDCE in drinking water (Shopp *et al*., 1985) (Table 5.19). US EPA (2020c) also derived subchronic and chronic provisional RfC (p-RfC) values for 1,2-tDCE based on a subchronic inhalation toxicity study in which decreased lymphocyte counts was observed in male rats (Kelly, 1998) (Table 5.20).

Table 5.19 US EPA 1,2-tDCE Non-Cancer Chronic Oral Toxicity Value

Chemical	POD (mg/kg-d)	UF	RfD (mg/kg-d)	Effect/Source
1,2-tDCE	65	3,000[a]	0.02	Immune effects (Shopp *et al*., 1985)

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; RfD = Reference Dose; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.
Sources: US EPA (2010a,b).
(a) A UF_H of 10 was applied for human variability, a UF_A of 10 was applied for animal-to-human extrapolation, a UF_D of 3 was applied for database deficiencies, and a UF_{Schr} of 10 was applied for extrapolation from subchronic to chronic exposure.

Table 5.20 US EPA 1,2-tDCE Non-cancer Provisional Subchronic and Provisional Chronic Inhalation Toxicity Values

Chemical	Exposure Duration	POD mg/m^3 (ppm)	UF	p-RfC μg/m^3 (ppb)	Effect/Source
1,2-tDCE	Subchronic	109 (27.5)	300[a]	400 (100)	Immune effects – Decreased lymphocyte counts in male mice (Kelly, 1998)
	Chronic	109 (27.5)	3,000[b]	40(10)	

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; μg/m^3 = Microgram per Cubic Meter; mg/m^3 = Milligram per Cubic Meter; POD = Point of Departure; ppb = Parts per Billion; ppm = Parts per Million; p-RfC = Provisional Reference Concentration; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_D = Database Uncertainty Factor; UF_H = Human Variability Uncertainty Factor; UF_{Schr} = Subchronic to Chronic Uncertainty Factor; US EPA = United States Environmental Protection Agency.

Source: US EPA (2020c).

(a) A UF_H of 10 was applied for human variability, a UF_A of 3 was applied for animal-to-human extrapolation to account for toxicodynamic differences between species, and a UF_D of 10 was used to account for database uncertainty.

(b) The same UFs were applied as in (a) except that an additional UF_{Schr} of 10 was applied to account for the use of a subchronic study.

5.2.5.2 ATSDR Toxicity Criteria

I reviewed ATSDR's toxicological profile for 1,2-tDCE (ATSDR, 2023) and its PHA for Camp Lejeune (ATSDR, 2017a) to identify the current ATSDR MRLs for 1,2-tDCE and the 1,2-tDCE toxicity values applied in the PHA for evaluation of human health risk. In its 2023 toxicological profile for 1,2-tDCE, using the same study (Shopp *et al*., 1985) that US EPA used to derive an oral RfD for 1,2-tDCE, ATSDR (2023) derived a provisional intermediate duration oral MRL for 1,2-tDCE. The intermediate MRL derivation is summarized in Table 5.21. ATSDR (2023) concluded that there was insufficient data for 1,2-tDCE for the derivation of a chronic-duration oral MRL.

Table 5.21 ATSDR 1,2-tDCE Non-Cancer Oral Toxicity Value

Chemical	Exposure Duration	POD mg/kg-d	UF	MRL (mg/kg-d)	Effect/Source
1,2-tDCE	Intermediate	16.75	100[a]	0.2	Decreased humoral immunity (Shopp *et al*., 1985)
	Chronic	-	-	-	-

Notes:

1,2-tDCE = *trans*-1,2-Dichloroethylene; ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; MRL = Minimal Risk Level; POD = Point of Departure; UF = Uncertainty Factor; UF_A = Interspecies Uncertainty Factor; UF_H = Human Variability Uncertainty Factor.

Source: ATSDR (2023).

(a) A UF_A of 10 was applied for animal-to-human extrapolation, and a UF_H of 10 was applied for human variability.

In addition to the RfD, RfC, and MRL values for 1,2-tDCE, in its PHA for Camp Lejeune, ATSDR derived a TTD for neurological effects *via* the oral route of exposure (ATSDR, 2017a). The oral TTD for neurological effects for 1,2-tDCE is summarized in Table 5.22. Although the PHA (ATSDR, 2017a) provides information on the effect, it does not specifically describe the derivation of the TTD, nor does it report the underlying study that provides the basis of the value. However, the POD of 336 mg/kg-day is approximately 10-fold lower than the lowest intermediate exposure NOAEL for neurological effects (3,245 mg/kg-day in rats) described in the 1,2-tDCE toxicological profile (ATSDR, 2023 [Table 2-2]). Application of a UF_A of 10 for animal to human extrapolation, and a UF_H of 10 for human variability, for a total UF of 100, would result in a TTD of 32 mg/kg-day if derived from the 3,245 mg/kg-day NOAEL. Therefore, the value derived by ATSDR in the PHA (3.36 mg/kg-day) is about 10-fold more protective than what the neurological studies in the toxicological profile suggest (ATSDR, 2023).

An inhalation neurological TTD was not derived for 1,2-tDCE in its PHA for Camp Lejeune (ATSDR, 2017a).

Table 5.22 ATSDR 1,2-tDCE TTD for Neurological Effects *via* the Oral Route of Exposure

Chemical	POD mg/kg-d	UF	TTD_{neuro} (mg/kg-d)	Effect	Source
1,2-tDCE	336	100[a]	3.36	Acute ataxia	Not provided

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; ATSDR = Agency for Toxic Substances and Disease Registry; mg/kg-day = Milligram per Kilogram Body Weight per Day; POD = Point of Departure; TTD_{neuro} = Target Organ Toxicity Dose for Neurological Effects; UF = Uncertainty Factor.
Source: ATSDR (2017a).
(a) UFs not discussed by ATSDR.

5.2.5.3 1,2-tDCE Toxicity Criteria Applied in the Risk Calculations for the Plaintiff

The toxicity criteria used in the 1,2-tDCE non-cancer risk calculations (protective of the most sensitive endpoints, including neurological effects) are summarized in Table 5.23.

Table 5.23 1,2-tDCE Toxicity Criteria Applied in the Risk Calculations

Chemical	Criteria	Duration	Endpoint	Value
1,2-tDCE	Oral TTD	Chronic/Subchronic	Neurological	3.36 mg/kg-d
	Inhalation RfC	Chronic	Immunological	40 µg/m^3
	Inhalation RfC	Subchronic	Immunological	400 µg/m^3

Notes:
1,2-tDCE = *trans*-1,2-Dichloroethylene; µg/m^3 = Microgram per Cubic Meter; mg/kg-day = Milligram per Kilogram Body Weight per Day; RfC = Reference Concentration; TTD = Target Organ Toxicity Dose.

As discussed in Section 3, exposures less than 7 years are considered subchronic. Thus, subchronic non-cancer toxicity criteria are applied when exposure durations are less than 7 years.

6 Plaintiff-Specific Regulatory Risk Evaluation

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

6.1 Plaintiff Background

As discussed in Ms. Rothchild's deposition (Rothchild, 2024), Ms. Rothchild worked at Camp Lejeune from August 1972 through December 1974 while living off-base (2.4 years). During her time at Camp Lejeune, Ms. Rothchild worked as a teacher at New River Air Station (from August 1972-May 1973) and Tarawa Terrace (from August 1973-June 1974, August 1974-December 1974), going into Camp Lejeune five times per week (Rothchild, 2024). While teaching, Ms. Rothchild testified that that she would use the water from the classroom sink to wash hands, clean desks, and perform science experiments (Rothchild, 2024). When Ms. Rothchild was not teaching, she recalled that she would go into Camp Lejeune approximately four to five times per week and would continue to participate in errands and social activities (*i.e.*, visiting friends' homes, grocery shopping, going to movies, dining in restaurants) on-base (Rothchild, 2024). With regards to water consumption, Ms. Rothchild testified that she would obtain drinking water from her classroom sink, the water fountains on-base, or the exchange store on-base; however, she did not provide an estimate for the amount of water she consumed per day (Rothchild, 2024). Ms. Rothchild also didn't recall using any bathing or showering facilities at the school or on-base (Rothchild, 2024).

In addition, Ms. Rothchild testified that she swam twice per month at the swimming pools on Camp Lejeune and took sailing lessons (Rothchild, 2024). Although Ms. Rothchild did not specify if the pools were indoor or outdoor pools, in her deposition in response to questions about the frequency of swimming in the pools, she stated that "most – a lot of our time was spent on the beach," suggesting that she was referring to outdoor pools during warm periods of the year (Rothchild, 2024). Ms. Rothchild also stated, in response to questions about the Camp Lejeune pools, that she was "not a swimmer," and when asked about swimming for recreation, she stated "I'm great at sunbathing. I can't swim" (Rothchild, 2024). Therefore, it is likely that the pools Ms. Rothchild is referring to are outdoor pools, though she does not specifically state that.

Ms. Rothchild is claiming that her exposure to the water at Camp Lejeune is the cause of her Parkinson's disease, stating her onset symptoms began in January 1993 (Rothchild, 2024).

6.2 Plaintiff Exposure Estimates

Exposure estimates for the plaintiff were calculated based on the average of the monthly average concentrations of TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE over the duration of the plaintiff's exposure period from modeled treatment plant finished water concentrations for both the HP and TT WTPs, which are available in ATSDR's Public Health Assessment (PHA) for Camp Lejeune drinking water (ATSDR, 2017a), as described in Dr. LaKind's report (LaKind, 2025). Exposures from drinking water were evaluated for both the HP and TT WTPs, while dermal and inhalation exposures from hand-washing were evaluated only for the WTP that supplied water to the plaintiff's job location – in this case, Tarawa Terrace. Thus, I evaluated hand-washing exposures for Ms. Rothchild from the TT WTP only. Plaintiff-specific TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE daily tap water exposure estimates, from drinking water (ingestion exposure pathway), showering (dermal and inhalation exposure pathways), and swimming (inhalation exposure pathway), are described in more detail in Dr. LaKind's expert report (LaKind, 2025). Risks were calculated for the following exposure pathways and scenarios for the exposure period of concern (approximately 2.4 years) for the plaintiff:

- Drinking Water Ingestion – For this exposure pathway, because it is not clear that the plaintiff's water ingestion occurred from only one of the two water treatment systems, I evaluated two 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; and (2) reasonable maximum exposure (RME), which assumes ingestion of 3.3 L of tap water per day.
    - Ms. Rothchild did not specify the amount of water she typically drank in her deposition. However, based on her job descriptions and statements regarding her activities when she was not teaching (she was involved in errands and social activities, and would come into Camp Lejeune three to four times per week) (Rothchild, 2024), her tap water ingestion exposures would likely have been consistent with CTE, and RME would likely be and overestimate of her exposure. However, I conservatively include the RME scenario for Ms. Rothchild (Rothchild, 2024). I assumed that Ms. Rothchild drank water on-base throughout the entire 2.4 years she was at Camp Lejeune.
- Dermal and Inhalation Exposures from Hand Washing (TT WTP) – For these exposure pathways, calculated risks based on the CTE (50^{th} percentile) and RME (95^{th} percentile) dermal dose and inhalation concentration outputs from a school bathroom facility exposure model (ATSDR, 2024a), and based on the plaintiff's job location during their time at Camp Lejeune (TT for Ms. Rothchild); these doses and concentrations were provided by Dr. LaKind and are discussed further in her report (LaKind, 2025). Both the CTE and RME exposures from the school bathroom facility exposure model are estimated based on a 250-person facility and a mean hand washing duration of 0.61 minutes, with a standard deviation (SD) time of 0.57 minutes (model defaults) (LaKind, 2025).
    - Ms. Rothchild's deposition testimony does not provide specific details on her frequency or duration of handwashing using the classroom sink (Rothchild, 2024). Since the RME exposure doses and concentrations are, by definition, reflective of the upper 95^{th} percentile of exposures for persons using the facility, Ms. Rothchild's daily handwashing activities are expected to be captured in the RME output from this handwashing model. Since Ms. Rothchild did not teach at Tarawa Terrace until August 1973, I assumed handwashing exposures occurred during the school months only, from August 1973-June 1974 and from August 1974-December 1974 (when she left Camp Lejeune).

In addition to the baseline exposure pathways outlined above, Dr. LaKind's expert report also includes a summary of air exposure concentrations relevant to the indoor swimming pool exposure pathway that was evaluated for Ms. Rothchild (LaKind, 2025). As discussed in the ATSDR "Public Health Assessment for Camp Lejeune Drinking Water," the main exposure pathway for an indoor swimming pool scenario is the inhalation pathway, with potential exposures from other pathways (*e.g.*, dermal pathway) being very minor and contributing little to the overall risk estimate (ATSDR, 2017a). Ms. Rothchild testified that she swam twice per month, took sailing lessons, and floated for 10 minutes in the swimming pools at Camp Lejeune (Rothchild, 2024). Although she did not provide a specific location, her testimony suggests the pools were outdoors and used during the summer months. However, since Ms. Rothchild did not specifically state that the pools were outdoors, I conservatively calculated swimming pool risks based on water concentrations from both the TT and HP WTPs, and assuming the pools were indoors. Based on Ms. Rothchild's deposition testimony, I assumed Ms. Rothchild swam for a total of 24 days during her time at Camp Lejeune; twice per month during five warm months of the year, May through September, for 1 hour on each of those days.[6] Additional details, including air exposure concentrations in the pool area, can be found in Appendix D.

Based on the above exposure pathways, the following exposure scenarios are evaluated for Ms. Rothchild:

- The CTE exposure scenario includes the following exposure pathways: CTE drinking water ingestion (HP and TT WTPs), CTE dermal and inhalation exposures from handwashing (TT WTP), and inhalation from swimming (HP and TT WTPs).
- The RME exposure scenario includes the following exposure pathways: RME drinking water ingestion (HP and TT WTPs), RME dermal and inhalation exposures from handwashing (TT WTPs), and inhalation from swimming (HP and TT WTPs).

6.3 Regulatory Risk Calculations

Non-cancer hazard indices (HIs) for the plaintiff based on the estimates of oral and dermal daily exposure doses (DEDs) and daily inhalation exposure concentrations (DECs), which were based on Dr. LaKind's expert report (LaKind, 2025), considering the exposure duration for the plaintiff (approximately 2.4 years) and applying the toxicity values summarized in Section 5, are presented in Appendix D. As discussed in Section 3, HIs can be segregated by target organ in accordance with US EPA risk assessment methodology (US EPA, 1989). The target organs that are the basis of the toxicity values in this case are the nervous system, immune system, and liver, as discussed in Section 5. Since PD is a neurological endpoint, Table 6.1 summarizes the HI results for the neurological endpoints only.

Table 6.1 Hazard Quotients (HQs) and Hazard Indices (HIs) by Exposure Pathway for Neurological Endpoints for the Plaintiff[a]

Exposure Pathway	Water Source	Hazard Quotients	
		Central Tendency	Reasonable Maximum
Ingestion of Drinking Water	HP WTP	0.008	0.02
	TT WTP	0.1	0.3
Dermal Contact from Hand-Washing at School	TT WTP	0.0006	0.001
Inhalation from Hand-Washing at School	TT WTP	0.0004	0.0007
Additional Exposure Pathway			
Indoor Air Inhalation During Swimming Pool Sessions	HP WTP	0.3	0.3
	TT WTP	0.9	0.9
Total Hazard Indices (All Pathways)			
Assumes drinking water and swimming water come from HP WTP and hand-washing water comes from TT WTP		0.3	0.3
Assumes drinking water, hand-washing water, and swimming water come from TT WTP		1	1

Notes:
HP = Hadnot Point; TT = Tarawa Terrace; WTP = Water Treatment Plant.
(a) All Hazard Quotients are rounded to 1 significant digit, and are based on values from tables in Appendix D.

[6] Note that this conservative exposure frequency is roughly consistent with a once per month indoor swimming pool exposure during the entire time Ms. Rothchild was at Camp Lejeune (2.4 years).

As shown, the neurological hazard indices do not exceed 1 for CTE or RME for exposures from HP or TT WTP, indicating that adverse health effects from these exposures are unlikely for Ms. Rothchild. Further, the maximum total HI for TCE – the only chemical that ATSDR [2017b] considers to be "equipoise and above for causation" for PD – is well below 1 (HI = 0.3, see Appendix D).

It is important to keep in mind that, as discussed in Section 5, 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. Further, although the toxicity values for TCE and PCE are based on neurological effects, the effects are wakefulness (TCE inhalation) and color vision changes (PCE) and not PD. Therefore, the HQs for these chemicals/pathways do not reflect, and are overly protective of, PD risk. See further discussion in Section 5.

It is also important to note that there is some uncertainty in the modeled finished water concentrations 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 from the ATSDR modeled concentrations may be overestimated.

6.4 Risk Evaluation Conclusion

Overall, even with the conservative assumptions that are the basis of the exposure and regulatory risk calculations for Ms. Rothchild, the risk calculations support the conclusion that Ms. Rothchild was not exposed to TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE in tap water at Camp Lejeune at levels that are of concern for human health, including for PD. Even at the highest exposure estimates for Ms. Rothchild, which likely overestimates her drinking water exposures (*i.e.*, RME scenario), and that assume her time spent at swimming pools was all indoors, Ms. Rothchild's exposures result in a neurological HI that does not exceed 1, consistent with the conclusion that adverse health effects would not be expected. Further, the maximum total HI (0.3) for TCE (the only chemical for which ATSDR [2017b] considers the evidence to be "equipoise and above for causation" for PD) is well below 1. Therefore, one cannot reasonably conclude that Ms. Rothchild's exposures to chemicals in Camp Lejeune water are causally associated with her PD.

Because the noncancer risks presented in Table 6.1 are overestimated for healthy individuals in a population (like Ms. Rothchild during the time of her 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 PD.

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 to which UFs are applied for the derivation of non-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 (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 (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:

$$\text{MoE} = \frac{\text{POD for the Toxicity Value}}{\text{Individual ADD or ADE}}$$

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

The PODs for the ingestion (or dermal) and inhalation pathways for each chemical assessed herein are presented in Section 5.2. The plaintiff-specific exposure levels and MoEs are presented in Appendix D. As shown in Tables D.1 and D.2, MoEs range from 200 to 14,000,000,000. Therefore, the MoEs are orders of magnitude above 1, indicating that the plaintiff's estimated exposure levels to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE in tap water (*via* inhalation, ingestion, and dermal exposure) at Camp Lejeune were well below the exposure doses and concentrations used to derive the toxicity criteria for these chemicals, providing additional support that the plaintiff's exposures would not have been expected to lead to adverse health effects, including PD.

8 Consideration of Epidemiology and Animal Studies Relevant to Parkinson's Disease

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 PD risk (Goodman, 2025).

Although Dr. Goodman reviewed epidemiology studies that evaluated potential correlations between chemical exposures and PD in study participants who were stationed (or lived) at Camp Lejeune, I did not consider exposure estimates in 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 with regard to these studies:

> "Overall, there were no consistent associations reported between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and PD. Most risk estimates were small and statistically null, and the few statistically significant risk estimates had wide CIs and were not reported across other analyses of the Camp Lejeune population, indicating a high likelihood of bias or confounding, such that they do not provide evidence of a causal link between exposure to contaminated water at Camp Lejeune and PD" (Goodman, 2025).

8.1 Trichloroethylene (TCE)

As discussed in Section 5, ATSDR (2019a) and US EPA (2011a, 2020a) do not conclude that TCE exposure is a known cause of PD. However, in its public health assessment (PHA) for Camp Lejeune drinking water, ATSDR concluded that there is "equipoise and above evidence for causation for TCE and Parkinson disease" (ATSDR, 2017b). Dr. Goodman concluded that the currently available evidence does not support a causal association between TCE exposure and PD (Goodman, 2025).

The only PD epidemiology study that Dr. Goodman evaluated that reported exposure information for TCE was a Finnish case-control occupational inhalation study conducted by Sallmén *et al*. (2023). Sallmén *et al*. (2023) reported no significant associations between PD and TCE inhalation exposures at concentrations as high as 225 ppm-years. I converted this exposure estimate from an occupational exposure estimate to a continuous daily exposure estimate for a resident (225 ppm-years × 5/7 days × 8/24 hours = 53.6 ppm-years). This TCE exposure estimate is orders of magnitude above (2,200-fold higher than) those estimated for Ms. Rothchild (0.024 ppm-years). I calculated Ms. Rothchild's cumulative TCE exposure in ppm-years by summing the maximum estimated cumulative ppm-year TCE exposures from showering and swimming.[7,8]

[7] 1 ppm TCE = 5,370 μg/m^3 (CDC, 2019a).
[8] I estimated the ppm-year concentrations by first estimating the total number of years for showering (2.4 year) and swimming (24 days or, or 24 days ÷ 365 days/year = 0.066 years). I then converted the maximum daily vapor concentration (in μg/m^3) for each pathway to ppm and multiplied that concentration by the number of years to calculate ppm-years for each pathway. I then summed over the pathways to get the total TCE ppm-year concentration. For example, the maximum TCE daily inhalation

Ms. Rothchild's oral TCE exposure estimates are well below those reported in the animal bioassays discussed by Dr. Goodman (2025) that evaluated PD-associated outcomes. Dr. Goodman describes a study conducted by De Miranda *et al*. (2021) that reported a significant loss of dopamine neurons and a significant decrease in dopamine transporter levels in rats treated with 200 mg/kg-day TCE (*via* oral gavage) for 6 weeks (Goodman, 2025). As described by Dr. Goodman (2025), the magnitude of the reported loss of dopamine neurons in the study (32%) was less than that required to produce clinical signs of PD in humans (60-86%). Moreover, 200 mg/kg-day is orders of magnitude above (41,700-fold higher than) Ms. Rothchild's maximum estimated TCE oral dose of 0.0048 mg/kg-day.

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

8.2 Tetrachloroethylene (PCE)

As discussed in Section 5, US EPA (2012b, 2020b) and ATSDR (2017b, 2019b) did not conclude that PCE exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) concluded that there was "below equipoise evidence for causation" for exposure to PCE and PD. Dr. Goodman concluded that the currently available evidence does not support a causal association between PCE exposure and PD in humans (Goodman, 2025).

The only PD epidemiology study that Dr. Goodman evaluated that reported exposure information for PCE was the occupational study conducted by Sallmén *et al*. (2023). Sallmén *et al*. (2023) reported no significant associations between PD and PCE inhalation exposures at concentrations as high as 145 ppm-years. I converted this exposure estimate from an occupational exposure estimate to a continuous daily exposure estimate for a resident (145 ppm-years × 5/7 days × 8/24 hours = 34.5 ppm-years). This PCE exposure estimate is orders of magnitude above (2,800-fold higher than) those estimated for Ms. Rothchild (0.012 ppm-years). I calculated Ms. Rothchild's cumulative PCE exposure in ppm-years by summing the maximum estimated cumulative ppm-year PCE exposures from showering and swimming.[9,10] See Appendix D for Ms. Rothchild's daily inhalation exposure estimate.

There were no oral animal bioassays discussed in Dr. Goodman's report (Goodman, 2025) that evaluated exposures to PCE and PD risk.

8.3 Benzene

As discussed in Section 5, ATSDR (2007a, 2015, 2017b) and US EPA (2002b) did not conclude that benzene exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) did not comment on a whether there is a causal association between exposure to benzene and PD. Dr. Goodman concluded that the currently available evidence does not support a causal association between benzene exposure and PD in humans (Goodman, 2025).

The only PD epidemiology study that Dr. Goodman evaluated that reported exposure information for benzene was the occupational study conducted by Sallmén *et al*. (2023). Sallmén *et al*. (2023) reported no

concentration from shower vapor = 0.0021 μg/m^3 (0.0000004 ppm). Multiplying 0.00000039 ppm by 2.4 years = 0.00000095 ppm-years. A similar calculation was conducted for swimming pool vapor (0.024 ppm-years). The sum of both pathways is as follows: 0.00000095 + 0.024 = 0.024 ppm-years.

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

[10] Calculations for PCE were conducted in the same manner as for TCE, but with PCE daily exposure concentrations for both exposure pathways, as presented in Appendix D.

significant associations between PD and benzene inhalation exposures at concentrations as high as 90 ppm-years. I converted this exposure estimate from an occupational exposure estimate to a continuous daily exposure estimate for a resident (90 ppm-years × 5/7 days × 8/24 hours = 21.4 ppm-years). This benzene exposure estimate is orders of magnitude above (~40,000-fold higher than) those estimated for Ms. Rothchild (0.0005 ppm-years). I calculated Ms. Rothchild's cumulative benzene exposure in ppm-years by summing the maximum estimated cumulative ppm-year benzene exposures from showering and swimming.[11,12] See Appendix D for Ms. Rothchild's daily inhalation exposure estimate.

There were no oral animal bioassays discussed in Dr. Goodman's report (Goodman, 2025) that evaluated exposures to benzene and PD risk.

8.4 Vinyl Chloride

As discussed in Section 5, US EPA (2003) and ATSDR (2024b) did not conclude that vinyl chloride exposure is a known cause of PD. In its assessment of the evidence for the Camp Lejeune site, ATSDR (2017b) did not comment on whether there is an association between vinyl chloride exposure and PD. Dr. Goodman concluded that the currently available evidence does not support a causal association between vinyl chloride exposure and PD in humans (Goodman, 2025).

As shown in Dr. Goodman's report (Goodman, 2025), there are no epidemiology studies that evaluated potential correlations between vinyl chloride exposure and PD that also included exposure information for the study participants. In addition, Dr. Goodman did not identify any animal bioassays that evaluated potential associations between vinyl chloride exposure and PD-related effects. Therefore, exposure comparisons cannot be made for vinyl chloride.

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

8.6 Conclusions from Epidemiology and Toxicology Studies

As described above, Ms. Rothchild's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE were well below exposure levels from epidemiology studies that reported no significant increases in PD (TCE, PCE, and benzene), or well below exposure levels reported in animal studies that evaluated health effects potentially related to PD (TCE). Therefore, these results provide additional support that Ms. Rothchild's estimated exposures would not have been expected to lead to her PD.

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

[12] Calculations for benzene were conducted in the same manner as for TCE and PCE, but with benzene daily exposure concentrations for all four exposure pathways, as presented in Appendix D.

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. Kristin Andruska (2025), who provided opinions on specific causation for Ms. Rothchild. 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, or benzene exposures for Ms. Rothchild 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 the plaintiff based on the water concentration of the chemical and the plaintiff's daily water 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 milligrams of chemical per kilogram of human body weight per day (mg/kg-day). Inhalation exposure estimates represent the daily exposure concentration of a chemical taken into the body, averaged over the appropriate exposure period and expressed in units of micrograms of a chemical per cubic meter of air ($\mu g/m^3$). As discussed in Section 3, doses and inhalation exposure estimates can then be used to calculate non-cancer hazard indices (HIs) using US EPA's chemical-specific toxicity criteria, and then the results can be compared to US EPA guidelines for acceptable HIs. Therefore, Dr. Reynolds' representation of exposure as the total ingested amount of a chemical (μg) cannot be used directly to evaluate potential health effects for the plaintiff. That is, the mass of ingested chemicals needs to be divided by body weight for the plaintiff and averaged over the appropriate averaging time, as described in Section 3, so that the oral doses can be used to calculate HIs per US EPA risk assessment guidelines.

Further, total mass cannot be compared to exposure estimates in most reliable animal or epidemiology studies. Doses (mg/kg-day) or inhalation concentrations ($\mu g/m^3$) are typically used in animal bioassays for evaluating potential health effects from chemical exposures. Most reliable epidemiology studies provide cumulative exposure estimates in ppm-years (*i.e.*, inhalation exposure concentration × number of years exposed) and ppb-months or ppb-years (*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. Andruska

Dr. Andruska's report (Andruska, 2025) does not provide a reliable analysis of specific causation or risk of PD with regard to Ms. Rothchild's alleged exposures. Dr. Andruska concludes that Ms. Rothchild's exposure to PCE and TCE in the water at Camp Lejeune is "at least as likely as not the cause of her Parkinson's Disease" without providing a robust analysis of the best available scientific information relevant to the potential causal association between Ms. Rothchild's exposure to these chemicals and PD. Below, I describe several flaws in Dr. Andruska's analysis:

- Dr. Andruska'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. Andruska relies on Dr. Reynolds' exposure charts as support that Ms. Rothchild's exposures were "significant" and "substantial," but provides no basis for this conclusion other than simply pointing to the total mass values reported by Dr. Reynolds (2025a). As discussed in the previous section, 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. Andruska's conclusions based on estimates of total chemical mass exposure for Ms. Rothchild are meaningless, misleading, and cannot be relied upon for risk evaluation for Ms. Rothchild.
- Dr. Andruska's comparison to US EPA maximum contaminant levels (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, 2024), which is much longer than Ms. Rothchild's approximately 2.4 years of exposure during her time at Camp Lejeune.
  - Further, the MCLs for TCE, PCE, vinyl chloride, and benzene are based on cancer health effects and not PD; therefore, an MCL exceedance for these chemicals, even over a longer period of time than the plaintiff was exposed, is not relevant to PD.
  - Therefore, a simple comparison of drinking water concentrations to MCLs, without considering exposure duration or the health effect on which the MCL is based, is not consistent with standard risk assessment practice, and is misleading.
  - More importantly, Dr. Andruska fails to mention that the range of TCE concentrations that Dr. Reynolds reports for Ms. Rothchild are *consistently below the TCE MCL* of 5 μg/L (0 to 1.74 μg/L). As discussed in Section 5, no agency has concluded that the other chemicals (PCE, benzene, or vinyl chloride) are causally associated with PD.

- Dr. Andruska's report refers to exposure information from several Camp Lejeune studies to support her 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, overall, Dr. Goodman states the following:

> Overall, there were no consistent associations reported between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and PD. Most risk estimates were small and statistically null, and the few statistically significant risk estimates had wide CIs and were not reported across other analyses of the Camp Lejeune population, indicating a high likelihood of bias or confounding, such that they do not provide evidence of a causal link between exposure to contaminated water at Camp Lejeune and PD (Goodman, 2025).

- In addition, as discussed in Section 5, based on a comprehensive review of the best available and most current epidemiology and animal studies, Dr. Goodman (2025) concludes that the scientific evidence does not support a causal association between TCE, PCE, benzene, vinyl chloride, or 1,2-tDCE exposure and PD.

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 hazard indices (HIs) estimated for Ms. Rothchild's exposures, for neurological effects for a healthy worker population, do not exceed US EPA's acceptable HI target.

Therefore, Dr. Reynolds' and Dr. Andruska's expert reports do not change my opinions, as discussed in my report and summarized in Section 10, regarding Ms. Rothchild's claim that exposures from Camp Lejeune are the cause of her PD.

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 PD epidemiology and animal studies discussed in Section 8, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Ms. Rothchild's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 2.4 years that she worked and spent time at Camp Lejeune are causally associated with her PD.

References

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Andruska, K. [California Movement Disorders Center]. 2025. "Expert Report of Kristin Andruska, MD, PhD, Rothchild v. United States, No. 7:23-cv-00858-D-KS." 138p., February 6.

Arito, H; Takahashi, M; Ishikawa, T. 1994. "Effect of subchronic inhalation exposure to low-level trichloroethylene on heart rate and wakefulness-sleep in freely moving rats." *Sangyo Igaku* 36(1):1-8. doi: 10.1539/joh1959.36.1.

Bove, FJ; Ruckart, PZ; Maslia, M; Larson, TC. 2014. "Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base camp Lejeune: A retrospective cohort study." *Environ. Health* 13:10. doi: 10.1186/1476-069X-13-10.

Cavalleri, A; Gobbs, F; Paltrinieri, M; Fantuzzi, G; Righi, E; Aggazzotti, G. 1994. "Perchloroethylene exposure can induce colour vision loss." *Neurosci. Lett.* 179(1-2):162-166. doi: 10.1016/0304-3940(94)90959-8.

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

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

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

Dankovic, DA; Naumann, BD; Maier, A; Dourson, ML; Levy, LS. 2015. "The scientific basis of uncertainty factors used in setting occupational exposure limits." *J. Occup. Environ. Hyg.* 12(Suppl. 1):S55-S68. doi: 10.1080/15459624.2015.1060325.

De Miranda, BR; Castro, SL; Rocha, EM; Bodle, CR; Johnson, KE; Greenamyre, JT. 2021. "The industrial solvent trichloroethylene induces LRRK2 kinase activity and dopaminergic neurodegeneration in a rat model of Parkinson's disease." *Neurobiol. Dis.* 153:105312. doi: 10.1016/j.nbd.2021.105312.

Echeverria, D; White, RF; Sampaio, C. 1995. "A behavioral evaluation of PCE exposure in patients and dry cleaners: A possible relationship between clinical and pre-clinical effects." *J. Occup. Med.* 37(6):667-680.

Gash, DM; Rutland, K; Hudson, NL; Sullivan, PG; Bing, G; Cass, WA; Pandya, JD; Liu, M; Choy, DY; Hunter, RL; Gerhardt, GA; Smith, CD; Slevin, JT; Prince, TS. 2008. "Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity." *Ann. Neurol.* 63(2):184-192.

Goodman, JE. 2025. "Trichloroethylene, Perchloroethylene, Benzene, Vinyl Chloride, and trans-1,2-Dichloroethylene and Parkinson's Disease." 282p., February 7.

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

Johnson, PD; Goldberg, SJ; Mays, MZ; Dawson, BV. 2003. "Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat." *Environ. Health Perspect.* 111:289-292.

Keil, DE; Peden-Adams, MM; Wallace, S; Ruiz, P; Gilkeson, GS. 2009. "Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease." J. Environ. Sci. Health A Tox. Hazard. Subst. Environ. Eng. 44(5):443-453. doi: 10.1080/10934520902719738.

Kelly, DP. [E. I. du Pont de Nemours and Co., Haskell Laboratory for Toxicology and Industrial Medicine]. 1998. "Trans-1,2-Dichloroethylene: 90-day Inhalation Toxicity Study in Rats. Volume 1 of 2." DuPont HL-1998-00952. 112p., December 1. Accessed at https://chemview.epa.gov/chemview/proxy?filename=09022526804d3104_Dupont199800952.pdf.

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

Lan, Q; Zhang, L; Li, G; Vermeulen, R; Weinberg, RS; Dosemeci, M; Rappaport, SM; Shen, M; Alter, BP; Wu, Y; Kopp, W; Waidyanatha, S; Rabkin, C; Guo, W; Chanock, S; Hayes, RB; Linet, M; Kim, S; Yin, S; Rothman, N; Smith, MT. 2004. "Hematotoxicity in workers exposed to low levels of benzene." *Science* 306:1774-1776.

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

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

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

Peden-Adams, MM; Eudaly, JG; Heesemann, LM; Smythe, J; Miller, J; Gilkeson, GS; Keil, DE. 2006. "Developmental immunotoxicity of trichloroethylene (TCE): Studies in B6C3F1 mice." *J. Environ. Sci. Health A Tox. Hazard. Subst. Environ. Eng.* 41(3):249-271. doi: 10.1080/10934520500455289.

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

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

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

Rosenthal, GJ; Snyder, CA. 1987. "Inhaled benzene reduces aspects of cell-mediated tumor surveillance in mice." *Toxicol. Appl. Pharmacol.* 88(1):35-43. doi: 10.1016/0041-008x(87)90267-5.

Rothchild, D. 2024. "Deposition and other relevant plaintiff-specific materials of Diane Rothchild [re: Diane Rothchild *vs.* United States of America]." Submitted to US District Court, Eastern District of North Carolina. Case No.: 723-CV-0858-D-KS.

Rothman, N; Smith, MT; Hayes, RB; Li, GL; Irons, RD; Dosemeci, M; Haas, R; Stillman,, WS; Linet, M; Xi, LQ; Bechtold, WE; Wiemels, J; Campleman, S; Zhang, L; Quintana, PJ; Titenko-Holland, N; Wang, YZ; Lu, W; Kolachana, P; Meyer, KB; Yin, S. 1996. "An epidemiologic study of early biologic effects of benzene in Chinese workers." *Environ. Health Perspect.* 104(Suppl. 6):1365-1370.

Sallmén, M; Burstyn, I; Uuksulainen, S; Koskinen, A; Hublin, C; Sainio, M. 2023. "Parkinson's disease and occupational exposure to organic solvents in Finland: A nationwide case-control study." *Scand. J. Work Environ. Health* 22:4125. doi: 10.5271/sjweh.4125.

Schneider, K; Dilger, M; Drossard, C; Ott, H; Kaiser, E. 2022. "Derivation of occupational exposure limits: Differences in methods and protection levels." *J. Appl. Toxicol.* 42(5):913-926. doi: 10.1002/jat.4307.

Shopp, GM Jr.; Sanders, VM; White, KL Jr.; Munson, AE. 1985. "Humoral and cell-mediated immune status of mice exposed to trans-1,2-dichloroethylene." *Drug Chem. Toxicol.* 8(5):393-407. doi: 10.3109/01480548509041066.

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

Thornton, SR; Schroeder, RE; Robison, RL; Rodwell, DE; Penney, DA; Nitschke, KD; Sherman, WK. 2002. "Embryo-fetal developmental and reproductive toxicology of vinyl chloride in rats." *Toxicol. Sci.* 68(1):207-219. doi: 10.1093/toxsci/68.1.207.

Til, HP; Immel, HR; Feron, VJ. 1982. "Life-Span Oral Carcinogenicity Study of Vinyl Chloride in Rats (Final report with attachments)." Civo Institutes, National Technical Information Service (NTIS). TNO Report No. V 83-285/291099, TSCATS Document FYI-AX-0194-0353, Fiche No. 0353, NTIS OTS0537124. 107p., June 29.

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

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

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

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

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

US EPA. 2002b. "Toxicological Review of Benzene (Noncancer Effects) (CAS No. 71-43-2)." EPA/635/R-02/001F. 166p., October.

US EPA. 2003. "IRIS record for vinyl chloride (CAS No. 75-01-4)." 50p., October 28. Accessed at http://www2.epa.gov/iris.

US EPA. 2004. "Risk Assessment Guidance for Superfund (RAGS). Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (Final)." Office of Superfund Remediation and Technology Innovation. EPA/540/R/99/005, OSWER 9285.7-02EP, PB99-963312. 156p., July. Accessed at http://www.epa.gov/oswer/riskassessment/ragse/pdf/part_e_final_revision_10-03-07.pdf.

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

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

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

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

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

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

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

US EPA. 2014. "Memorandum to Superfund National Policy Managers, Regions 1-10 re: Human Health Evaluation Manual, Supplemental Guidance: Update of standard default exposure factors." Office of Solid Waste and Emergency Response (OSWER); Stalcup, D. OSWER Directive 9200.1-120. 7p., February 6. Accessed at https://www.epa.gov/sites/production/files/2015-11/documents/oswer_directive_9200.1-120_exposurefactors_corrected2.pdf.

US EPA. 2020a. "Risk Evaluation for Trichloroethylene (CASRN: 79-01-6) (Final)." EPA Document # 740R18008. 803p., November. Accessed at https://www.epa.gov/sites/production/files/2020-11/documents/1._risk_evaluation_for_trichloroethylene_tce_casrn_79-01-6.pdf.

US EPA. 2020b. "Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro-) (CASRN: 127-18-4) (Final)." EPA Document # 740-R1-8011. 714p., December. Accessed at https://www.epa.gov/sites/production/files/2020-12/documents/1_risk_evaluation_for_perchloroethylene_pce_casrn_127-18-4_0.pdf.

US EPA. 2020c. "Provisional Peer Reviewed Toxicity Values for trans-1,2-Dichloroethylene (CASRN 156-60-5) (Final)." EPA/690/R-20/006F. 72p., September.

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

US EPA. 2024. "National Primary Drinking Water Regulations." 40 CFR 141. 567p. Accessed at https://www.ecfr.gov/current/title-40/chapter-I/subchapter-D/part-141#p-141.23(i)

Appendix A

Curriculum Vitae of 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

4/16/2025

Select Projects

Confidential Client: In support of toxic tort litigation, reviewed toxicology, epidemiology, and exposure information related to claims of specific causal associations between oral, dermal, and inhalation exposures to trichloroethylene, perchloroethylene, and benzene and health effects (*e.g.*, Parkinson's disease, leukemia, kidney cancer, and bladder cancer).

Confidential Client: In support of litigation, reviewed site-specific soil, groundwater, soil vapor, and indoor air data related to claims of potential health effects associated with inhalation and oral exposures to trichloroethylene.

Confidential Client: In support of litigation, reviewed mold species data related to claims of potential health effects associated with exposures to mold on wet surfaces in a residential environment.

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 (*e.g.*, benzene, PAHs, particulate matter) 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 benzene, diesel exhaust, diesel fuel, silica, asbestos, and cancer endpoints (e.g., lung cancer, colon cancer, and hematological cancers).

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Awards and Honors

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

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

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

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

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

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

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

Publications and Book Chapters

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Rhomberg, LR; Goodman, JE; Bailey, LA; Prueitt, RL; Beck, NB; Bevan, C; Honeycutt, M; Kaminski, NE; Paoli, G; Pottenger, LH; Scherer, RW; Wise, KC; Becker, RA. 2013. "A survey of frameworks for best practices in weight-of-evidence analyses." *Crit. Rev. Toxicol.* 43(9):753-784.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Poster Presentations

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Oral Public Comments, Presentations, and Testimony

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Appendix B

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

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

Dr. Bailey has provided expert testimony as follows:

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

Appendix C

List of Materials Considered

Appendix D

Plaintiff Risk Calculations

Table D.1 Risk Calculations for the Baseline Daily Drinking Water and School Bathroom Exposures for Diane Rothchild

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Average Daily Dose (ADD) or Exposure (ADE)[a]		Toxicity Reference Value		Target Organ	Hazard Quotient[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	4.4E-05	mg/kg-day	4.4E-05	mg/kg-day	1.5E-01	mg/kg-day	Nervous system	2.9E-04	1.5E+01	mg/kg-day	3.4E+05	N
				trans -1,2-Dichloroethylene	2.2E-03	mg/kg-day	2.2E-03	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	6.5E-04	3.4E+02	mg/kg-day	1.5E+05	N
				Tetrachloroethylene	3.0E-05	mg/kg-day	3.0E-05	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	5.0E-03	2.6E+00	mg/kg-day	8.7E+04	N
				Trichloroethylene	1.9E-03	mg/kg-day	1.9E-03	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	1.9E-03	1.0E+03	mg/kg-day	5.3E+05	N
				Vinyl Chloride	9.4E-05	mg/kg-day	9.4E-05	mg/kg-day	3.0E-03	mg/kg-day	Liver	3.1E-02	9.0E-02	mg/kg-day	9.6E+02	N
											Total for Hadnot Point Ingestion (CTE):	**4E-02**				
CTE	Tarawa Terrace	Drinking water	Ingestion	Benzene	NA	mg/kg-day	NA	mg/kg-day	1.5E-01	mg/kg-day	NA	NA	1.5E+01	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	7.2E-05	mg/kg-day	7.2E-05	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	2.1E-05	3.4E+02	mg/kg-day	4.7E+06	N
				Tetrachloroethylene	6.6E-04	mg/kg-day	6.6E-04	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	1.1E-01	2.6E+00	mg/kg-day	3.9E+03	N
				Trichloroethylene	2.6E-05	mg/kg-day	2.6E-05	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	2.6E-05	1.0E+03	mg/kg-day	3.8E+07	N
				Vinyl Chloride	3.6E-05	mg/kg-day	3.6E-05	mg/kg-day	3.0E-03	mg/kg-day	Liver	1.2E-02	9.0E-02	mg/kg-day	2.5E+03	N
											Total for Tarawa Terrace Ingestion (CTE):	**1E-01**				
CTE	Tarawa Terrace	Bathroom water	Dermal	Benzene	NA	mg/kg-day	NA	mg/kg-day	1.5E-01	mg/kg-day	NA	NA	1.5E+01	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	1.5E-07	mg/kg-day	8.2E-08	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	2.5E-08	3.4E+02	mg/kg-day	4.1E+09	N
				Tetrachloroethylene	6.8E-06	mg/kg-day	3.7E-06	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	6.2E-04	2.6E+00	mg/kg-day	7.0E+05	N
				Trichloroethylene	7.3E-08	mg/kg-day	4.0E-08	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	4.0E-08	1.0E+03	mg/kg-day	2.5E+10	N
				Vinyl Chloride	4.5E-08	mg/kg-day	2.5E-08	mg/kg-day	3.0E-03	mg/kg-day	Liver	8.2E-06	9.0E-02	mg/kg-day	3.6E+06	N
											Total for Tarawa Terrace Dermal (CTE):	**6E-04**				
CTE	Tarawa Terrace	Indoor air	Inhalation	Benzene	NA	μg/m^3	NA	μg/m^3	2.0E+01	μg/m^3	NA	NA	5.8E+03	μg/m^3	NA	NA
				trans -1,2-Dichloroethylene	4.1E-03	μg/m^3	2.3E-03	μg/m^3	4.0E+02	μg/m^3	Immune system	5.6E-06	1.1E+05	μg/m^3	4.8E+07	N
				Tetrachloroethylene	3.2E-02	μg/m^3	1.8E-02	μg/m^3	4.0E+01	μg/m^3	Nervous system	4.4E-04	1.5E+04	μg/m^3	8.5E+05	N
				Trichloroethylene	1.3E-03	μg/m^3	7.1E-04	μg/m^3	1.9E+02	μg/m^3	Nervous system	3.7E-06	6.4E+04	μg/m^3	8.9E+07	N
				Vinyl Chloride	2.2E-03	μg/m^3	1.2E-03	μg/m^3	5.0E+01	μg/m^3	Liver	2.4E-05	1.5E+03	μg/m^3	1.2E+06	N
											Total for Tarawa Terrace Inhalation (CTE):	**5E-04**				
Reasonable Maximum Exposure (RME)																
RME	Hadnot Point	Drinking water	Ingestion	Benzene	1.1E-04	mg/kg-day	1.1E-04	mg/kg-day	1.5E-01	mg/kg-day	Nervous system	7.3E-04	1.5E+01	mg/kg-day	1.4E+05	N
				trans -1,2-Dichloroethylene	5.3E-03	mg/kg-day	5.3E-03	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	1.6E-03	3.4E+02	mg/kg-day	6.3E+04	N
				Tetrachloroethylene	7.3E-05	mg/kg-day	7.3E-05	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	1.2E-02	2.6E+00	mg/kg-day	3.6E+04	N
				Trichloroethylene	4.8E-03	mg/kg-day	4.8E-03	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	4.8E-03	1.0E+03	mg/kg-day	2.1E+05	N
				Vinyl Chloride	2.3E-04	mg/kg-day	2.3E-04	mg/kg-day	3.0E-03	mg/kg-day	Liver	7.7E-02	9.0E-02	mg/kg-day	3.9E+02	N
											Total for Hadnot Point Ingestion (RME):	**1E-01**				
RME	Tarawa Terrace	Drinking water	Ingestion	Benzene	NA	mg/kg-day	NA	mg/kg-day	1.5E-01	mg/kg-day	NA	NA	1.5E+01	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	1.8E-04	mg/kg-day	1.8E-04	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	5.4E-05	3.4E+02	mg/kg-day	1.9E+06	N
				Tetrachloroethylene	1.6E-03	mg/kg-day	1.6E-03	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	2.7E-01	2.6E+00	mg/kg-day	1.6E+03	N
				Trichloroethylene	6.5E-05	mg/kg-day	6.5E-05	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	6.5E-05	1.0E+03	mg/kg-day	1.5E+07	N
				Vinyl Chloride	8.9E-05	mg/kg-day	8.9E-05	mg/kg-day	3.0E-03	mg/kg-day	Liver	3.0E-02	9.0E-02	mg/kg-day	1.0E+03	N
											Total for Tarawa Terrace Ingestion (RME):	**3E-01**				

Exposure Scenario	Exposure Point	Exposure Medium	Exposure Route	Analyte	Daily Exposure Dose (DED) or Concentration (DEC)		Average Daily Dose (ADD) or Exposure (ADE)[a]		Toxicity Reference Value		Target Organ	Hazard Quotient[a]	Point of Departure (POD)		Margin of Exposure[b]	Exposure Exceeds POD? (Y/N)
					Value	Units	Value	Units	Value	Units			Value	Units		
RME	Tarawa Terrace	Bathroom water	Dermal	Benzene	NA	mg/kg-day	NA	mg/kg-day	1.5E-01	mg/kg-day	NA	NA	1.5E+01	mg/kg-day	NA	NA
				trans -1,2-Dichloroethylene	2.5E-07	mg/kg-day	1.4E-07	mg/kg-day	3.4E+00	mg/kg-day	Nervous system	4.1E-08	3.4E+02	mg/kg-day	2.4E+09	N
				Tetrachloroethylene	1.1E-05	mg/kg-day	6.0E-06	mg/kg-day	6.0E-03	mg/kg-day	Nervous system	1.0E-03	2.6E+00	mg/kg-day	4.3E+05	N
				Trichloroethylene	1.2E-07	mg/kg-day	6.6E-08	mg/kg-day	1.0E+00	mg/kg-day	Nervous system	6.6E-08	1.0E+03	mg/kg-day	1.5E+10	N
				Vinyl Chloride	7.2E-08	mg/kg-day	4.0E-08	mg/kg-day	3.0E-03	mg/kg-day	Liver	1.3E-05	9.0E-02	mg/kg-day	2.3E+06	N
Total for Tarawa Terrace Dermal (RME):												**1E-03**				
RME	Tarawa Terrace	Indoor air	Inhalation	Benzene	NA	μg/m³	NA	μg/m3	2.0E+01	μg/m³	NA	NA	5.8E+03	μg/m³	NA	NA
				trans -1,2-Dichloroethylene	6.3E-03	μg/m³	3.5E-03	μg/m3	4.0E+02	μg/m³	Immune system	8.7E-06	1.1E+05	μg/m³	3.1E+07	N
				Tetrachloroethylene	4.9E-02	μg/m³	2.7E-02	μg/m3	4.0E+01	μg/m³	Nervous system	6.7E-04	1.5E+04	μg/m³	5.6E+05	N
				Trichloroethylene	2.1E-03	μg/m³	1.2E-03	μg/m3	1.9E+02	μg/m³	Nervous system	6.0E-06	6.4E+04	μg/m³	5.5E+07	N
				Vinyl Chloride	3.4E-03	μg/m³	1.9E-03	μg/m3	5.0E+01	μg/m³	Liver	3.7E-05	1.5E+03	μg/m³	8.0E+05	N
Total for Tarawa Terrace Inhalation (RME):												**7E-04**				

Notes:

μg/m³ = Micrograms per Cubic Meter; mg/kg-day = Milligrams per Kilogram Body Weight per Day; N = No; NA = Not Applicable; Y = Yes.

(a) Average daily doses (ADDs), average daily exposures (ADEs), and hazard quotients (HQs) are calculated using the following equations:

Ingestion and Dermal Contact:

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

$$HQ = \frac{ADD}{RfD}$$

Inhalation:

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

$$HQ = \frac{ADE}{RfC}$$

where:

Variable	Definition	Units	Value	Source/Notes
ADD	Average Daily Dose (Oral and Dermal)	mg/kg-day	Chemical specific	Calculated
ADE	Average Daily Exposure (Inhalation)	μg/m³	Chemical specific	Calculated
DED	Daily Exposure Dose	mg/kg-day	Chemical specific	LaKind (2025)
DEC	Daily Exposure Concentration	μg/m³	Chemical specific	LaKind (2025)
EF	Exposure Frequency	days/year	365	Assumes daily exposure while on-base
ED_{DW}	Exposure Duration (Drinking Water)	years	2.4	Time spent visiting or working on-base
ED_{school}	Exposure Duration (School Bathroom)	years	1.3	Time spent working on-base
AT	Averaging Time	days	882	Total time spent on-base
HQ	Hazard Quotient	unitless	Chemical specific	Calculated
RfD	Reference Dose	mg/kg-day	Chemical specific	Section 5 of report
RfC	Reference Concentration	μg/m³	Chemical specific	Section 5 of report

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

Table D.2 Risk Calculations for Swimming Exposures for Diane Rothchild

Exposure Point	Analyte	Vapor Concentration (VC) in Pool Area (µg/m^3)	Daily Exposure Concentration (DEC)[a] (µg/m^3)	Average Daily Exposure (ADE)[a] (µg/m^3)	RfC (µg/m^3)	HQ[a]	POD	MoE[b]	Exposure Exceeds POD? (Y/N)
Hadnot Point	Benzene	620	26	7.0E-01	2.0E+01	3.5E-02	5.8E+03	8.3E+03	N
	trans -1,2-Dichloroethylene	50,800	2,117	5.8E+01	4.0E+02	1.4E-01	1.1E+05	1.9E+03	N
	Tetrachloroethylene	1,330	55	1.5E+00	4.0E+01	3.8E-02	1.5E+04	9.9E+03	N
	Trichloroethylene	47,900	1,996	5.4E+01	1.9E+02	2.8E-01	6.4E+04	1.2E+03	N
	Vinyl Chloride	6,540	273	7.4E+00	5.0E+01	1.5E-01	1.5E+03	2.0E+02	N
					Total for Swimming Exposures – Hadnot Point:	**6E-01**			
Tarawa Terrace	*trans* -1,2-Dichloroethylene	1,690	70	1.9E+00	4.0E+02	4.8E-03	1.1E+05	5.7E+04	N
	Tetrachloroethylene	29,900	1,246	3.4E+01	4.0E+01	8.5E-01	1.5E+04	4.4E+02	N
	Trichloroethylene	649	27	7.4E-01	1.9E+02	3.8E-03	6.4E+04	8.7E+04	N
	Vinyl Chloride	2,520	105	2.9E+00	5.0E+01	5.7E-02	1.5E+03	5.3E+02	N
					Total for Swimming Exposures – Tarawa Terrace:	**9E-01**			

Notes:

µg/m^3 = Micrograms per Cubic Meter; MoE = Margin of Exposure; N = No; POD = Point of Departure; Y = Yes.

(a) Daily exposure concentrations (DECs), average daily exposures (ADEs), and hazard quotients (HQs) are calculated using the following equations:

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

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

$$HQ = \frac{ADE}{RfC}$$

where:

Variable	Definition (Units)	Units	Value	Source/Notes
VC	Vapor Concentration in Pool Area	µg/m^3	Chemical specific	LaKind (2025)
DEC	Daily Exposure Concentration	µg/m^3	Chemical specific	Calculated
ADE	Average Daily Exposure	µg/m^3	Chemical specific	Calculated
ET	Exposure Time	hours/day	1	Professional Judgment
EF	Exposure Frequency	days/event	1	Professional Judgment
EV	Events During Exposure Duration	number of events	24	Professional Judgment
AT	Averaging Time	days	882	Total time spent on-base
HQ	Hazard Quotient	unitless	Chemical specific	Calculated
RfC	Reference Concentration	µg/m^3	Chemical specific	Section 5 of report

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

Table D.3 Summary of Risks By Exposure Pathway (Baseline + Additional) for Diane Rothchild

Exposure Scenario	Exposure Point	Analyte	Exposure Pathways									Additional Pathways			Total HQ
			Ingestion (Drinking Water)			Dermal (School Bathroom)			Inhalation (School Bathroom)			Inhalation (Swimming Pool)			
			HQ	%	Target Organ	HQ	%	Target Organ	HQ	%	Target Organ	HQ	%	Target Organ	
Central Tendency Exposure (CTE)															
CTE	Hadnot Point: Drinking Water and Swimming Tarawa Terrace: Dermal and Inhalation Exposures from Bathroom Hand washing	Benzene	2.9E-04	0.7%	Nervous system	NA	--	Nervous system	NA	--	Immune system	3.5E-02	5%	Immune system	3.5E-02
		trans -1,2-Dichloroethylene	6.5E-04	2%	Nervous system	2.5E-08	0.004%	Nervous system	5.6E-06	1%	Immune system	1.4E-01	22%	Immune system	1.4E-01
		Tetrachloroethylene	5.0E-03	13%	Nervous system	6.2E-04	99%	Nervous system	4.4E-04	93%	Nervous system	3.8E-02	6%	Nervous system	4.4E-02
		Trichloroethylene	1.9E-03	5%	Nervous system	4.0E-08	0.006%	Nervous system	3.7E-06	0.8%	Nervous system	2.8E-01	44%	Nervous system	2.8E-01
		Vinyl Chloride	3.1E-02	80%	Liver	8.2E-06	1%	Liver	2.4E-05	5%	Liver	1.5E-01	23%	Liver	1.8E-01
		Pathway-Specific Total:	**4E-02**			**6E-04**			**5E-04**			**6E-01**			**7E-01**
		Target-Organ Specific Hazard Indices													
		Nervous System	*8E-03*			*6E-04*			*4E-04*			*3E-01*			***3E-01***
		Immune System	*0E+00*			*0E+00*			*6E-06*			*2E-01*			***2E-01***
		Liver	*3E-02*			*8E-06*			*2E-05*			*1E-01*			***2E-01***
CTE	Tarawa Terrace: All Exposure Pathways	Benzene	NA	--	Nervous system	NA	--	Nervous system	NA	--	Immune system	NA	--	Immune system	0.0E+00
		trans -1,2-Dichloroethylene	2.1E-05	0.02%	Nervous system	2.5E-08	0.004%	Nervous system	5.6E-06	1%	Immune system	4.8E-03	0.5%	Immune system	4.8E-03
		Tetrachloroethylene	1.1E-01	90%	Nervous system	6.2E-04	99%	Nervous system	4.4E-04	93%	Nervous system	8.5E-01	93%	Nervous system	9.6E-01
		Trichloroethylene	2.6E-05	0%	Nervous system	4.0E-08	0.006%	Nervous system	3.7E-06	0.8%	Nervous system	3.8E-03	0.4%	Nervous system	3.9E-03
		Vinyl Chloride	1.2E-02	10%	Liver	8.2E-06	1%	Liver	2.4E-05	5%	Liver	5.7E-02	6%	Liver	6.9E-02
		Pathway-Specific Total:	**1E-01**			**6E-04**			**5E-04**			**9E-01**			**1E+00**
		Target-Organ Specific Hazard Indices													
		Nervous System	*1E-01*			*6E-04*			*4E-04*			*9E-01*			***1E+00***
		Immune System	*0E+00*			*0E+00*			*6E-06*			*5E-03*			***5E-03***
		Liver	*1E-02*			*8E-06*			*2E-05*			*6E-02*			***7E-02***
Reasonable Maximum Exposure (RME)															
RME	Hadnot Point: Drinking Water and Swimming Tarawa Terrace: Dermal and Inhalation Exposures from Bathroom Hand washing	Benzene	7.3E-04	0.8%	Nervous system	NA	--	Nervous system	NA	--	Immune system	3.5E-02	5%	Immune system	3.6E-02
		trans -1,2-Dichloroethylene	1.6E-03	2%	Nervous system	4.1E-08	0.004%	Nervous system	8.7E-06	1%	Immune system	1.4E-01	22%	Immune system	1.5E-01
		Tetrachloroethylene	1.2E-02	13%	Nervous system	1.0E-03	99%	Nervous system	6.7E-04	93%	Nervous system	3.8E-02	6%	Nervous system	5.2E-02
		Trichloroethylene	4.8E-03	5%	Nervous system	6.6E-08	0.006%	Nervous system	6.0E-06	0.8%	Nervous system	2.8E-01	44%	Nervous system	2.9E-01
		Vinyl Chloride	7.7E-02	80%	Liver	1.3E-05	1%	Liver	3.7E-05	5%	Liver	1.5E-01	23%	Liver	2.3E-01
		Pathway-Specific Total:	**1E-01**			**1E-03**			**7E-04**			**6E-01**			**7E-01**
		Target-Organ Specific Hazard Indices													
		Nervous System	*2E-02*			*1E-03*			*7E-04*			*3E-01*			***3E-01***
		Immune System	*0E+00*			*0E+00*			*9E-06*			*2E-01*			***2E-01***
		Liver	*8E-02*			*1E-05*			*4E-05*			*1E-01*			***2E-01***
RME	Tarawa Terrace: All Exposure Pathways	Benzene	NA	--	Nervous system	NA	--	Nervous system	NA	--	Immune system	NA	--	Immune system	0.0E+00
		trans -1,2-Dichloroethylene	5.4E-05	0.02%	Nervous system	4.1E-08	0.004%	Nervous system	8.7E-06	1%	Immune system	4.8E-03	0.5%	Immune system	4.9E-03
		Tetrachloroethylene	2.7E-01	90%	Nervous system	1.0E-03	99%	Nervous system	6.7E-04	93%	Nervous system	8.5E-01	93%	Nervous system	1.1E+00
		Trichloroethylene	6.5E-05	0.02%	Nervous system	6.6E-08	0.006%	Nervous system	6.0E-06	0.8%	Nervous system	3.8E-03	0.4%	Nervous system	3.9E-03
		Vinyl Chloride	3.0E-02	10%	Liver	1.3E-05	1%	Liver	3.7E-05	5%	Liver	5.7E-02	6%	Liver	8.7E-02
		Pathway-Specific Total:	**3E-01**			**1E-03**			**7E-04**			**9E-01**			**1E+00**
		Target-Organ Specific Hazard Indices													
		Nervous System	*3E-01*			*1E-03*			*7E-04*			*9E-01*			***1E+00***
		Immune System	*0E+00*			*0E+00*			*9E-06*			*5E-03*			***5E-03***
		Liver	*3E-02*			*1E-05*			*4E-05*			*6E-02*			***9E-02***

Notes:
HQ = Hazard Quotient; NA = Not Applicable.