Exhibit 576

Rebuttal Report by Richard J. Schuhmann, Ph.D.: Observations regarding expert reports of Lisa A. Bailey, Ph.D.

In re Camp Lejeune Water Litigation, **No. 7:23-cv-00897 (E.D.N.C.).**

Prepared by

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May 13, 2025

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I. Background.

I was retained by The Plaintiff Leadership Group and asked to review and comment on expert reports issued by Dr. Lisa Bailey relating to water contamination at Camp Lejeune, North Carolina. Dr. Bailey provided reports with similar methodological approaches and content applied to numerous Plaintiffs.[1] I reviewed her reports in general and reviewed several (cited below) more specifically with respect to the method(s) applied and conclusions associated with her evaluations. In addition to reviewing Dr. Bailey's reports, I have also reviewed discovery documents, documents in my files, and relevant Internet resources. These documents and resources are footnoted throughout this report as they are relied upon.[2]

While much of Dr. Bailey's report is premised upon spreadsheet calculations, these spreadsheets were not available to me. A precise evaluation of Dr. Bailey's calculations was therefore not possible and this report focuses on her assertions and methods. Because I may be asked to review data, such as those in Dr. Bailey's spreadsheets, documents, and reports as they may become available and provided to me in the future, I reserve the right to amend and/or revise my observations and opinions based upon this additional information.

II. Qualifications.

I hold a Ph.D. in environmental engineering from the Pennsylvania State University (Penn State), a Master of Science degree in environmental engineering from the University of Houston and a Bachelor of Science degree in geology from the University of New Hampshire. I taught engineering topics (*e.g.*, civil engineering, environmental engineering and engineering design) at Penn State and the Massachusetts Institute of Technology (MIT). These teaching topics included quantitative risk assessment in the context of environmental engineering at Penn State, and risk perception, risk analysis and risk management in the context of engineering project planning, management and leadership at both Penn State and MIT. I also supervised undergraduate (cross-disciplinary) and graduate (surface water hydrologic modeling) engineering thesis research at both institutions for over sixteen years. These projects included the design and operation of mechanical systems and stormwater modeling at Penn State, as well as a multi-year hydrologic modeling study in eastern Uganda for MIT.

I have more than 30 years of experience as a technical consultant in the assessment and evaluation of environmental and engineered systems including groundwater contamination, ambient air dispersion, and indoor air exposures (*e.g.*, volatile contaminants partitioning to air during showering, soil vapor intrusion, particle infiltration from ambient air) in the United States

[1] The reports are dated 4/8/25 and are for the following plaintiffs, categorized by disease: 1) Bladder Cancer: Jefferson Criswell, Terry Dyer, Mark Cagiano, Jimmy Laramore, Edward Raymond; 2) Kidney Cancer: Frank Mousser, Allan Howard, David Fancher, David Downs, Jacqueline Tukes; 3) Leukemia: Joseph Gleesing, Vivian Connard (Stephen Connard, decedent), Bruce Hill, Robert Fiolek, Karen Amsler; 4) Non-Hodgkin's Lymphoma: Frances Carter (Ronald Carter, decedent), Robert Kidd, Cometto Davis, Jose Vidana, Scott Keller.

[2] A reference list is also be provided at the end of this report

and overseas. I have specific experience with the CDC-ATSDR SHOWER model,[3] having downloaded V.3.0 in April 2023 and subsequently V.4.0 when it was released in September 2024.[4] I have also been authorized for access to the restricted Public Health Assessment Site Tool (PHAST)[5] since September 2024. I have managed multi-disciplinary teams of scientists and engineers on large multi-year, multi-media environmental evaluation projects associated with litigation. I have been recognized and testified in state court as an expert in the quantification of industrial emissions and the modeling of their transport and fate in the environment and recognized by state and federal courts as an expert in quantitative risk assessment.

My curriculum vitae and an abbreviated summary of consulting projects (1993 – 2025) are provided separately.

III. Overview.

This report consists of three sections. An overview of each section appears below:

A. Critiques of Sections 5 and 6: When creating plaintiff-specific risk evaluations, Dr. Bailey makes the following errors which undermine the accuracy and reliability of her assessments:

1) Dr. Bailey ignores the absence of necessary quantitative inputs (*e.g.*, a cancer slope factor applicable to TCE and bladder cancer or benzene and NHL) and instead applies inapplicable dose-response data in an attempt to, for litigation purposes, apply quantitative risk assessments for all chemicals to all plaintiffs and their specific disease(s).

2) Dr. Bailey's plaintiff-specific risk evaluation improperly concludes that any individual with an estimated cumulative exposure that does not exceed US EPA standard has an acceptable risk and therefore cannot prove causation.

3) Dr. Bailey inappropriately applies risk assessment principles to individuals who have already been diagnosed with a relevant illness in an effort to conclude that it was not caused by the exposure to contaminants in the Camp Lejeune water.

[3] For general information see ATSDR website, webpage entitled "Estimating Site-Specific Inhalation Exposures" at https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/epcs_and_exposure_calculations/estimating_inhalation_exposures.html (last visited 5/6/25).

[4] Note: V.4.0.1 was released November 19, 2024 as an update.

[5] *See generally* Burk T, Mellard D, Ulirsch GV, Li Z. Public Health Assessment Site Tool and Affiliated Applications: A Key Resource for Evaluating the Health Impact of Community Exposure to Hazardous Chemicals. J Environ Health. 2022 Nov. 85(4):40-42 (describing that ATSDR conducts public health assessments (PHAs) to investigate exposure to environmental contaminants, evaluate potential health effects, and develop action plans; to improve quality and consistency of PHA work, ATSDR developed PHAST).

4) Dr. Bailey's assertion that there exist acceptable, risk-free exposure levels for mutagenic carcinogenic chemicals is contradicted by the NRC, US EPA and ATSDR.

B. Critiques of Section 7: When creating plaintiff-specific margins of exposure (MoE), Dr. Bailey applies an MoE analysis to carcinogens, which is not supported by the US EPA and ATSDR, nor did she provide any literature references in support of this method. Therefore, the results of the MoE analysis are misguided and unreliable.

C. Other Observations: Dr. Bailey's statistics on the background incidence of cancers are misleading and irrelevant to Camp Lejeune.

IV. Discussion.

A. Critiques of Sections 5 and 6 of Dr. Bailey's Reports.

In Sections 5 and 6 of her reports, entitled Hazard Assessments and Toxicity Criteria and Plaintiff-Specific Regulatory Risk Evaluation, Dr. Bailey improperly endeavors to calculate cancer risks based upon exposure estimates for each the plaintiffs. Dr. Bailey makes the following errors:

1. Dr. Bailey ignores the absence of necessary quantitative inputs resulting in the unreliability of her calculated cancer risks.

In an attempt to apply the quantitative risk assessment method to all plaintiffs, their associated diseases, and the chemicals present in the water at Camp Lejeune, Dr. Bailey has, in the absence of necessary relevant quantitative parameters, applied irrelevant quantitative parameters, resulting in unreliable and irrelevant analyses.

Dr. Bailey states that her charge was to review materials relevant to the plaintiffs and develop opinions related to whether there is scientific support for the plaintiff's claim that exposure to chemicals in tap water while residing at Camp Lejeune is causally associated with the plaintiff's cancer diagnosis (*i.e.*, non-cancer disease plaintiffs are not part of the analysis).[6] Dr. Bailey states that as part of her evaluation, she "[a]pplied standard risk assessment methodology to conduct a risk evaluation for the plaintiff[s]."[7]

A "standard" quantitative risk assessment is performed establishing an estimated human population exposure level (lifetime average daily dose; lifetime average daily exposure concentration) and identifying appropriate dose-response data for the chemical(s) of interest (cancer slope factor; inhalation unit risk), and characterizes the product of those two values as an estimate of the lifetime excess cancer risk (ELCR) of that population for that specific chemical and disease. As Dr. Bailey writes, one of the most fundamental concepts in the field of toxicology

[6] *e.g.*, Bailey report, Karen Amsler, 4/8/25, page 2
[7] Bailey report, Karen Amsler, 4/8/25, page 9

is the dose-response relationship of a chemical(s) and its specific associated disease(s).[8] That relationship is quantified by a cancer slope factor (CsF) or inhalation unit risk (IUR) and is an essential element required to perform a "standard" quantitative assessment of risk.
ATSDR defined causative relationships between chemicals at Camp Lejeune and diseases including as follows in its 2017 publication:[9]

Disease	TCE	PCE	Benzene	VC
Bladder cancer		Sufficient		
Kidney cancer	Sufficient			
Leukemias	Equipoise		Sufficient	
Liver cancer	Equipoise			Sufficient
Multiple myeloma	Equipoise		Equipoise	
NHL	Sufficient	Equipoise	Sufficient	

These relationships differ from the quantitative relationships represented by dose-response CsF/IUR and available on the US EPA Integrated Risk Information System (IRIS):[10]

Disease	TCE	PCE	Benzene	VC
Bladder cancer		CsF/IUR		
Kidney cancer	CsF/IUR			
Leukemias			CsF/IUR	
Liver cancer	CsF/IUR			CsF/IUR
Multiple myeloma				
NHL	CsF/IUR	CsF/IUR		

The gaps between these two sets of causative information reveal limitations in the application of quantitative risk assessment (*i.e.*, where CsF/IUR data are not available):

Disease	TCE	PCE	Benzene	VC
Bladder cancer				
Kidney cancer				
Leukemias	Equipoise			
Liver cancer				
Multiple myeloma	Equipoise		Equipoise	
NHL		Equipoise	Sufficient	

[8] Bailey report, Karen Amsler, 4/8/25, page 10
[9] *See generally* ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases, Jan. 13, 2017, pp. 13-14; *see also* ATSDR, Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune, April 2018, p. 8 (citing same); *see also* Bove, F.J. et al., Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study, Environmental Health Perspectives, 132 (10), Oct. 2024, page 2, n. 9 (citing same)
[10] US EPA, Integrated Risk Information System (IRIS), Chemical Assessment Summaries for Perchloroethylene, 2012; Trichloroethylene, 2011; Vinyl Chloride, 2000; Benzene, 2003. EPA's IRIS Program identifies and characterizes health hazards of chemicals found in the environment. *See generally* EPA website, "Basic Information about the Integrated Risk Information System," available at https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system.

Lacking a CsF/IUR does not mean the causative link between the chemical and disease does not exist; however, what it does mean is that one cannot properly apply a quantitative risk assessment to establish a numerical risk.

Although the risk assessment process is predicated upon the existence of a CsF/IUR, Dr. Bailey calculated risks in the absence of these parameters for chemicals and diseases for which there are quantitative causative gaps (*i.e.*, lack of CsF/IUR). For example, she explained her method for lacking CsF/IUR for calculating the risk of NHL from benzene exposure this way for one plaintiff:[11]

> "The benzene toxicity criteria are based on leukemia as the most sensitive endpoint, and therefore are overly protective for NHL."[12]

It appears from this text that:

1. Dr. Bailey recognizes there is an association between benzene and NHL.[13]

2. Dr. Bailey also recognizes that a CsF/IUR for benzene-NHL has not yet been established.

3. Lacking this/these critical risk quantification parameter(s), Dr. Bailey uses the dose-response relationship for benzene and leukemia (*i.e.*, toxicity criteria), a completely different disease than NHL.

4. Dr. Bailey justifies this substitution by claiming it is "overly protective."

5. In order to quantitatively state it is overprotective, one must compare the CsF/IUR of leukemia, which exists, with the CsF/IUR of NHL, which does not.

Bailey's claim of "overly protective" is unsupportable. If one is uncertain as to the degree of the dose-response because that relationship has not yet been quantified, then it is impossible to know if calculations using unrelated data are "overly protective" (*i.e.*, quantitatively conservative). Until a dose-response relationship is established, it is not possible to know whether this assumption is conservative or not, rendering Dr. Bailey's calculations simply irrelevant.

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[11] *See* Bailey report for Frances Carter as Representative of Ronald Carter dated 4/8/25

[12] Bailey report for Carter, page 6

[13] *See, e.g.*, Rana I, Dahlberg S, Steinmaus C, Zhang L. Benzene exposure and non-Hodgkin lymphoma: a systematic review and meta-analysis of human studies. Lancet Planet Health. 2021 Sep;5(9):e633-e643. doi: 10.1016/S2542-5196(21)00149-2 ("Our findings suggest a causal link between benzene exposure and non-Hodgkin lymphoma, especially for diffuse large B-cell lymphoma."); Yang Liu, Jingxin Wang, Benzene exposure increases the risk of non-Hodgkin's lymphoma: a systematic review and meta-analysis of observational studies, Translational Cancer Research, Vol. 11, No. 6 (June 29, 2022) ("Benzene exposure was positively correlated with the incidence of NHL.").

Dr. Bailey repeats this peculiar method calculating a risk of NHL for vinyl chloride, a chemical with a CsF/IUR specifically associated with liver cancer, and then explicitly states that the results of her calculations while "not predictive" are, yet again "overly conservative", which is mathematically unsupportable; as such these statements are unreliable [emphasis added]:

> "Therefore, cancer risk calculations for benzene, PCE, and vinyl chloride are not predictive of, and are overly conservative for, NHL risk from exposure to these chemicals."[14]

Dr. Bailey then proceeds to estimate a plaintiff's overall excess lifetime cancer risk to all diseases for which she has, and doesn't have, cancer slope factors, even though the plaintiffs typically have a single specific disease, stating that "I conservatively apply the criteria for these chemicals to estimate the plaintiff's overall excess lifetime cancer risk."[15]

Dr. Bailey's risk calculations are based upon a mélange of unsound assumptions, and assertions regarding the conservative nature of the results mathematically unsupportable; at best, the quantitative risk assessment results are accurately described by Dr. Bailey as "not predictive".

A risk assessment should be conducted and presented in a manner which is consistent with EPA policy; it should be reasonable, *i.e.*, based on sound judgment, with methods and assumptions consistent with the current state-of-the-science. Dr. Bailey's methods suffer from inconsistency and contain methods and assumptions unsupported by the current state-of-the-science.

Each plaintiff typically suffers from a specific disease[16] related causally to the chemicals in the water at Camp Lejeune, rendering results of assessments for overall excess lifetime cancer risk from other diseases irrelevant, and conclusions based upon risk calculations for which there are no dose-response data available unreliable.

2. Dr. Bailey improperly concludes that any individual with an estimated cumulative exposure not exceeding a range of 1×10^{-6} to 1×10^{-4} has an acceptable risk and cannot show causation.

ATSDR explicitly states that there is not an acceptable cancer risk range for human health assessment and that the US EPA risk range of 1E-6 to 1E-4,[17] which is used to decide remedial clean-up actions at Superfund sites, should not be used as justification for what is acceptable or not acceptable in a human health assessment. In a remarkably direct and specific contradiction of ATSDR, Dr. Bailey explicitly states that the US EPA risk range of 1E-6 to 1E-4 is the acceptable cancer risk range for human health assessment and cites two over 30-year-old Superfund documents as literature support for this assertion.

[14] Bailey Report for Carter, page 6
[15] Bailey Report for Carter, page 6
[16] Note: Mr. Howard suffers from both kidney cancer and NHL.
[17] Dr. Bailey references the US EPA's ELCR (excess lifetime cancer risk) range of 1×10^{-6} (1 cancer case in 1,000,000 people exposed) to 1×10^{-4} (1 cancer case in 10,000 people exposed). Bailey report for Carter, page 4

Dr. Bailey then uses the 1×10^{-4} (1 cancer case in 10,000 people exposed) value as a causative bright line; if a plaintiff's calculated risk estimate is equal to or less than 1×10^{-4}, "then it does not exceed US EPA's acceptable excess cancer risk range" and "to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that" the plaintiff's specific "exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the" period of "years that he was" exposed "at Camp Lejeune were causally associated with his" disease.[18]

In her characterization of plaintiffs' cancer risks, Dr. Bailey writes [emphasis added]:[19]

> "US EPA has established a target ELCR range of 1×10^{-6} (1 cancer case in 1,000,000 people exposed) to 1×10^{-4} (1 cancer case in 10,000 people exposed); an exposure that may result in an ELCR that falls within this range, that is calculated using conservative assumptions, is considered acceptable (US EPA, 1990, 1991)."

This assertion of a bright line at 1×10^{-4} is refuted by multiple sources of ATSDR policy and guidance to health assessors [emphasis added]: [20]

> a. "ATSDR does not have an acceptable cancer risk range."[21]
>
> b. ATSDR instructs health assessors to "[a]void stating that the estimated cancer risk is within EPA's acceptable cancer risk range of 1E-4 to 1E-6. EPA uses this cancer risk range to decide remedial clean-up actions at Superfund sites. ATSDR should not use this range as justification for what is acceptable or not acceptable nor should we report it in our documents."[22]
>
> c. While ATSDR may present cancer results quantitatively, "[t]hese theoretical risk estimates are calculated assuming people have the same exposures (*e.g.*, the same soil concentration, soil ingestion rate, specified duration), and do not represent individual cancer risks or account for variation in exposure in people living around a site."[23]

The two references Dr. Bailey supplied in support of this assertion (US EPA, 1990, 1991) are, in fact, both specific to Superfund cleanup standards, which ATSDR has explicitly stated are inapplicable. Dr. Bailey then makes an inappropriate application of these references to individual human health risk assessment, as specifically directed against by ATSDR. As such, the method and results of Dr. Bailey's assessment are contradictory to ATSDR principles, US EPA intent, and are unreliable.

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[18] Elements of text taken from Bailey report for Carter, pages 7-8, but common to other reports.
[19] Bailey Report for Carter, page 16
[20] *See* ATSDR website, Public Health Assessment Guidance Manual (PHAGM), https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/indepth_toxicological_analysis/EvaluateEvidenceCancerEffects.html.
[21] From same.
[22] *Id.*
[23] *Id.*

Notwithstanding the significant specific misinterpretations and misapplications discussed above, when reviewed in context, the multi-decade old site remediation documents referenced in Dr. Bailey's report provide a meaningful context to the nature of the "cancer risk range of 1E-4 to 1E-6" site cleanup risk levels.

The 1990 reference in Dr. Bailey's report is a 35-year-old update to the 1985 CERCLA National Contingency Plan (NCP) from the Federal Register. Below are excerpts from the published rule associated with risk-based cleanup levels [emphasis added]:[24]

> "A few commenters supported the proposed risk range of 10^{-4} to 10^{-7}, though generally with qualifications. One commenter's position on the point of departure makes clear that they view the risk range only as a fallback when 10^{-6} cannot be attained.… Other reasons for opposing a risk range with a boundary at 10^{-7} are that such a range could lead to fewer cleanups of high-risk sites or less overall risk reduction…Based on the comments received, EPA has decided to revise the boundaries of the acceptable risk range for Superfund cleanups to 10^{-4} to 10^{-6} but to allow for cleanups more stringent than 10^{-6} when warranted by exceptional circumstances…EPA's preference, all things being equal, is to select remedies that are at the more protective end of the risk range. Therefore, when developing its preliminary remediation goals, EPA uses 10^{-6} as a point of departure…."

As can be seen from the text above, this reference applies specifically to a risk range for contaminated site cleanups, which may possess complexities that affect cleanup targets and outcomes, as can be seen explicitly in the text below, again from the 1990 Federal Register [emphasis added]:[25]

> "In the Superfund program, remediation decisions must be made at hundreds of diverse sites across the country. Therefore, as a practical matter, the remediation goal for a medium typically will be established by means of a two-step approach. First, EPA will use an individual lifetime excess cancer risk of 10^{-6} as a point of departure for establishing remediation goals for the risks from contaminants at specific sites. While the 10^{-6} starting point expresses EPA's preference for setting cleanup levels at the more protective end of the risk range, it is not a presumption that the final Superfund cleanup level will attain that risk level."

While EPA prefers cleanups that result in risk levels less than 10^{-6}, at some sites achieving this level of cleanup may be technologically unfeasible; therefore, the risk range widened to 10^{-4} allows EPA the ability to make site-specific judgments that may result in higher than preferrable residual risks (*e.g.*, 10^{-4}).

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[24] US EPA, National Oil and Hazardous Substances Pollution Contingency Plan (NCP), EPA 540-Z-00-001, PB92-963261, January 1992: Federal Register, Vol. 55, No. 46, Thursday, March 8, 1990, Proposed Rules, pages 8715-8716

[25] US EPA, National Oil and Hazardous Substances Pollution Contingency Plan (NCP), EPA 540-Z-00-001, PB92-963261, January 1992: Federal Register, Vol. 55, No. 46, Thursday, March 8, 1990, Proposed Rules, page 8717

The 1991 reference in Dr. Bailey's report is a 34-year-old, 11-page memorandum,[26] again associated with CERCLA Superfund cleanup criteria and consistent with the 1990 reference and other US EPA documents on the topic where, depending on site-specific information, the risk assessment results falling within the range of 10^{-4} to 10^{-6} may support a decision for no further action or for a remedial action at a contaminated site.[27] State remediation criteria often reflect the US EPA use of a lifetime excess cancer risk of 10^{-6} as a point of departure for establishing remediation goals for the risks from contaminants at specific sites. For example, in North Carolina "[t]he target cancer risk and hazard index values are established in law";[28] "[t]arget risks for individual chemicals are $1.0x10^{-6}$ for carcinogens",[29] a target level which is structurally embedded within North Carolina Department of Environmental Quality (NCDEQ) risk assessment equations.[30]

Regulatory balancing between risk, ability to remediate, and cost of remediation, is not restricted to the cleanup of contaminated dump sites and extends to drinking water as well [emphasis added]:[31]

> "In 1974, Congress passed the Safe Drinking Water Act. This law requires EPA to determine safe levels of chemicals in drinking water which do or may cause health problems. These non-enforceable levels, based solely on possible health risks and exposure, are called Maximum Contaminant Level Goals. The MCLG for trichloroethylene has been set at zero because EPA believes this level of protection would not cause any of the potential health problems described below. Based on this MCLG, EPA has set an enforceable standard called a Maximum Contaminant Level (MCL). MCLs are set as close to the MCLGs as possible, considering the ability of public water systems to detect and remove contaminants using suitable treatment technologies.
>
> The MCL has been set at 5 parts per billion (ppb) because EPA believes, given present technology and resources, this is the lowest level to which water systems can reasonably be required to remove this contaminant should it occur in drinking water."

The US EPA is clear that there is no safe level for TCE greater than zero; however, because of the need to balance technology and cost with the protection of human health, 5 ppb of TCE in

[26] US EPA, Memorandum, Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions, April 22, 1991.
[27] US EPA, RAGS Volume 3 Part A, Process for Conducting Probabilistic Risk Assessment, Chapter 2, Dec. 31, 2001.
[28] NCDEQ Risk Calculator User Guide, February 2024, page 13
[29] NCDEQ Risk Calculator User Guide, February 2024, page 18
[30] NCDEQ Risk Evaluation Equations and Calculations, July 2024: *e.g.*, Standard Carcinogenic Equation for Resident Water Ingestion, page 40
[31] US EPA, Consumer Factsheet on TCE, https://archive.epa.gov/water/archive/web/pdf/archived-consumer-fact-sheet-on-trichlorethylene.pdf.

drinking water is reflective of a feasible[32] standard that will present a health risk greater than zero (and greater than 10^{-6}).

In 1989, the US EPA was forced to wrestle with the concept of "acceptable" risk (*i.e.*, not a "safe" level) in context with community exposure to benzene, a hazardous air pollutant (National Emission Standards for Hazardous Air Pollutants, NESHAP, CAA) [emphasis added]:

> "The goal, which came to be known as the "fuzzy bright line", held that risks would be deemed acceptable if few, if any, individuals were exposed above a 1 in 10,000 lifetime cancer risk, and, as much of the exposed population as possible was below a lifetime risk of 1 in 1,000,000."[33]

For example, in a large city (*e.g.*, Houston, TX), where controlling emissions of benzene from industry is difficult, control technology would be required to keep the cancer risk for those few individuals in close proximity to the emission source(s) (*e.g.*, living near a refinery fenceline) at less than 1 in 10,000, while the majority of the city would be exposed to a risk below 1 in 1,000,000. This pragmatic risk management concept, whereby the US EPA engaged in balancing, is quite different from the unsupported view advanced by Dr. Bailey that all exposures that result in a risk less than 1 in 10,000 are not only acceptable but safe and are not causative to an individual.

By 1990, the Amendments to the Clean Air Act (CAA) specified that [emphasis added]:[34]

> "…for known, probable, or possible human carcinogens, the administrator is to promulgate revised standards if the MACT standards do not reduce the risk incurred by "the individual most exposed to emissions" from the source of pollution to less than one in a million."

As a conclusion to her report, Dr. Bailey uses the fallacious "acceptable" risk level of 10^{-4} she has established to arrive at specific causation opinions regarding plaintiffs' diseases [emphasis added]:[35]

> "Based on standard risk assessment methodology, which includes overly health-protective assumptions about exposure and risk, the maximum risk estimate calculated for Ms. Dyer's estimated exposures (1×10^{-4}, or 1 cancer cases in 10,000 exposed people, or 0.01% risk), for all the exposure scenarios evaluated, does not exceed US EPA's target excess cancer risk of 1 in 10,000 (i.e., 1 cancer case in 10,000 exposed people, or 0.01%).… The PCE risk estimates based on Ms. Dyer's estimated exposures (2×10^{-5}, or 2 cancer cases in 100,000 exposed people, or

[32] US EPA, The Safe Drinking Water Act as Amended by the Safe Drinking Water Act of 1996, PL 104–182, Aug. 6, 1996 (*e.g.*, page 12, para. (5))
[33] Committee on Risk Assessment of Hazardous Air Pollutants, National Research Council, *Science and Judgment in Risk Assessment*, 1994, App. A, page 321, https://www.ncbi.nlm.nih.gov/.
[34] 1990 Amendments to the Clean Air Act, Title III, § 301 (d)(9), found in NRC (2009) page 52
[35] Bailey report for Dyer, pages 7-8

> 0.002% risk) are within US EPA's acceptable cancer risk range. Therefore, the calculated ELCRs for Ms. Dyer are not reflective of bladder cancer risk… Based on the results of my analysis described above, it is my opinion, to a reasonable degree of scientific certainty, that there is insufficient evidence to conclude that Ms. Dyer's exposures to TCE, PCE, benzene, vinyl chloride, and 1,2-tDCE from tap water during the 14.5 years that she lived and spent time at Camp Lejeune are causally associated with her bladder cancer."

Dr. Bailey appears to arrive at this specific causation opinion based solely upon the application of quantitative risk assessment to a specific individual (*i.e.*, applying a theoretical risk estimate which is not representative of an individual cancer risk),[36] then interpreting the quantitative results through a flawed conceptual model of "acceptable" (*i.e.*, any risk less than 1E-04 is acceptable). Arriving at a specific causation opinion typically requires additional investigation and depth. For example, a specific causation opinion can be supported by what is commonly referred to as a "differential etiology" (DE). DE is succinctly defined by David Faigman, Dean of the University of California, Hastings College of the Law, in "The Judges' Book", as consisting of the following method:[37]

> 1. Diagnosis: an expert must accurately diagnose the illness from which the plaintiff suffers.
>
> 2. General causation: an expert must determine that the proffered cause of the plaintiff's injury is in fact capable of causing that kind of injury.[38]
>
> 3. Specific causation: an expert must determine that the general cause was in fact the cause of this particular plaintiff's injury. If general causation determines that a substance can cause the plaintiff's condition, specific causation assesses whether the substance did cause the plaintiff's condition.[39] In this third step, the appropriately qualified expert must rule out alternative causes based on scientific knowledge applied to the specifics of the case.

Dr. Bailey failed to perform a "specific causation" assessment of the type described above. The "methods" she has applied – "acceptable" ELCR and "MOE" (Margin of Exposure estimates, discussed below) are both flawed; they are unsupported and their results unreliable and misinterpreted. Her opinion is also absent any consideration of alternative causes; given the plaintiffs no longer have a "probability" or "risk" of getting their diseases – they have them – she

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[36] *Id.*

[37] Faigman, David L. (2022) "Evidence: A Brief Guide to Differential Etiology," The Judges' Book: Vol. 6, Article 9, *e.g.*, pages 56-57

[38] *See In re Camp Lejeune Water Litigation,* No. 7:23-CV-897 (E.D.N.C. June 5, 2024) ("Ordinarily, toxic exposure torts proceed in two steps - an expert demonstrates that a particular type of harm can be caused by the exposure to a degree of scientific certainty (general causation) and an expert opines that this plaintiff's exposure was a cause in fact of his or her harm (specific causation).")

[39] *See id.*

fails to consider alternative causes, or lack thereof, to their documented exposure to chemicals at Camp Lejeune. In other words, but for their chemical exposures at Camp Lejeune, what causative reasons exist for why the plaintiffs might have acquired their disease(s)?

3. Dr. Bailey misapplies risk assessment principles to individuals who have already been diagnosed with a relevant condition in an effort to conclude that it was not caused by the exposure to contaminants in the Camp Lejeune water.

Throughout her report, Dr. Bailey claims to employ "standard risk assessment methodology" to identify the "maximum risk estimate[s]" for the estimated exposure of individual plaintiffs.[40] But there is little standard about her application of that method. As discussed earlier, standard risk assessment and "theoretical risk estimates are calculated assuming people have the same exposures (*e.g.*, the same soil concentration, soil ingestion rate, specified duration), and do not represent individual cancer risks or account for variation in exposure in people living around a site."[41]

The primary objective of a regulatory risk assessment, like that applied by Dr. Bailey, is to provide decision-makers with a quantitative estimate of the health risks associated with a site-specific exposure. These calculations are used to screen sites as to their relative hazard, rank sites for remediation prioritization, and provide guidance for risk management decisions. While for large hypothetical populations these assessments provide reasonably accuracy and are clearly useful, any claim to their precision with respect to an individual is false.

When assessing whether a particular potentially toxic substance is a substantial contributing factor to an individual's disease or illness, regulatory assessments provide little value, even if much of the same toxicological and epidemiological data are used to evaluate causation. This is because the risk assessment procedure captures an imprecise estimate of the risk to a given population. Regulatory agencies use these population-scale estimates to determine whether a risk is "intolerable" and requires government intervention:

> "The resulting decisions—whether or not to regulate and, if so, the nature and form of regulation—seek to protect human health and the environment where appropriate, in part on the basis of scientific analysis and in part on the basis of consideration of information on costs, societal values, legal requirements, and other factors. As the proponent of any new regulation, EPA generally has the burden of proving that the proposed regulation meets statutory standards. That is not a requirement for EPA to prove "cause and effect "in the customary scientific sense, but rather to demonstrate by way of science-based analysis that the proposed regulation meets statutory criteria

[40] *e.g.*, Bailey report for Amsler, page 7
[41] *Id.*

related to adverse effects, unreasonable risks, and other statutory thresholds for regulation."[42]

In short, there are substantial differences in how toxicological data are used in a regulatory framework to protect public health versus how those data are used to make determinations regarding general or specific causation.

Like most scientific processes, risk assessment involves uncertainty; however, because its purpose to identify unacceptable outcomes requires accuracy but not strict precision,[43] U.S. EPA and other agencies appropriately rely on assumptions in the face of those uncertainties. Assumptions fill the gaps such as to how humans typically behave, the nature of the dose-response curve in the region of very low doses, or the relevance to humans of various toxicity responses observed in high-dose animal experiments. The principles and practices of choosing among available inference options were outlined in the seminal 1983 National Research Council report generally referred to as "The Red Book", which stated that "[t]he field has been developing rapidly, and the greatest improvements in risk assessment result from the acquisition of more and better data, which decreases the need to rely on inference and informed judgment to bridge gaps in knowledge.[44]

The assumptions a quantitative risk assessment relies upon undermine its ability to measure a "true risk" of a population much less that of an individual. The few attempts to investigate the accuracy of risk assessment calculations note their imprecision. After finding that "uncertainty in cancer risk estimates was huge, at least several orders of magnitude," one study described cancer risk estimates presented as a single number as "misleading"[45] and, contrary to Dr. Bailey's repeated characterization of all aspects of risk assessment as "conservative," the authors note that "[t]he cancer estimate resulting from linear extrapolation cannot even be regarded as conservative."[46]

While it's possible to compare the theoretical figures resulting from risk assessment calculations to real-world data on a population level, it is virtually impossible to validate the ELCR for an individual. The determination that someone has, say, a 1-in-1000 chance of developing a specific disease cannot be proven or disproven by whether that person gets sick with that disease or

[42] Committee on Improving Risk Analysis Approaches Used by the U.S. EPA, National Research Council, Science and Decisions: Advancing Risk Assessment, 978-0-309-12046-3, (2009), page 29

[43] This does not imply that risk assessment is not an exercise of scientific rigor; however, for the purposes of identifying where government intervention is necessary, it is of little value to distinguish between a risk of 2×10^{-4} and a risk of 4×10^{-4} - both are intolerable risks and likely will require remediation.

[44] National Research Council (US) Committee on the Institutional Means for Assessment of Risks to Public Health, Risk Assessment in the Federal Government: Managing the Process, National Academies Press (1983), page 6

[45] Wout Slob, Martine I. Bakker, Jan Dirk te Biesebeek, and Bas G.H. Bokkers, Exploring the Uncertainties in Cancer Risk Assessment Using the Integrated Probabilistic Risk Assessment (IPRA) Approach, Risk Analysis, Vol. 34, No. 8, 2014, page 17

[46] *Id.* page 17

not.[47] This is one of the reasons this type of quantitative approach typically functions at a population level.

Risk assessment is particularly imprecise when applied to actual individuals given its failure to consider inter-individual variabilities in human exposure and toxicological susceptibility. The EPA assumes "[t]he linear default approach for dose-response assessment [used for carcinogens] provides upper-bound calculation of potential risk at low doses," which is "thought to be public health protective at low doses for the range of human variation."[48] According to EPA, the use of a linear no-threshold model "adequately accounts for human variation unless there is case-specific information for a given agent or mode of action that indicates a particularly susceptible subpopulation or lifestage, in which case the special information will be used."[49] The rationale being that the linear-extrapolation procedure will generally overestimate the risk to an extent that will account for the underestimation bias related to the omission of inherent human heterogeneity. However, despite this generalization, the "EPA provides no evidence to support that assumption and in essence establishes a default (no variability in susceptibility) that is unsubstantiated."[50]

Individuals may differ in their "susceptibility to the toxic effects of a given chemical exposure because of such factors as genetics, lifestyle, predisposition to diseases and other medical conditions, and other chemical exposures that influence underlying toxic processes."[51] Studies have shown systematic inter-individual variabilities in human exposure and toxicological susceptibility due to a mixture of intrinsic and extrinsic factors (*e.g.*, genetics,[52] development stage,[53] dietary pattern,[54] and behavior.[55]) For example, smokers have been shown to be more susceptible to arsenic-induced lung cancer than non-smokers.[56] As increasing data show the depths of intra-species variability, some scientists have suggested current population cancer risk

[47] This is particularly true for ELCR which operate in the context of a baseline cancer risk.
[48] U.S. EPA, *Guidelines for Carcinogen Risk Assessment*, EPA/630/P-03/001F (March 2005) at A-9.
[49] *Id.* page A-9
[50] Committee on Improving Risk Analysis Approaches Used by the U.S. EPA, National Research Council, *Science and Decisions: Advancing Risk Assessment*, 978-0-309-12046-3, (2009), page 132
[51] *Id.* page 109
[52] *See e.g.,* Fenna, D., Mix, L., Schaefer, O., and Gilbert, J. A. L. (1971). Ethanol metabolism in various racial groups. *Can. Med. Assoc. J.* 105, 472–475; Zhang, F. F., Cardarelli, R., Carroll, J., Fulda, K. G., Kaur, M., Gonzalez, K., et al. (2011). Significant differences in global genomic DNA methylation by gender and race/ethnicity in peripheral blood. *Epigenetics* 6, 623–629
[53] *See e.g.,* Bearer, C. (1995). How are children different from adults? *Environ. Health Perspect.* 103, 7–12; Garí, M., and Grimalt, J. O. (2013). Inverse age-dependent accumulation of decabromodiphenyl ether and other PBDEs in serum from a general adult population. *Environ. Int.* 54, pages 119–127
[54] Awata, H., Linder, S., Mitchell, L. E., and Delclos, G. L. (2017). Biomarker levels of toxic metals among Asian populations in the United States: NHANES 2011-2012. *Environ. Health Perspect.* 125, pages 306–313
[55] *See e.g.,* Li, L., Hoang, C., Arnot, J. A., and Wania, F. (2020). Clarifying temporal trend variability in human biomonitoring of polybrominated diphenyl ethers through mechanistic modeling. *Environ. Sci. Technol.* 54, pages 166–175
[56] California Department of Health Services, Report to the air Resources Board on inorganic arsenic. Part B. health effects of inorganic arsenic. Air Toxicology and Epidemiology Section. Hazard Identification and Risk Assessment Branch. Department of Health Services: Berkeley; 1990, page 9-25

estimates may underestimate human variability, consequently underestimating an individual's risk.[57] Risk assessment inability to account for individuals is direct evidence it should not be applied with false precision to individuals.

The purpose of agency risk assessment is to provide regulatory decision-makers with a quantitative estimate of the health risks associated with a site-specific exposure. These estimates are then used to determine where a risk is "intolerable" and requires government intervention.[58] Instead, Dr. Bailey unreliably misuses this methodology to "inappropriately downplay or dismiss concerns about exposure" and suggest "levels "insufficient" for harm to occur, even when epidemiology or toxicology evidence suggests otherwise." [59]

4. The NRC, US EPA and ATSDR contradict Dr. Bailey's assertion that there exist acceptable, risk-free exposure levels for mutagenic carcinogenic chemicals.

Dr. Bailey rejects the plausibility of the non-threshold dose conceptual model. This model is not only plausible (*i.e.*, reasonable; probable), it is accepted by the National Research Council, the US EPA and the ATSDR.

Dr. Bailey writes [emphasis added]:

> "A nonthreshold mechanism of carcinogenesis implies that any level of exposure, even as low as a single molecule of a substance in a cell, potentially presents some level of response because of the possibility that (in theory) one molecule of the substance could react with DNA in a critical gene, and the consequent DNA damage could result in a mutation in that gene (permanent change in DNA) that could then result in carcinogenesis. However, this theoretical carcinogenic mechanism is not biologically plausible, even for carcinogens that are known to react directly with DNA. Several scientific reviews on this topic (*e.g.*, Cardarelli and Ulsh, 2018; Golden et al., 2019; Calabrese, 2023) describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk. Although a nonthreshold approach is reasonable to consider on a theoretical basis, the probability that it will occur in humans (*i.e.*, is it biologically plausible?) needs to be considered in the context of the high levels of DNA damage that human cells experience and efficiently repair on a daily basis."[60]

[57] Adam M. Finkel, EPA Underestimates, Oversimplifies, Miscommunicates, and Mismanages Cancer Risks by Ignoring Human Susceptibility, Risk Analysis (Oct. 21, 2014), Abstract; Varshavsky JR, Rayasam SDG, Sass JB, Axelrad DA, Cranor CF, Hattis D, Hauser R, Koman PD, Marquez EC, Morello-Frosch R, Oksas C, Patton S, Robinson JF, Sathyanarayana S, Shepard PM, Woodruff TJ. Current practice and recommendations for advancing how human variability and susceptibility are considered in chemical risk assessment. Environ Health. (Jan 12, 2023), page 15

[58] In 2017, ATSDR used risk assessment to show past exposures imposed intolerably high risks on workers and residents. Legislators used that information published in the Public Health Assessment of Camp Lejeune Drinking Water to inform their decision to enact the Camp Lejeune Justice Act.

[59] Laura N Vandenberg, et. al, Addressing systemic problems with exposure assessments to protect the public's health, 21 Environ Health 121 (2022), page 11

[60] *See* Bailey report for Dyer, page 19

Dr. Bailey has made assertions that are both contradictory and unsupported by literature [emphasis added]:

> 1. "A nonthreshold … theoretical carcinogenic mechanism is not biologically plausible, even for carcinogens that are known to react directly with DNA…. [a]lthough a nonthreshold approach is reasonable to consider on a theoretical basis."[61]
>
> a. Dr. Bailey at once asserts the approach is "not … plausible" (*i.e.*, not able to happen, exist, or be true,[62] not reasonable[63]) and that it is "reasonable to consider."
>
> 2. "Several scientific reviews on this topic (*e.g.*, Cardarelli and Ulsh, 2018; Golden et al., 2019; Calabrese, 2023) describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk."[64]
>
> Reviewing the cited publications:
> a. In Calabrese, 2023: The author presents, while cautioned by his editors, the controversial concept of "hormesis", whereby low doses of carcinogens are thought to be beneficial.[65] The author does not describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk.
>
> b. In Golden et al, 2019: The authors conclude that "[i]t was not an objective of this project, however, to propose alternative approaches to cancer risk assessment beyond LNT [Linear No-threshold]…".[66] The authors do not describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk.
>
> c. In Cardarelli and Ulsh, 2018: The authors conclude that there is no scientific consensus for using the LNT model specifically in LDDR [low dose, low-dose rate] radiation environments.[67] The authors do not describe that there is no scientific consensus regarding the use of the nonthreshold approach for estimating cancer risk.

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[61] *Id*., page 19.
[62] Cambridge Dictionary: https://dictionary.cambridge.org/us/thesaurus/implausible
[63] Oxford Dictionary: https://www.oxfordlearnersdictionaries.com/definition/english/implausible
[64] Bailey report for Dyer, page 19.
[65] Calabrese, EJ. 2023. "Dose-response: A fundamental concept in toxicology." In Hayes' Principles and Methods of Toxicology (Seventh Edition). (Eds.: Hayes, AW; Kobets, T), CRC Press, Boca Raton, FL., p 95-141, page 141
[66] Golden, R; Bus, J; Calabrese, E. 2019. "An examination of the linear no-threshold hypothesis of cancer risk assessment: Introduction to a series of reviews documenting the lack of biological plausibility of LNT." Chem. Biol. Interact. 301:2-5, page 4
[67] Cardarelli, JJ II; Ulsh, BA. 2018. "It is time to move beyond the linear no-threshold theory for low-dose radiation protection." Dose Response 16(3), page 1

While the papers above are at best equivocal, disagreement with a scientific consensus does not destroy the consensus. There are "scientists" who do not agree with the consensus regarding an anthropogenic influence on climate change,[68] or that the earth resides within a heliocentric system;[69] this does not change the fact that there exists a consensus on each of these topics, outside of which these "scientists" reside. At best, the literature Dr. Bailey points to indicates that these select authors may not agree in part with the scientific consensus.

Setting aside the inconsistency of Dr. Bailey's syntax and the problems with the three papers she references, other authorities have been consistent with respect to the world view within the scientific community regarding a lack of threshold for carcinogens. For example, the National Research Council wrote in 1977 regarding "safe" levels of carcinogens [emphasis added]:

> "With respect to carcinogenesis, it seems plausible at first thought, and it has often been argued, that a threshold must exist below which even the most toxic substance would be harmless. Unfortunately, a threshold cannot be established experimentally that is applicable to a total population. A time-honored practice of classical toxicology is the establishment of maximal tolerated (no-effect) doses in humans based on finding a no-observed-adverse-effect dose in chronic experiments in animals, and to divide this dose by a "safety factor" of, say, 100, to designate a "safe" dose in humans. There is no scientific basis for such estimations of safe doses in connection with carcinogenesis."[70]

A well-worn argument voiced by Dr. Bailey in her report is "the fact that they [regulatory criteria] often reflect exposure levels that are much lower than the exposure levels in the animal or human studies at which effects were reported".[71] In other words, the animal studies used very high concentrations of the subject chemical which are unrepresentative of the exposure concentrations that humans would be exposed to. The National Research Council addressed this argument when writing in 1977 regarding "safe" levels of carcinogens:

> "To obtain statistically valid results from such small groups of animals requires the use of relatively large doses so that effects will occur frequently enough to be detected. For example, an incidence as low as 0.01% would represent 20,000 people in a total population of 200 million and would be considered unacceptably high, even if benefits were sizable. To detect such a low incidence in experimental animals directly would require hundreds of thousands of animals. For this reason, we have no choice but to give large doses to relatively small experimental groups and then to use biologically reasonable models in extrapolating the results to estimate risk at low doses. Several methods of making such calculations have been

[68] https://en.wikipedia.org/wiki/Frederick_Seitz.
[69] https://en.wikipedia.org/wiki/Robert_Sungenis.
[70] Safe Drinking Water Committee, Advisory Center on Toxicology, Assembly of Life Sciences, National Research Council, Drinking Water and Health, National Academy of Sciences, 1977, page 54
[71] Bailey report for Dyer, Section 3.5, page 21

> considered and used, but we think that the best method available to us today is to assume that there is no threshold, and that the incidence of tumors is directly proportional to dose. However, it is important to recognize that such calculations may give either too small or too large an estimate risk. The actual risk to humans might be even greater over a human lifetime, because it is about 35 times that of a mouse; and there is evidence that the risk of cancer increases rapidly with the length of exposure. Moreover, experimental assays are conducted under controlled dietary and environmental conditions with genetically homogeneous animals, whereas humans live under diverse conditions, are genetically heterogeneous, and are likely to include subpopulations of unusual susceptibility. It should be emphasized that these general considerations give only a minimal estimate of human risk; moreover, they do not take into consideration differences in susceptibility between species. For example, beta-naphthylamine is well established as a human carcinogen on the basis of epidemiologic studies of occupationally exposed workers, whereas experiments have not shown it to be carcinogenic in the hamster, which is relatively resistant."[72]

With respect to the precautionary principle in the context of radiation, a topic covered in each of the three papers referenced by Dr. Bailey, the National Research Council wrote in 1990:[73]

> "It must be emphasized again that virtually all mutations have harmful effects. Some mutations have drastic effects that are expressed immediately, and these are eliminated from the population quite rapidly. Other mutations have milder effects and persist for many generations, spreading their harm among many individuals in the distant future.
>
> "Use simple linear extrapolation between the lowest reliable dose data and the spontaneous or zero dose rate. In order to get any kind of precision from experiments of manageable size, it is necessary to use dosages much higher than those expected for the human population. Some mathematical assumption is necessary, and the linear model, if not always correct, is likely to err on the safe side."

Writing in 2009, the NRC summarized the state of policy regarding a threshold for carcinogens:[74]

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[72] Safe Drinking Water Committee, Advisory Center on Toxicology, Assembly of Life Sciences, National Research Council, Drinking Water and Health, National Academy of Sciences, 1977, pages 55-56

[73] Committee on the Biological Effects of Ionizing Radiations Board on Radiation Effects Research Commission on Life Sciences National Research Council, Health Effects of Exposure to Low Levels of Ionizing Radiation Beir V, National Academy Press, 1996, page 84, page 67

[74] National Research Council (NRC). 2009. "Toward a unified approach to dose-response assessment: The need for an improved dose-response framework." In Science and Decisions: Advancing Risk Assessment. Committee on Improving Risk Analysis Approaches Used by the US EPA. The National Academies Press, Washington, DC. pages 127-128, page 130

> "Dose-response assessments for carcinogenic end points have been conducted very differently from noncancer assessments. For carcinogens, it has been assumed that there is no threshold of effect, and dose-response assessments have focused on quantifying the risk at low doses…In practice, EPA carcinogen assessments do not account for differences among humans in cancer susceptibility other than from possible early-life susceptibility…if a compound is determined to be 'DNA reactive and [to] have direct mutagenic activity' or to have high human exposures or body burdens 'near doses associated with key precursor events', a no-threshold approach is applied; risk below the POD is assumed to decrease linearly with dose. For carcinogens with sufficient MOA data to conclude nonlinearity at low doses, such as those acting through a cytotoxic MOA, the RfD approach outlined above for noncancer end points is applied…Effects of exposures that add to background processes and background endogenous and exogenous exposures can lack a threshold if a baseline level of dysfunction occurs without the toxicant and the toxicant adds to or augments the background process. Thus, even small doses may have a relevant biologic effect… Human variability with respect to the individual thresholds for a nongenotoxic cancer mechanism can result in linear dose-response relationships in the population."

TCE is a carcinogen and a mutagen. Mutagens cause DNA mutations that can lead to altered gene expression, loss of function, or even the development of cancer, with the genetic effects of mutagenic chemicals expressed almost immediately or potentially even multiple generations later in the great-grandchildren of children exposed to chemicals at Camp Lejeune. In contrast to the three papers offered by Dr. Bailey, additional quotes in support of the non-threshold approach and its consensus in the scientific community appear below as examples [emphasis added]:

> "The distinction between genotoxic and non-genotoxic carcinogens has traditionally been regarded as particularly relevant for risk assessment, with the assumption of the existence of no-effect concentrations (threshold levels) in case of the latter group. In contrast, genotoxic carcinogens, their metabolic precursors and DNA reactive metabolites are considered to represent risk factors at all concentrations since even one or a few DNA lesions may in principle result in mutations and, thus, increase tumour risk."[75]

> "For all mutagens, there may be a level of exposure below which chemical-induced mutation levels cannot be distinguished from background…In most cases, however, the available evidence is insufficient to enable a conclusion on the existence of a threshold, and the risk assessment should proceed as if there is no threshold. This is because even should a threshold exist, there would be

[75] Andrea Hartwig et al, Mode of action-based risk assessment of genotoxic carcinogens, Archives of Toxicology (2020) 94:1787–1877, page 1787

considerable uncertainty, potentially by orders of magnitude, as to the dose at which it occurs."[76]

"The principle of Paracelsus cannot be applied to the regulation of genotoxic chemicals. Genotoxic chemicals are substances that interact with DNA and may subsequently induce mutations. Owing to their DNA interaction properties, genotoxic chemicals are not considered to have a safe threshold or dose (2-4)."[77]

"For carcinogen risk assessment, NIOSH generally treats exposure-response as low-dose linear unless a non-linear mode of action has been clearly established, in which case NIOSH will adopt a modeling approach defined by the data (including non-linear approaches when appropriate). In general, whether the model forms are linear or non-linear, any nonzero exposure to a carcinogen is expected to yield some excess risk of cancer…Genotoxic ("DNA-damaging") carcinogens are presumed to act via non-threshold mechanisms… Carcinogens that act through non-genotoxic mechanisms (e.g., endocrine-modification, tumor-promotion, immunosuppression, and inflammation) or through indirect mechanisms (such as genotoxicity secondary to cytolethality and cell proliferation) may have responses at low doses that are nonlinear, including a threshold below which there is no added risk [Streffer et al., 2004]. Any potential threshold for a carcinogen can be adequately modeled by a sublinear, but non-threshold, mathematical model. Because of this, it is highly unlikely that one can demonstrate empirically that a threshold exists [Crump 2011]. Therefore, NIOSH acknowledges that even when a threshold cannot be empirically demonstrated, in some cases the true risk at low doses may be zero."[78]

"Trichloroethylene is a non-threshold carcinogen for which no threshold can be determined below which exposure would be safe." [79]

[76] Hazard Identification and Characterization, EHC 240: Principles for Risk Assessment of Chemicals in Food, Section 4.5, Genotoxicity, World Health Organization, Second edition (2020), page 4-51

[77] Takehiko Nohmi, Division of Pathology, Biological Safety Research Center, National Institute of Health Sciences, Kanagawa, Japan, Thresholds of Genotoxic and Non-Genotoxic Carcinogens, Toxicol. Res. Vol. 34, No. 4, (2018), page 282

[78] NIOSH [2016]. Current intelligence bulletin 68: NIOSH chemical carcinogen policy. By Whittaker C, Rice F, McKernan L, Dankovic D, Lentz TJ, MacMahon K, Kuempel E, Zumwalde R, Schulte P, on behalf of the NIOSH Carcinogen and RELs Policy Update Committee. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2017-100, page 19

[79] ECHA, European Chemicals Agency, Annex XV Report, An assessment of whether the use of Trichloroethylene in articles should be restricted in accordance with Article 69(2) of REACH, 2022, page 2

The US EPA applies a non-threshold approach to mutagenic carcinogens (*e.g.*, TCE) and non-mutagenic carcinogens in the absence of data indicating the contrary:[80]

> "Under the guidelines, linear extrapolation is appropriate when the evidence supports the mode of action of gene mutation due to direct DNA reactivity or another mode of action that is thought to be linear in the low dose region. A linear mode of action will also be the approach when available evidence is not sufficient to support a nonlinear extrapolation procedure, even in the absence of evidence of DNA reactivity."

The ATSDR also applies a non-threshold approach to all carcinogens "because cancer risk is a linear response without a threshold and very low doses can still cause an increased risk of cancer."[81]

B. Critiques of Section 7 of Dr. Bailey's Reports.

In Section 7 of her report, entitled Plaintiff-Specific Margins of Exposure, Dr. Bailey applies a Margin of Exposure (MoE) estimate, wherein she attempts to compare the exposures predicted for each of the bellwether plaintiffs and the exposure levels at which health effects have been observed to occur in human or extrapolated animal studies. Dr. Bailey's application of MoE in this context is unsupported and directed against by regulatory agencies. The US EPA provides guidance for application of the MOE only to performing non-cancer risk assessments at Superfund sites; the ATSDR states explicitly that the MOE can be applied "for noncancer effects only". Dr. Bailey's report contains less than 370 words (*i.e.*, ~3/4 page) associated with her MOE method, and this meager text is devoid of any supporting references.[82] The lack of support for this method and caution against its application by regulatory agencies renders the associated conclusions unreliable.

The ATSDR is clear on the value of an MOE evaluation for non-carcinogens (*e.g.*, developmental, neurological, toxicants), which Dr. Bailey has not performed [emphasis added]:

> "When evaluating the non-cancer public health implications of an exposure dose or concentration above an MRL, one useful tool is the Margin of Exposure (or MOE). The MOE is the effect level dose or concentration (for air) derived from a study divided by the exposure dose or concentration (for air) from your site. The

[80] US EPA website, Risk Assessment for Carcinogenic Effects, available at https://www.epa.gov/fera/risk-assessment-carcinogenic-effects#:~:text=Under%20the%20guidelines%2C%20linear%20extrapolation,per%20cubic%20meter%20of%20air (last visited 5/6/25).

[81] ATSDR website, Public Health Assessment Guidance Manual (PHAGM), Evaluate the Evidence to Examine Cancer Effects, available at https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/indepth_toxicological_analysis/EvaluateEvidenceCancerEffects.html (last visited 5/6/25).

[82] In Dr. Bailey's reports, see text in section 7 associated with MOE, and similar text in section 3.6.

> MOE should be part of your overall weight-of-evidence evaluation in the public health implications section."[83]

Furthermore, the ATSDR provides guidance for the MOE approach for non-carcinogens on its website and in fact directs readers to use the same ratio of 1 that Dr. Bailey inappropriately uses for carcinogens to evaluate measured exposure doses to determine non-carcinogenic significance (*e.g*, a ratio of LOAEL:site-specific exposure dose or concentration <1 is accepted as problematic for non-carcinogenic exposure):

> "…an MOE below 1 indicates that your site-specific dose is above the health effect study level which likely indicated harmful effects are possible. An MOE above 1 means your site-specific dose is below the health effect study level and a health assessor will need to make a professional judgement with the help of a toxicologist as to whether harmful effects are possible".[84]

In contrast, the ATSDR website is devoid of any guidance directing health assessors to use this method for evaluating carcinogens as Dr. Bailey did, much less to apply the target ratio of 1 applicable to non-carcinogens. In fact, an ATSDR toxicological evaluation training module provides direction to the contrary [emphasis added]:[85]

> "For noncancer effects only, you may consider numerically comparing the site-specific doses or concentrations to the study's health effect doses or concentrations. [Health assessors can make comparisons by] dividing the critical study's NOAEL, LOAEL … by the site-specific exposure dose…. The resulting value is often referred to as a margin-of-exposure (MOE)."[86]

> "Do not calculate a comparison between the cancer effect level (CEL) and site dose to assess cancer effects."[87]

ATSDR guidance also states:

> "Health assessors should not do the following: Conduct a comparison between the cancer effect level (CEL; the lowest dose level observed to produce a significant increase in the incidence of cancer or tumors) and site exposure dose

[83] ATSDR, DCHI Guidance & Clearance News, Update for ATSDR Health Assessors Including APPLETREE Partners (Internal Use Only), January 2020

[84] ATSDR website, Public Health Assessment Guidance Manual (PHAGM), Evaluate the Evidence to Examine Non-Cancer Effects, available at https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/indepth_toxicological_analysis/EvaluateEvidenceNon-CancerEffects.html (last visited 5/6/25)

[85] ATSDR, Mini-Module: Toxicological Evaluation slides 28-29, available at https://www.atsdr.cdc.gov/training/pha-training-section1/Module7/html/Toxicological_Evaluation_Mini_Module-508.pdf (last visited 5/6/25)

[86] *Id.*

[87] *Id.*, slides 69-70

to assess cancer effects. This is because cancer risk is a linear response without a threshold and very low doses can still cause an increased risk of cancer."[88]

"Health assessors should not … use a CEL to make a health hazard conclusion."[89]

The US EPA also offers written guidance associated with the use of the MOE approach for Superfund sites exclusively under the section titled [emphasis added]: "Performing a Non-Cancer Human Health Risk Assessment for SF Submissions".[90] There is no guidance for applying and MOE to carcinogens.

C. Other Observations.

1. Dr. Bailey's statistic on the background incidence of all cancers is misleading and irrelevant to Camp Lejeune.

Dr. Bailey appears to imply that because the incidence of cancer in general among Americans is 40% that the plaintiffs already had a 40% chance of acquiring their specific disease without the exposure to chemicals at Camp Lejeune.

Dr. Bailey writes:

> "To provide perspective on what a target ELCR of 1 in 10,000 or 1 in 1,000,000 means, it is helpful to understand how these risks compare to the overall lifetime probability of being diagnosed with cancer. According to the American Cancer Society (ACS), the lifetime probability of developing any cancer (i.e., background lifetime cancer risk for all cancers combined) is approximately 40% on average across the population (ACS, 2024). Individual risk will vary and is based on a number of different factors, including age, sex, race, lifestyle (e.g., diet, exercise), and family history."[91]

This statement is irrelevant with respect to plaintiffs given this statistic applies to over 100 cancer types,[92] most of which are not the specific diseases associated with the specific chemicals present in drinking water at Camp Lejeune; the plaintiffs do not have "all cancers."

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[88] ATSDR website, Public Health Assessment Guidance Manual (PHAGM), Evaluate the Evidence to Examine Cancer Effects, available at https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/indepth_toxicological_analysis/EvaluateEvidenceCancerEffects.html (last visited 5/6/25)

[89] *Id.*

[90] US EPA, Sustainable Futures / P2 Framework Manual, 2012, EPA-748-B12-001, 13. Quantitative Risk Assessment Calculations (https://www.epa.gov/sites/default/files/2015-05/documents/13.pdf)

[91] *e.g.,* Bailey report on Amsler, page 4

[92] https://my.clevelandclinic.org/health/diseases/12194-cancer

In addition, "[i]n the United States, over one-third of cancer deaths are attributed to diet, lack of physical activity, and obesity while another third relates to exposure to tobacco products".[93] Statistics on specific cancers, such as those associated with exposure to chemicals present at Camp Lejeune, are considerably less dramatic. For example, according to the National Institute of Health (NIH), while the lifetime probability of developing any cancer (*i.e.*, background lifetime cancer risk for all cancers combined) is approximately 39 percent on average across the population, only "approximately 0.1 percent of men and women will be diagnosed with acute lymphocytic leukemia at some point during their lifetime". [94] In addition, according to the American Cancer Society, "[t]here are only a handful of known risk factors for acute lymphocytic leukemia (ALL)";[95] these risk factors include: (1) exposure to high levels of radiation, and (2) exposure to certain chemicals.

D. Conclusion.

It is my expert opinion to a reasonable degree of scientific certainty that Dr. Bailey's reports and analyses have the defects and inadequacies as I have identified above.

[93] Inequalities in Environmental Cancer Risk and Carcinogen Exposures: A Scoping Review, Int. J. Environ. Res. Public Health 2023, page 2

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