

**IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DIVISION
No. 7:23-cv-00897**

IN RE:)	
)	
CAMP LEJEUNE WATER LITIGATION)	NOTICE OF FILING—PURSUANT
)	TO LOCAL RULE 79.2 AND
)	ELECTRONIC CASE FILING
This Document Relates To:)	ADMINISTRATIVE POLICIES
ALL CASES)	AND PROCEDURES MANUAL
)	(V)(G)(1)(E)
)	

The United States files this Notice of Filing pursuant to Local Rule 79.2 and Electronic Case Filing Administrative Policies and Procedures Manual (V)(G)(1)(e) and states that the following documents filed at D.E. 411 have been designated as “confidential” by Plaintiffs’ Leadership Group (“PLG”) pursuant to the Second Amended Stipulated Protective Order (Case Management Order No. 15), (D.E. 266), and presumably contain confidential and/or sensitive information and are, therefore, not being filed publicly on the docket:

1. Exhibit 5, Rough Draft of the Deposition Transcript of Thomas Longo, M.D.¹
2. Exhibit 16, Report by Damian A. Laber, M.D. – *Fiolek v. United States*
3. Exhibit 17, Report by Damian A. Laber, M.D. – *Gleesing v. United States*
4. Exhibit 18, Report by Richard T. Hoppe, M.D.– *Davis v. United States*
5. Exhibit 20, Rough Draft of the Deposition Transcript of Richard Hoppe, M.D.
6. Exhibit 21, Report by Paul J. Michaels, M.D. – *Vidana v. United States*

¹ Under the Second Amended Stipulated Protective Order, transcripts of depositions are deemed confidential for a period of thirty (30) days, and therefore, the United States also has filed Exhibits 5 and 20 provisionally under seal. The United States has redacted certain information in the remaining exhibits; none of the redacted information is relevant to the Motion.

Dated: June 23, 2025

Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that on June 23, 2025, I electronically filed the foregoing using the Court's Electronic Case Filing system, which will send notice to all counsel of record.

/s/ Joshua G. Carpenito
Joshua G. Carpenito

Exhibit 5

UNCERTIFIED ROUGH DRAFT

1 ROUGH DRAFT DISCLAIMER

 - - - - -

2 IMPORTANT NOTICE
3 AGREEMENT OF PARTIES

4 We, the party working with rough draft
5 transcripts, understand that if we choose to use
6 the rough draft screen or the printout, that we
7 are doing so with the understanding that the
8 rough draft is an uncertified copy.

9 We further agree not to share, give, copy, scan,
10 fax or in any way distribute this rough draft in
11 any form (written or electronic) to any party.
12 However, our own experts, co-counsel, and staff
13 may have LIMITED INTERNAL USE of same with the
14 understanding that we agree to destroy our rough
15 draft and/or any electronic form, if any, and
16 replace it with the final transcript upon its
17 completion.

18 By accepting a rough draft transcript, I am
19 hereby agreeing to the above-mentioned terms, and
20 I further agree to pay for these reporting
21 services that have been provided. I also
22 understand that receipt of this rough draft will
23 constitute an order for the final transcript.

24 WITNESS: Dr. Thomas Longo

25 DATE: June 16, 2025

26 REPORTER'S NOTE:

27 Since this deposition has been in rough draft
28 form, please be aware that there may be a
29 discrepancy regarding page and line number when
30 comparing the rough draft with the final
31 transcript.

32 Also, please be aware that the uncertified rough
33 draft transcript may contain untranslated steno,
34 reporter's notes in asterisks, misspelled proper
35 names, incorrect or missing Q/A symbols or
36 punctuation, and/or nonsensical English word

19 in that region.

20 Q. And so is Wilmington North Carolina;
21 correct?

22 A. Correct. That's the they're all in
23 close proximity.

24 Q. You conclude for both Mr. Criswell
25 and Ms. Dyer it is at least as likely as not that

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1 their exposure to chemicals in the water at Camp
2 Lejeune caused their bladder cancer; correct?

3 A. Are that is correct.

4 Q. And this conclusion was based on your
5 differential etiology that you performed in both
6 cases; right?

7 A. Yes, it is.

8 Q. And your differential etiology ruled
9 in exposure to the chemicals at Camp Lejeune in
10 part based on Dr. Reynolds exposure calculations;
11 right?

12 MR. WALLACE: Objection.

13 THE WITNESS: Yes, I did.

14 BY MS. GADDY:

15 Q. And you determined that the
16 epidemiological literature was sufficient to rule

17 in the chemicals at Camp Lejeune despite not
18 being an epidemiologist yourself; correct?

19 A. Not quite. So I did my own
20 literature review, looked at the studies that
21 exist both epidemiological and at the data that
22 exists for each individual compound alone and in
23 conjunction, drew my own independent conclusions
24 but then I deferred to the epidemiologists that
25 are trained to do that to make certain that what

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1 I thought I was reading was, in fact, supported
2 by a trained epidemiologist. I found that my
3 opinion was agreed with our epidemiologist. But
4 I would defer to the epidemiologist to really go
5 through the my insurance awe of each study and
6 depend papers. So yes, I'm conversant in those
7 types of studies I should be able to read them
8 and understand what's going on. Had I read it
9 and come to a different conclusion I would have
10 either said that that I was I disagree or I'd
11 have to educate myself in order to understand the
12 conclusions that were being drawn by the
13 epidemiologists.

14 Q. Okay. And the same way -- in the

15 same sense that you're not a epidemiologist with
16 respect to the chem calculation at Camp Lejeune
17 and bladder cancer you're also not trying to look
18 at the epidemiological studies around chewing
19 tobacco and bladder cancer; right?

20 A. Well I am trained in order to read
21 those studies. I don't have a formal diploma at
22 the end. So no, I don't consider myself an
23 expert. As a trained urologic oncologist I'm
24 expected to be able to read those papers and
25 understand the data that's being put forth.

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1 Q. Okay. In your own review as the
2 urologic oncologist of the epidemiological
3 literature about the chemicals at Camp Lejeune
4 and bladder cancer you reached your own
5 conclusion that the literature was sufficient;
6 right?

7 A. I independently confirmed what our
8 epidemiologists have said, yes.

9 Q. Okay. But again you determined that
10 the literature was insufficient when it came to
11 secondhand smoke as a risk factor for bladder
12 cancer; right?

13 any studies for your report?

14 A. No.

15 Q. You also state in your reports that
16 you reviewed the plaintiffs general causation
17 reports as we discussed; right?

18 A. Yes, I did.

19 Q. And you also state that you
20 incorporated and relied on those general
21 causation reports for your opinions; right?

22 A. Yes,ly I did.

23 Q. How did you decide which studies to
24 include or not include in your reporteds?

25 A. Again I'll rely epidemiologist and

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1 toxicologists and exposure to experts. But you
2 look for patients -- you look at the population
3 in the report and how close it matches the
4 question that you're asking. So you would
5 include anybody that had contaminated water from
6 obviously if you can get Camp Lejeune and there
7 was a Camp Pendleton that came out in the paper.
8 Those two populations match as closely we could
9 hope for. You look for other the dry cleaning
10 workers because we ever to those chemicals

11 talking about and it was felt that this water
12 contamination came from the dry cleaning worker.
13 You would look for contaminated water populations
14 because, again, that tends to match the
15 population that we're speaking about.

16 Q. Okay. And you have an entire section
17 in your report entitled general causation; right?

18 A. Yes.

19 Q. And I'll direct your attention to
20 we're looking at the Criswell report which is
21 Exhibit 2, I believe your general causation
22 section begins on page 11 and it continues until
23 page 14.

24 A. Yes.

25 Q. Now going to direct your attention to

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1 what I'll mark as Exhibit 14.

2 (Exhibit 14 marked for
3 identification.)

4 BY MS. GADDY:

5 Q. I'll just briefly scroll here. It's
6 a long document.

7 Do you recognize this, Doctor?

8 A. No, I do not.

9 Q. This is one of the specific causation
10 reports reported by John Sfakianos right?

11 MR. WALLACE: Objection foundation.

12 THE WITNESS: Na's what the title
13 said I've not.

14 BY MS. GADDY:

15 Q. Arrest you are you aware of
16 Dr. Sfakianos is another one of plaintiff's
17 urology experts in this case?

18 A. No I am not.

19 Q. Okay. So you have not reviewed his
20 reports in this case; right?

21 A. No, I have not.

22 Q. And turning to page 9, starting on
23 page 9 do you see where it says general
24 causation?

25 A. Yes, I do.

↑

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1 Q. And I'll just briefly scroll here
2 through here. Does this look to be the same
3 general causation section that is in your
4 reports?

5 MR. WALLACE: Objection.

6 THE WITNESS: It's a 49 page

7 document. I haven't read the whole thing.

8 BY MS. GADDY:

9 Q. Would you like to take time to read
10 this section and compare it to your report?

11 A. Certainly.

12 Q. We can go off the record while?

13 MR. WALLACE: No we can stay on
14 you're asking him to stay on it's okay he
15 spend time on the record doing it.

16 MS. GADDY: Okay that's fine.

17 THE WITNESS: Can you scroll down?

18 BY MS. GADDY:

19 Q. Sure.

20 A. Can you go down to the next page
21 please.

22 Q. Sure.

23 A. And can you continue to scroll.

24 And then on to the next page.

25 Okay.

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UNCERTIFIED ROUGH DRAFT

1 Q. Okay. Thank you for reviewing.

2 Understanding that you have not reviewed

3 Dr. Sfakianos's report until today does this look

4 to be the same section on general causation that

5 is in your reports?

6 A. Yes it looks the same.

7 Q. Okay. Were you asked to provide your
8 section on general causation to anyone?

9 A. I provided several drafts of my
10 report to the counsel.

11 Q. Okay. But you haven't had any
12 conversations with Dr. Sfakianos?

13 A. No, I have not.

14 Q. Okay. Thank you.

15 Would you agree that occupational
16 exposures -- exposure levels would be magnitude
17 higher than chemical levels present in Camp
18 Lejeune?

19 A. No I would probably tend to disagree
20 with is that statement. Because occupational
21 exposures are usually only one route of exposure
22 they're limited to discrete amount of time while
23 a person at work. Versus at Camp Lejeune these
24 patients had exposure both through consumption,
25 dermal when they took a shower inhalation when

↑

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UNCERTIFIED ROUGH DRAFT

1 they cooked and it was also an unremitting

2 exposure because they didn't get to go home from

Exhibit 16

Robert Fiolek v. United States of America

U.S. District Court for Eastern District of NC, Southern Division
Case No. 4:23-CV-00062

Specific Causation Expert Report of Damian A. Laber, M.D., F.A.C.P.,

Prepared by

Damian A. Laber, MD, FACP
Senior Member, Dept Ambulatory Hematology and Oncology
Section Chief of Moffitt at TGH, Moffitt Cancer Center
Professor of Medicine and Oncologic Sciences, University of South Florida

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VI. VOLATILE ORGANIC COMPOUNDS (VOCs) AND CAMP LEJEUNE EXPOSURE

Volatile organic compounds (VOCs) are compounds that have a high vapor pressure and low water solubility. Many VOCs are human-made chemicals used and produced in the manufacture of paints, pharmaceuticals, and refrigerants. VOCs are typically industrial solvents, such as trichloroethylene (TCE); fuel oxygenates, such as methyl tert-butyl ether (MTBE); or by-products produced by chlorination in water treatment, such as chloroform. VOCs are also components of petroleum fuels, hydraulic fluids, paint thinners, and dry-cleaning agents, making them common groundwater contaminants.

Some volatile organic compounds (VOCs) known to cause leukemia and lymphoma include:

- **Benzene**
- **Tetrachloroethylene (PCE)**
- **Trichloroethylene (TCE)**

Certain VOCs, including benzene, can pose a lifetime cancer risk via different exposure routes (i.e., inhalation, oral, and dermal) and have been classified as known and probable human carcinogens by the United States Environmental Protection Agency (U.S. EPA), respectively. Based on compelling human and animal evidence, the U.S. EPA has estimated that exposure to 1 $\mu\text{g}/\text{m}^3$ of benzene and in the air over a lifetime would respectively cause 2.2–7.8 cases of leukemia and 13 cases of lung and nasopharyngeal cancers per million people.²² Both benzene and TCE are known to be both genotoxic and immunotoxic, which makes the combination effect of these two chemicals in the water at Camp Lejeune at least additive, if not synergistic.^{23,24}

U.S. Marine Corps Base Camp Lejeune in North Carolina was established in 1942. Specific volatile organic compounds (VOCs) were later discovered in the base's drinking water systems. The Agency for Toxic Substances and Disease Registry (ATSDR) has conducted several epidemiological studies to determine if Marines, Navy personnel, and civilians residing and working at Camp Lejeune were at increased risk for certain health effects due to exposure to water contaminated with VOCs.

I have independently reviewed the ATSDR epidemiological studies, as well as the ATSDR's Assessment of the Evidence.^{25–31} I also have read materials relating to general causation for benzene, TCE, and PCE, including the general causation reports of Drs. Felsher, Hu, Gilbert, Bird, and Mallon, which I rely on based on their experiences in the field of epidemiology. I conclude that it is more likely than not that there is a causal relationship between TCE, PCE, and benzene exposure at Camp Lejeune and CLL.

I have reviewed the New Jersey study conducted by Cohn et al. (1994)³², which reports that in areas with total volatile organic compound (VOC) concentrations above 20 ppb, there was an observed increased risk of Non-Hodgkin lymphoma (NHL) among women. The study further identified that exposure to trichloroethylene (TCE) at levels of 5 ppb was associated with an increased risk of NHL in both women and men. Additionally, exposure to tetrachloroethylene (PCE) at the same concentration level was linked to an increased risk of NHL, particularly high-grade NHL among women.

Exhibit 17

Joseph Gleesing v. United States of America
U.S. District Court for Eastern District of NC, Southern Division
Case No. 7:23-cv-1486

**Specific Causation Expert Report of
Damian A. Laber, M.D., F.A.C.P.**

Prepared by

Damian A. Laber, MD, FACP
Senior Member, Dept Ambulatory Hematology and Oncology
Section Chief of Moffitt at TGH, Moffitt Cancer Center
Professor of Medicine and Oncologic Sciences, University of South Florida

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- **Benzene**
- **Tetrachloroethylene (PCE)**
- **Trichloroethylene (TCE)**

Certain VOCs, including benzene, can pose a lifetime cancer risk via different exposure routes (i.e., inhalation, oral, and dermal) and have been classified as known and probable human carcinogens by the United States Environmental Protection Agency (U.S. EPA), respectively. Based on compelling human and animal evidence, the U.S. EPA has estimated that exposure to 1 µg/m³ of benzene and formaldehyde in the air over a lifetime would respectively cause 2.2–7.8 cases of leukemia and 13 cases of lung and nasopharyngeal cancers per million people.^{22–28} Both benzene and TCE are known to be both genotoxic and immunotoxic, which makes the combination effect of these two chemicals in the water at Camp Lejeune at least additive, if not synergistic.^{29,30}

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I have reviewed the New Jersey study conducted by Cohn et al. (1994)³¹, which reports that in areas with total volatile organic compound (VOC) concentrations above 20 ppb, there was an observed increased risk of non-Hodgkin lymphoma (NHL) among women. The study further identified that exposure to trichloroethylene (TCE) at levels of 5 ppb was associated with an increased risk of NHL in both women and men. Additionally, exposure to tetrachloroethylene (PCE) at the same concentration level was linked to an increased risk of NHL, particularly high-grade NHL among women.

Exhibit 18

Cometto J. Davis v. United States of America
U.S. District Court for Eastern District of NC, Southern Division
Case No. 7:23-cv-00043

**Specific Causation Expert Report of
Richard T. Hoppe, MD, FACR, FASTRO, FARS**

Confidential – Subject to Protective Order

This evaluation: In order to conduct this expert medical evaluation, I reviewed and relied upon the following documents and reports:

- Medical records for Cometto Davis from:

[REDACTED]

- Transcript from the April 29, 2024, deposition of [REDACTED], MD
- Transcript from the February 15, 2024, deposition of the Plaintiff, Cometto Davis
- Discovery Pool Profile Form from Cometto Davis
- Short Form Complaint filed on behalf of Cometto Davis on 11/6/23
- The Expert Report of Morris Maslia dated 10/25/2024
- The Expert Reports on General Causation of Drs Felsher, Hu, Gilbert, and Bird
- The Expert Report of Dr. Kelly Reynold

In addition, I relied upon the peer-reviewed scientific literature that, in my opinion, is the most rigorous and relevant to the issues inherent in this evaluation. As appropriate, such evidence will be cited during the course of this report.

Summary of Opinion:

It is my opinion that it is more likely than not that Mr. Davis's non-Hodgkin lymphoma (NHL), marginal zone lymphoma (MZL), was caused by his exposure to the contaminated water at Camp Lejeune.

I begin with a discussion of the chemicals in the water at Camp Lejeune, briefly turn to their causative effect of NHL generally, and then to the differential etiology of Mr. Davis's NHL.

CHEMICALS AT CAMP LEJUENE

Based on reports and testing, the water at Camp Lejeune contained TCE, PCE, benzene and the by-products of their degradation.

TCE:

Trichloroethylene (TCE) is an industrial solvent that has been widely used in various applications, including degreasing metals and in the production of adhesives and paints. TCE has a biologic half-life of three days. In humans, it is metabolized to trichloroepoxyethane (TCE oxide), then to trichloroacetaldehyde, chloral hydrate and other metabolites including trichloroacetic acid, dichlorovinyl glutathione, and dichlorovinyl cysteine. Some of these metabolites may be more toxic than the parent compound.

Epidemiological studies suggest a link between TCE exposure and the development of non-Hodgkin lymphoma (NHL). Lymphoma is a cancer that affects the lymphatic system, a crucial part of the immune system. There are several mechanisms through which TCE may contribute to the development of lymphoma.

TCE is a genotoxic agent (a property of chemical agents that damages the genetic information [DNA] in a cell) [Guha N et al., 2012] via both direct and indirect effects on the DNA. It may cause chromosome aberrations, chromosome breaks, and sister chromatid exchanges [Varshney M et al., 2014] [Kligerman AD et al 1994]. This genetic damage can lead to mutations that contribute to the development of lymphoma.

TCE can cause immune system dysfunction. [Lash LH 2025] It has been shown to have immunotoxic effects, potentially altering immune function and leading to an increased risk of lymphoproliferative disorders, including lymphoma. Evidence from animal studies indicates that TCE exposure causes immunomodulation including autoimmune disease and immunosuppression [National Research Council of the National Academies, 2006]. Both autoimmune disease and immunosuppression are associated with NHL [Armitage JD et al., 2017]. Studies conducted of Chinese factory workers exposed to TCE have observed alterations in immune function markers that have been associated with an increased risk of NHL, indicating that the associations observed between TCE and NHL are biologically plausible [Bassig B et al. 2013]. In another study of the cohort of Chinese factory workers, total lymphocyte counts decreased with increasing exposures to TCE. Similar exposure-response trends were observed for CD4+ T cells, CD8+ T cells, B cells and NK cells [Bassig B et al. 2016]. The study concluded that these results provided evidence that TCE exposure can lead to immunosuppression, which is associated with an increased risk of NHL.

Karami et al. conducted a meta-analysis of TCE exposure and risk of lymphatic and hematopoietic cancers [Karami S et al 2013]. They examined studies published between 1950 and 2011. The meta-analysis for NHL included 293 NHL cases from 12 cohort studies and 8140 cases from 12 case-control studies. Their conclusion was that the data supported an association between TCE exposure and increased risk of NHL (relative risk = 1.32, 95% confidence interval 1.29-1.79). Scott and Jinot conducted another systematic review of the epidemiologic evidence for an association between TCE exposure and NHL [Scott CS, Jinot J 2011]. They calculated a relative risk for developing NHL following TCE exposure to be 1.23 (95% CI 1.07-1.42) and for the highest exposure group to be 1.43 (95% CI 1.13-1.82).

Benzene:

Benzene is a colorless, toxic chemical compound that is widely recognized as an environmental and occupational hazard. It is primarily used in the manufacture of chemicals, plastics, and synthetic fibers. Benzene exposure has been implicated as a causative agent in the development of NHL.

Research indicates that benzene is a hematotoxic agent [Lan Q et al., 2004], meaning it can adversely affect the blood-forming organs, including the bone marrow. This toxic effect can

lead to disruptions in the production of blood cells, including lymphocytes, which are crucial components of the immune system. Epidemiological studies have consistently demonstrated an association between benzene exposure and an increased risk of developing various hematological malignancies, including NHL.

There are several possible mechanisms by which benzene contributes to lymphomagenesis. Benzene metabolites can induce genetic mutations, compromise immune function, and promote inflammation, all of which may lead to malignant transformation of lymphocytes. Multiple studies show that it produces genotoxicity in the lymphocytes of exposed humans [IARC 2012]. It may produce multiple cytogenetic abnormalities in lymphocytes, and it induces specific chromosomal changes associated with NHL in human lymphocytes. The immunosuppression induced by benzene may lead to decreased immunosurveillance. In a recent study of the cohort of Chinese factory workers, benzene exposure was associated with alterations in lymphoid cell types and B-cell activation markers indicative of immunosuppression that could result in an increased risk of NHL [Bassig B et al. 2016]. Chronic exposure to benzene may result in genetic and epigenetic alterations that enhance lymphocyte proliferation and survival, further contributing to the development of lymphoma.

Benzene has also produced lymphomas in animal studies [IARC 2012]. Accordingly, there is considerable support for the notion that it may cause human lymphatic tumors [IARC 2012]. Linet et al. conducted a large study of mortality among benzene-exposed workers in China [Linet MS et al., 2015]. They compared causes of mortality in 73,789 benzene-exposed workers with 34,504 non-exposed workers in 12 cities in China. The benzene-exposed workers experienced increased risk for all-cause mortality. Notably, the relative risk for NHL was 3.9 (95% CI 1.5-13). In a large meta-analysis of human studies, Rana et al., reviewed 20 case-control and eight cohort studies that included 9587 patients with NHL [Rana I et al., 2021]. They reported increases in the risk for a wide variety of lymphomas and specifically a doubling of the risk for diffuse large B-cell lymphoma.

PCE:

Tetrachloroethylene (PCE) is a colorless, non-flammable liquid used for dry cleaning and as a metal degreasing solvent. It is regarded as a toxic substance, a human health hazard, and an environmental hazard. Numerous toxicology agencies regard it as a carcinogen [US EPA 2020].

A study conducted in four Nordic countries found that high exposure to PCE was associated with an elevated hazard ratio for NHL of 1.23 (95% CI 1.00-1.52) [Vlaanderen J. et al., 2013]. Furthermore, in a long-term mortality study of aircraft manufacturing workers, Boice et al. found an increased standardized mortality rate of 1.70 (95% CI 0.73-3.34) for workers exposed to PCE. [Boice JD. et al., 1999]. In a long term follow up of the same study cohort, Lipworth et al. defined a standardized mortality ratio of 1.43 (1.00-1.98) related to PCE exposure and the risk for developing non-Hodgkin lymphoma [Lipworth et al., 2011]. Thus, the scientific literature supports an association between occupational PCE exposure and NHL.

ATSDR:

In light of the test results of the water at Camp Lejeune, the Government conducted a number of studies of the water. The leading study is what is known as the ATSDR report (ATSDR).

The ATSDR Report, or more fully, the “ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases” published January 13, 2017, reviewed epidemiological studies involving TCE and PCE exposure conducted by the EPA [US Environmental Protection Agency. 2011.], [IARC 2014] and NTP [NTP Monograph on Trichloroethylene. 2015]; meta-analyses conducted by NCI researchers [Karami S et al 2013], EPA [EPA 2011, summarized in Scott CS, Jinot J 2011] and an IARC workgroup [Vlaanderen J et al. 2013] for TCE and hematopoietic cancers. ATSDR utilized these reviews and meta-analyses to identify epidemiological studies for TCE and PCE. Meta-analyses of benzene and hematopoietic cancers [Khalade A et al. 2010; Vlaanderen J et al. 2011; Vlaanderen J et al., 2012] were used to identify epidemiological studies for benzene. In addition, literature searches using PubMed were conducted to identify epidemiological studies conducted after the meta-analyses and reviews were completed.

The ATSDR classified the evidence between exposure to the chemical agent and the development of cancer as “sufficient evidence for causation,” “equipoise and above evidence for causation,” “below equipoise evidence for causation,” and “evidence against a causal relationship.” “Sufficient evidence” was further defined as sufficient evidence from human studies in which chance and biases (including confounding) can be ruled out with reasonable confidence, **or** there is less than sufficient evidence from human studies but sufficient evidence in animal studies and strong evidence that the agent acts through a relevant mechanism in humans. Sufficient evidence from human studies can be provided by a meta-analysis and/or by several studies considered to have high utility. Considerations with respect to the quality of the evidence included temporal relationship, consistent positive associations (e.g., risk ratio or odds ratio greater than 1.1), magnitude of the effect estimate, exposure-response relationship, and biological plausibility.

“Equipoise and above” evidence implied that the evidence was sufficient to conclude that a causal relationship was at least as likely as not, but not sufficient to conclude that a causal relationship existed. For example, if the degree of evidence from human studies was less than sufficient but there was supplementary evidence from animal studies and/or mechanistic studies that supported causality, **or** a meta-analysis did not provide convincing evidence (e.g., the summary risk estimate was close to the null value of 1.0, i.e., ≤ 1.1), or if the meta-analysis observed a non-monotonic exposure-response relationship) but there was at least one epidemiological study considered to be of high utility occurring after the meta-analysis had been conducted, in which an association between the exposure and increased risk of the disease of interest had been found and in which chance and biases could be ruled out with reasonable confidence, **or** a meta-analysis has not been conducted, but there was at least one epidemiological study considered to be of high utility in which an association between the exposure and increased risk of the disease of interest had been found and in which chance and biases could be ruled out with reasonable confidence.

The 2017 ATSDR report concluded, based upon its review, that there was sufficient evidence for causation between TCE exposure and the development of NHL, equipoise and above evidence for causation between PCE and the development of NHL and sufficient evidence for causation between benzene exposure and the development of NHL.

In 2024, the ATSDR published a report that evaluated cancer incidence at Camp Lejeune [Bove FJ. 2024]. In this report, they calculated hazard ratios comparing cancer incidence between Camp Lejeune (where the drinking water was contaminated) and Camp Pendleton (where the drinking water was not contaminated) cohorts. They found that the hazard ratio for developing marginal zone B-cell lymphoma for the comparison of Camp Lejeune vs. Camp Pendleton was 1.45 (95% confidence interval 0.92, 2.28).

The data from the ATSDR Reports combined with the meta-analyses related to TCE exposure (Karami et al., 2013; Scott and Jinot 2011) provide compelling evidence that TCE exposure increases the risk for developing NHL. The ATSDR reports combined with the cohort study of Linet et al (2015) provide a similar degree of evidence for the relationship between benzene exposure and NHL.

Based upon my years of experience, I agree with the ATSDR's definition of "at least as likely as not" or "equipoise and above". Based on available evidence, I also agree that the TCE, PCE, and benzene all cause NHL, at or exceeding the "at least as likely as not" standard.

General Causation Reports of Dr. Felsher, Hu, Gilbert, and Bird

I have reviewed and considered the general causation reports of Drs. Felsher, Hu, Gilbert, and Bird. Based on my background, education, and experience, the reports of these experts are robust and reliable. All have concluded, as do I, that the contaminants in the Camp Lejeune water supply were sufficient to cause NHL. See, for instance, Felsher Report, p. 37-40; Gilbert Report, p.31-35.

Concentrations of Contaminants at Camp Lejeune

I have reviewed the expert report of Morris Masila, dated October 24, 2024. Based on this report, Appendix H1 in particular, it appears that the concentrations of PCE were well in excess of 100 micrograms per liter of water peaking at 182 micrograms per liter in June 1984.

I also have reviewed the expert report of Dr. Kelly Reynold regarding the likely cumulative amounts of TCE, PCE and benzene that Mr. Davis ingested during his time at Hadnot Point and Tarawa Terrace at Camp Lejeune. Considering his days at these locations and cumulative contaminant exposure concentrations, and based upon his deposition-based informed activities, his cumulative consumption (total $\mu\text{g} = \text{days} \times \text{concentration per deposition exposure assumptions}$) for TCE was 2,036,600 μg , for PCE was 503,816 μg , and for benzene was 33,244 μg .

Exhibit 20

1 ROUGH DRAFT DISCLAIMER

2 KATHLEEN A. MALTBIE
3 CERTIFIED SHORTHAND REPORTER, CSR No. 10068
4 RPR, CRR, CCRR, CLR, RMR

4 DEPOSITION OF: Richard T. Hoppe, M.D.

5 DATE: June 9, 2025

6

7 It is understood by all attorneys and/or their
8 staff using, saving onto a hard computer disk, or
9 receiving a realtime ASCII or e-mailed rough draft
10 transcript that:

10 1. The following is an unedited rough draft
11 transcript. Various corrections and/or changes may
12 be made before the final version is complete. The
13 use of this rough draft transcript is limited by
14 C.C.P. 2025.540(b). This reporter, as well as any
15 affiliated court reporting agency, will not be
16 responsible for any variance of this draft from the
17 final transcript.

18 2. Because of the nature of stenographic
19 outlines, differences WILL exist between the
20 realtime rough draft copy and the certified
21 transcript prepared by the reporter. Those
22 differences will include the following, among
23 others:

- 18 a. Words may change;
- 19 b. Page and line numbers may change;
- 20 c. Punctuation may change; and/or
- 21 d. Quotes may change.

22 3. Providing a realtime ASCII and/or
23 e-mail or saving realtime onto a computer hard drive
will only be provided when a certified copy is
purchased and there will be a charge for the
realtime rough transcript in addition to the charge
for the certified copy.

9 A. That's correct.

10 Q. And you are not an expert in environmental
11 risk assessments; fair?

12 A. That's fair.

13 Q. Have you ever conducted any human health
14 environmental risk assessments?

15 A. No, I haven't.

16 Q. Did you not conduct a Bradford Hill
17 analysis in this case, right?

18 A. That's correct.

19 Q. Are you familiar with what Bradford Hill
20 is?

21 A. Somewhat, from reading the papers and
22 documents that are cited here.

23 Q. Prior to this case were you familiar with
24 what a Bradford Hill analysis was?

25 A. No, I was not.

↑

UNCERTIFIED ROUGH DRAFT

32
1 Q. Your general causation analysis, which if
2 you turn to Exhibit 1 and Exhibit 3, which are your
3 two reports, that's found in the sections chemicals

4 at Camp Lejeune. It's on page 2 of Exhibit 1.

5 A. Okay.

6 Q. And general causation reports of
7 Dr. Felsher, Hu, Gilbert and Bird, which is the
8 section following it. Is that fair or --

9 A. I'm not sure I understand your question.

10 Q. Sure.

11 Well, let me step back. You offer the
12 same general causation analysis in your reports for
13 Mr. Howard and Mr. Davis, correct?

14 A. That's correct, although I always refer to
15 them as specific causation reports.

16 Q. Sure.

17 Do you understand the difference between a
18 general causation analysis and a specific causation
19 analysis?

20 A. Well, I'm not sure I understand
21 completely.

22 Q. What is your understanding between the
23 difference between a general causation and specific
24 causation analysis?

25 A. Well, the specific causation analysis

23 on -- in your sections that are titled "chemicals at
24 Camp Lejeune"?
25 A. Okay.

↑

UNCERTIFIED ROUGH DRAFT

34

1 Q. TCE, Benzene, PCE, ATSDR?
2 A. Right.
3 Q. And then general causation reports of
4 Dr. Felsher, Hugh, Gilbert and Bird?
5 A. Right.
6 Q. Are there any other sections in your
7 report that go to general causation?
8 A. Well, unless you, you know, refer to my
9 conclusions and the differential diagnosis
10 methodology, but for the most part, I would say that
11 the general causation is in those sections that you
12 cited.
13 Q. Sure.
14 And if you turn to Exhibit 3, which is
15 Mr. Howard's report, the general causation opinions
16 you offer are in the sections 4, 5 and 6.
17 A. Correct.

18 Q. Were you -- or are you aware that the
19 expert reports in this case are phased such that
20 general causation was considered phase 2 and
21 specific causation is considered phase 3?

22 A. I'm not familiar with that terminology.

23 Q. And you reviewed the reports of doctors
24 Felsher, Hugh, Gilbert and Bird; is that fair?

25 A. Yes. I -- I recall reviewing --

↑

UNCERTIFIED ROUGH DRAFT

35

1 Q. Page 6?

2 A. -- Dr. Felsher and Dr. Hugh, and the third
3 one, Bird, yes.

4 Q. I think I asked you this already, but you
5 don't recall whether you've reviewed the report of
6 Dr. Goodman?

7 A. No. I -- I don't recall reviewing the
8 report of Dr. Goodman.

9 Q. Have you reviewed the report of
10 Dr. Lipscomb?

11 A. That name is not familiar.

12 Q. Have you reviewed the report of
13 Dr. McCabe?

14 A. I don't believe so.

15 Q. Have you reviewed the report of
16 Dr. Shields?

17 A. Not to my knowledge.

18 Q. If it would be helpful, you're welcome to
19 look at your material considered list that were
20 marked as Exhibits 2 and 4?

21 A. Oh, okay.

22 Q. And I believe you looked at
23 Dr. Goodman's -- or you've considered Dr. Goodman's
24 transcript.

25 To the best of your memory, have you

↑

UNCERTIFIED ROUGH DRAFT

36
1 reviewed any other of the United States experts'
2 deposition transcripts?

3 A. To the best of my knowledge, no.

4 Q. Turning to your report, do you do your own
5 general causation analysis or did you reply -- rely
6 on the reports of Dr. Felsher, Hugh, Gilbert and

7 Bird for their determinations?

8 A. Certainly a lot of it was my own. I may
9 have reviewed Dr. Felsher and -- and Hugh's general
10 causation, but I think I put together most of this
11 based on my own review of the materials.

12 Q. If you turn to page 6 of Exhibit 1.

13 The section general causation reports of
14 Dr. Felsher, Hugh, Gilbert and Bird.

15 Do you see that section?

16 A. Yes.

17 Q. You state (as read):

18 I have reviewed and considered
19 the general causation reports of
20 Dr. Felsher, Hugh, Gilbert and
21 Bird, based on my background,
22 education, and experience, the
23 reports of these experts are robust
24 and reliable.

25 Did I read that correctly?

↑

UNCERTIFIED ROUGH DRAFT

1 A. Yes, you did.

2 Q. What did you do to conclude the reports
3 were robust and reliable?

4 A. Well, I -- I read their -- their
5 scientific analysis of the data, which in my mind
6 seemed rigorous. And their conclusions were
7 reasonable.

8 Q. Did you review all of the studies that
9 they reference in their reports?

10 A. No, I did not review all of the studies
11 that were referenced.

12 Q. The studies that did you review for
13 general causation are -- those are the studies that
14 you cite to in your report, correct?

15 A. Yes.

16 Q. When you were developing your general
17 causation opinions, what steps did you take to do
18 so?

19 A. Well, I read a number of references,
20 highlighted certain points, took some notes and then
21 I synthesized that together to make this report.

22 Q. And to gather your references, did you run
23 searches, or what steps did you take to gather your
24 references?

25 A. I did some searches. Also initially,

1 there was some references, citations provided to me
2 by the attorneys.

3 Q. Do you recall what searches you ran?

4 A. They were on PubMed, PubMed searches for
5 the PCA -- PCE, TCE and Benzene related to
6 Non-Hodgkin lymphoma.

7 Q. Do you recall any of the specific search
8 terms you used?

9 A. PCE, TCE, Benzene, Non-Hodgkin lymphoma.
10 Probably also searched spelling out those chemical
11 names.

12 Q. If you turn to -- or maybe you're still at
13 page 6 of your report for Mr. Davis?

14 A. Yes.

15 Q. You state (as read):

16 Based on all available
17 evidence, I also agree that TCE,
18 PCE and Benzene all cause NHL at or
19 exceeding the at least as likely as
20 not standard.

21 Did I read that correctly?
22 A. I'm not sure. Where on that page is it.
23 Q. Oh, sorry. So it's the middle of the
24 page. Just above your general causation reports of
25 the other experts?

↑

UNCERTIFIED ROUGH DRAFT

39

1 A. Okay.
2 Q. I can read it again. It's the last
3 sentence. It says (as read):
4 Based on available evidence, I
5 also agree that the TCE, PCE and
6 Benzene all cause NHL at or
7 exceeding the as likely as not
8 standard.
9 A. Right.
10 Q. Did I read that correctly?
11 A. Yes.
12 Q. How did you decide to use the at least as
13 likely as not standard?
14 A. I believe that it was the attorneys that

Exhibit 21

Jose Vidana v. United States of America

U.S. District Court for Eastern District of NC, Southern Division
Case No. 7:23-CV-01575

**Specific Causation Expert Report of
Paul J. Michaels, M.D.**

Confidential – Subject to Protective Order

I. BACKGROUND AND QUALIFICATIONS

I am certified by the American Board of Pathology in Anatomic Pathology, Clinical Pathology, and Cytopathology. I attended and received my medical degree from the University of California, Los Angeles (UCLA) School of Medicine where I was elected to the Alpha Omega Alpha Honor Society and completed a year-long post-sophomore fellowship in pathology through a combined UCLA-Cedars Sinai program. I then completed a residency in anatomic and clinical pathology at Massachusetts General Hospital, an affiliate of the Harvard School of Medicine, where I was a Clinical Fellow in Pathology. Following my residency, I completed a year of subspecialization in Cytopathology, also at Massachusetts General Hospital. As a board-certified pathologist and cytopathologist, my day-to-day responsibilities include, amongst many other tasks in the anatomic and clinical laboratory, the microscopic examination of various tissues to evaluate for the presence of cancer development.

I am a pathologist affiliated with Pathology Consultants, one of the first medical groups formed in Oregon, and am one of two pathologists based at Bay Area Hospital in Coos Bay, Oregon, the largest medical center on the Oregon coast, where I serve as the Chair of Pathology and am a member of the hospital Medical Executive Committee and the Cancer Committee. I am also affiliated with Southern Coos Hospital in Bandon, Oregon, where I serve as the Laboratory Medical Director. Additionally, I am a Clinical Assistant Professor in the Department of Pathology & Laboratory Medicine at Oregon Health & Science University (OHSU), am actively involved in teaching for the pathology residency program, and was the recipient of the “Most Innovative Teaching Award” in 2023 at the OHSU Pathology Residency graduation. Prior to joining Pathology Consultants, I was a member of two large groups in Las Vegas, Nevada and Austin, Texas, serving on the faculty of Touro University School of Osteopathic Medicine and Dell Medical School at the University of Texas in Austin, respectively. In addition, while affiliated with Dell Medical School at the University of Texas in Austin, I served as the Division Chief of Head and Neck Pathology. During my career, I have had a strong subspecialty focus in breast and gynecologic pathology, as well as cytopathology, but have routinely been considered by my colleagues to be an expert in the diagnosis of disease processes and cancer throughout all organ systems, often serving as one of the main internal consultants for challenging tumors, including hematopoietic neoplasms. Throughout my entire career, I have regularly attended and participated in tumor multidisciplinary conferences both in rural and community settings, as well as within large urban centers and sprawling academic facilities. My current curriculum vitae is attached to this report.

I have been asked to review the medical records, including the pathology report, of Mr. Jose Vidana. I have also reviewed other materials, including numerous applicable scientific studies, deposition testimony (including that of Mr. Vidana and Drs. Ann Mohrbacher and Radhika Tulpule), and other materials in arriving at my findings and opinions in this case, a list of which is attached to my report. All of my opinions stated below are held to a reasonable degree of medical and scientific certainty, and I reserve the right to modify or change my opinions based on further documents or information that may be provided to me in the future.

II. SUMMARY OF OPINIONS

1. [REDACTED]
2. *Diffuse Large B-cell Lymphoma is a subtype of non-Hodgkin lymphoma.*
3. *Chemicals found in the water in camp Lejeune, including trichloroethylene (TCE), tetrachloroethylene (PCE), and benzene, are carcinogens and have been found to increase the risk for development of various non-Hodgkin lymphomas in both animals and humans, including Diffuse Large B-cell Lymphoma.*

Contaminants at Camp Lejeune and Specific Cancers and Other Diseases,” released on January 13, 2017.¹⁰ I have also been provided with the expert report and Appendixes of Morris L Maslia, PE, and additionally incorporated the values in that report in forming my opinion. The results reported from their models showed that the water at Camp Lejeune was contaminated with varying levels of trichloroethylene (TCE), tetrachloroethylene (PCE), benzene, and vinyl chloride (VC) as measured in mean monthly concentrations. During the period of time when Mr. Vidana was at Camp Lejeune, the concentrations in finished water ranged from 449-546 µg/L for TCE, 22-27 µg/L for PCE, 7-8 µg/L for benzene, and 36-45 µg/L for VC. These levels of contamination have been shown to cause disease, including NHL.

VII. CAMP LEJEUNE CARCINOGENS AND NON-HODGKIN LYMPHOMA:

A. CARCINOGENESIS

A carcinogen is defined as any substance capable of causing cancer in living tissue. Carcinogens can be broadly categorized into two classes, genotoxic and non-genotoxic. Genotoxic carcinogens are chemicals or substances that are able to exert carcinogenicity via the induction of mutations. Because of this ability to directly interact with the underlying DNA framework and sequence, genotoxic agents are considered to have no safe exposure threshold or dose.^{11,12} In contrast, non-genotoxic carcinogens induce tumor growth through mechanisms other than those associated with direct DNA mutations, such as through inflammation, angiogenesis, cell proliferation, cytotoxicity, or hormonal effects.^{13,14} Thus, these agents are thought to have a exposure threshold at which an acceptable daily intake (ADI) or tolerable daily intake (TDI) is determined by authorities based on no observed adverse effect level (NOAEL).¹⁵ However, some of these carcinogens that typically exert their effects via non-genotoxic pathways, such as in the case of inflammation-induced cell proliferation, can ultimately potentiate DNA damage through direct genetic mutations.^{16,17} It is these non-genotoxic carcinogens that Paracelsus, the 15th Century Swiss scientist, alchemist, and physician, known as the “Father of Toxicology” was referring to when he stated that “the dose makes the poison,” as it is thought that a non-genotoxic carcinogen, or “poison,” can be

¹⁰ Agency for Toxic Substances and Disease Registry (ATSDR). *Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases*. U.S. Dept. of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry; 2017.

¹¹ Nohmi T. Thresholds of genotoxic and non-genotoxic carcinogens. *Toxicol Res* 2018;34:281-90.

¹² Nohmi T and Matsumoto K. Effects of DNA polymerase kappa and mismatch repair on does-responses of chromosome aberrations induced by three oxidative genotoxins in human cells. *Environ Mol Mutagen* 2020;61:193-9.

¹³ Panigrahy D, Gartung A, Yang Y, et al. Preoperative stimulation of resolution and inflammation blockade eradicates micrometastases. *J Clin Invest* 2019;129:2964-79.

¹⁴ Panigrahy D, Singer S, Shen LQ, et al. PPAR-gamma ligands inhibit primary tumor growth and metastasis by inhibiting angiogenesis. *J Clin Invest* 2002;110:923-32.

¹⁵ Fitzgerald DJ and Robinson NI. Development of a tolerable daily intake for N-nitrosodimethylamine using a modified benchmark dose methodology. *J Toxicol Environ Health* 2007;70:1670-8.

¹⁶ Kiraly O, Gong G, Olipitz W, et al. Inflammation-induced cell proliferation potentiates DNA damage-induced mutations in vivo. *PLoS Genet* 2015;11:e1004901.

¹⁷ Tomatis L, Huff J, Hertz-Picciotto I, et al. Avoided and avoidable risks of cancer. *Carcinogenesis* 1997;18:97-105.

non-toxic if the dose is below a certain threshold. This concept was further detailed within a fundamental principle in toxicology known as Haber's law or rule, a formula originally developed by the German physical chemist Fritz Haber, who elucidated that a concentration (C) of an agent multiplied by the duration or time (T) of exposure, would yield a specific biological response.^{18,19} As cancer risk estimates are generally based on the average lifetime daily dose which is derived from the total cumulative exposure, this concept has been shown to be both theoretically and empirically valid for studies on non-genotoxic induced carcinogenesis.²⁰

The chemicals to which Mr. Vidana was exposed are genotoxic. Carcinogenesis is a complex and multistep process in which previously normal cells undergo molecular and cellular changes that ultimately result in the development of a malignancy.²¹ The previously described carcinogens found as contaminants within the water supply in Camp Lejeune are generally considered to represent genotoxic chemicals capable of initiating malignant transformation. Exposure to these types of mutagens, even when only for a short period of time and at low levels, can still lead to irreversible cellular damage that can result in a malignant tumor.²² This is consistent with the establishment of a minimum duration at Camp Lejeune of 30 days in order to be eligible for the health benefits under the Camp Lejeune Act. This is specifically addressed in the ATSDR 2017 *Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases* where it is noted that "the results from the Camp Lejeune mortality studies suggest that a 30-day minimum duration requirement may be appropriate since elevated risks for some of the diseases evaluated were observed for exposure durations of 1-3 months." It went on to report that those "results should not be surprising given that the levels of TCE, PCE, and vinyl chloride measured or estimated in the drinking water systems at Camp Lejeune considerably exceeded their respective MCLs (maximum contaminant levels)." The current U.S. MCLs for TCE, PCE, and benzene are 5 ppb, but only 2 ppb for vinyl chloride. The concentrations present in the water during Mr. Vidana's time there were well in excess of the MCLs.

B. TRICHLOROETHYLENE (TCE)

The International Agency for Research on Cancer (IARC) has classified trichloroethylene (TCE) as *carcinogenic to humans*, a Group 1 carcinogen.²³ It was noted in their monograph that the Working Group was unanimous in its conclusion that TCE is a Group 1 carcinogen, stating that supporting evidence included the absorption, distribution, metabolism, and excretion of TCE is well characterized in both animals and humans, and that the oxidative metabolism of TCE is catalyzed by cytochrome P450 enzymes, causing the formation of reactive metabolites in the kidney which are genotoxic on the basis of

¹⁸ Witschi J. Some notes on the history of Haber's law. *Toxicol Sci* 1999;50:164-8.

¹⁹ Miller FJ, Schlosser PM, and Janszen DB. Haber's rule: A special case in a family of curves relating concentration and duration of exposure to a fixed level of response for a given endpoint. *Toxicology* 2000;149:21-34.

²⁰ Gaylor DW. The use of Haber's law in standard setting and risk assessment. *Toxicology* 2000;149:17-9.

²¹ Boyland E. Tumour initiators, promoters, and complete carcinogens. *Br J Ind Med*. 1985;42:716-8.

²² Stewart BW. Mechanisms of carcinogenesis: from initiation and promotion to the hallmarks. In: Baan RA, Stewart BW, Straif K, editors. *Tumour Site Concordance and Mechanisms of Carcinogenesis*. Lyon (FR): International Agency for Research on Cancer; 2019.

²³ IARC. Trichloroethylene, tetrachloroethylene, and some other chlorinated agents. *IARC Monog Eval Carcinog Risks Hum*. 2014;106:1-512.

consistent results in several available test systems. The authors noted that information specifically as it related to non-Hodgkin lymphoma was available from 8 independent cohort studies and 8 case-control studies which, although complicated by varying systems used to classify lymphomas, showed that there were modestly elevated relative risks seen with many of the cohort studies and that the case-control studies also showed a modest positive association of non-Hodgkin lymphoma with exposure to TCE. They reported that a meta-analysis performed of cohort and case-control studies found a statistically significant RR of 1.2 (95% CI, 1.1-1.4) for non-Hodgkin lymphoma and any exposure to TCE, and a RR of 1.4 (95% CI, 1.1-1.8) for higher exposure, indicative of an exposure-dose response. In addition, the National Toxicology Program, 15th edition, also lists TCE as “known to be a human carcinogen” based on sufficient evidence of carcinogenicity from human studies, including those related to non-Hodgkin lymphoma, though the evidence is noted to be less consistent than for kidney cancer, possibly related to the diverse groups of cancers included in the non-Hodgkin lymphoma category, many of which are known to have varied mutations and etiologies.²⁴

In a meta-analysis and review of 14 occupational cohort and 4 case-control studies of workers exposed to TCE, the summary relative risk estimates (SRRE) for the group of cohort studies that had more detailed information on TCE exposure was 1.29 (95% CI = 1.00-1.66) for the total cohort and 1.59 (95% CI = 1.21-2.08) for the 7 studies that identified a specific TCE exposed sub-cohort,²⁵ though the authors felt the findings were hampered by the variability in results, limited exposure assessments, and other limitations of the various analyses. A separate meta-analysis focusing on studies with high potential for TCE exposure and associations with various cancers, showed a summary relative risk estimate for both overall exposure and for the highest risk group, with respect to non-Hodgkin lymphoma, of 1.23 (95% CI = 1.07-1.42) and 1.43 (95% CI = 1.13-1.82), respectively.²⁶ A study of histologically confirmed non-Hodgkin lymphoma cases in Swedish workers exposed to various solvents, found markedly increased risk with exposure to TCE (RR = 7.2; 95% CI, 1.3-42), in which even relatively short-term exposures under 30 days (1-17 days) were associated with an elevated risk for non-Hodgkin lymphoma (RR = 6.5; 95% CI, 2.1-18).²⁷ However, although a pooled analysis of 4 international case-control studies of NHL that included detailed assessments of exposure to TCE did find evidence of an increased risk of NHL, when evaluating specific subtypes, there did not appear to be an association with DLBCL.²⁸ However, as the authors noted, the study was limited by the small number of subjects that were actually exposed to TCE (9% of study participants) as well as the different study designs used and the lack of a detailed assessment of concurrent exposure to other carcinogens. In addition, a pooled cohort study of workers in Nordic countries with documented exposure to TCE, although noted an increased standardized incidence

²⁴ National Toxicology Program. 15th Report on Carcinogens [Internet]. Research Triangle Park (NC): National Toxicology Program; 2021. *Trichloroethylene*: CAS No. 79-01-6.

²⁵ Mandel JH, Kelsh MA, Mink PJ, et al. Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review. *Occup Environ Med*. 2006;63:597-607.

²⁶ Scott CS, Jinot J. Trichloroethylene and cancer: systematic and quantitative review of epidemiologic evidence for identifying hazards. *Int J Environ Res Public Health*. 2011;8:4238-72.

²⁷ Hardell L, Eriksson M, Degerman A. Exposure to phenoxyacetic acids, chlorophenols, or organic solvents in relation to histopathology, stage, and anatomical localization of non-Hodgkin's lymphoma. *Cancer Res*. 1994;54:2386-9.

²⁸ Cocco P, Vermeulen R, Flore V, et al. Occupational exposure to trichloroethylene and risk of non-Hodgkin lymphoma and its major subtypes: a pooled InterLymph [correction of IinterLymph] analysis. *Occup Environ Med*. 2013;70:795-802.

ratio (SIR) for non-Hodgkin lymphoma (SIR = 1.26; 95% CI = 0.89-1.73), this was not statistically significant.²⁹ Similarly, a mortality study of aircraft maintenance workers at Hill Air Force Base in the United States exposed to TCE showed moderately increased hazard ratios for risk of non-Hodgkin lymphoma at the time of initial follow-up (HR = 2.0), as well as for follow-up 10 years later (HR = 1.36), though neither value was statistically significant.³⁰ In this case, it has been specifically noted in the literature that there is strong evidence for multiple environmental exposures in diffuse large B-cell lymphoma, including with trichloroethylene and benzene.³¹

C. TETRACHLOROETHYLENE (PCE)

The International Agency for Research on Cancer (IARC) has classified tetrachloroethylene/perchloroethylene (PCE) as *probably carcinogenic to humans* (Group 2A), based on sufficient evidence in experimental animals and limited evidence in humans for carcinogenicity.^{IARC 2014} It was noted in the monograph that three separate cohort studies showed an increased risk specifically for non-Hodgkin lymphoma, though the studies were based on small sample sizes, and case-control studies failed to identify significant associations. In 2012 the U.S. Environmental Protection Agency (EPA) performed an extensive review of epidemiological data regarding the carcinogenicity of PCE and concluded that there was evidence associating exposure to several types of cancer in humans, including non-Hodgkin lymphoma.³² In addition, according to the National Toxicology Program, PCE is noted to be “reasonably anticipated to be a human carcinogen.”³³ A relatively recent review published in the literature by authors employed by Gradient, a private environmental consulting firm involved in litigation, purported to conduct a systematic review of the literature regarding the association between PCE and non-Hodgkin lymphoma and concluded that the evidence did not support a link between exposure and malignancy.³⁴

In Sweden, tetrachloroethylene (PCE) has been noted to be the quantitatively most important agent for dry-cleaning during the second half of the 20th century, therefore, in an indirect assessment of PCE exposure, a cancer morbidity study of Swedish dry-cleaners and laundry workers noted a statistically significant standardized cancer incidence ratio (SIR) of 2.05 (95% CI = 1.30-3.07) for non-Hodgkin lymphoma in this cohort, suggesting an association.³⁵ An Italian case-control study evaluating

²⁹ Hansen J, Sallmén M, Seldén AI, et al. Risk of cancer among workers exposed to trichloroethylene: analysis of three Nordic cohort studies. *J Natl Cancer Inst.* 2013;105:869-77.

³⁰ Radican L, Blair A, Stewart P, Wartenberg D. Mortality of aircraft maintenance workers exposed to trichloroethylene and other hydrocarbons and chemicals: extended follow-up. *J Occup Environ Med.* 2008;50:1306-19.

³¹ Wang SS. Epidemiology and etiology of diffuse large B-cell lymphoma. *Semin Hematol.* 2023;60:255-266.

³² U.S. Environmental Protection Agency. Toxicological review of tetrachloroethylene (Perchloroethylene). *In Support of Summary Information on the Integrated Risk Information System (IRIS)*. February 2012.

³³ National Toxicology Program. 15th Report on Carcinogens [Internet]. Research Triangle Park (NC): National Toxicology Program; 2021. *Tetrachloroethylene*: CAS No. 127-18-4.

³⁴ Goodman JE, Ticknor RC, Zhou J. Systematic review of perchloroethylene and non-Hodgkin's lymphoma. *Glob Epidemiol.* 2022;4:100077.

³⁵ Seldén AI, Ahlborg G Jr. Cancer morbidity in Swedish dry-cleaners and laundry workers: historically prospective cohort study. *Int Arch Occup Environ Health.* 2011;84:435-43.

occupational exposure to solvents and the risk of lymphomas (Hodgkin and non-Hodgkin), a nearly 2-fold increased risk of diffuse-type non-Hodgkin lymphoma was observed for exposure to PCE (OR = 1.9), though this was not statistically significant.³⁶ Similarly, a non-significantly elevated lymphoma risk was noted with high exposure to PCE (adjusted OR = 3.4), though the power of the study was limited due to the low number of control subjects that were classified as PCE exposure (4%).³⁷ In addition, a study evaluating occupational exposure to PCE and TCE found that HRs for non-Hodgkin lymphoma were elevated in those subjects with high exposure to PCE, as well as in the setting of continuous exposure.³⁸ An analysis using a series of case-control studies nested in cohorts of laundry and dry-cleaning workers in Nordic countries did not find a statistically significant increased risk of non-Hodgkin lymphoma in the exposed population from this study.³⁹ Similar findings were described in adults working in Sweden during the 1960 and 1970 census with an occupation as a dry cleaner, launderer, or presser serving as a surrogate for PCE exposure.⁴⁰

D. BENZENE

The International Agency for Research on Cancer (IARC) has classified benzene as *carcinogenic to humans*, a Group 1 carcinogen, based on sufficient evidence of carcinogenicity in both experimental animals and humans, specifically with respect to non-Hodgkin lymphoma, among other malignancies.⁴¹ In the most recent IARC Monograph, twenty-one studies on the association between non-Hodgkin lymphoma and exposure to benzene in occupational cohorts were included in their analysis. In addition, the National Toxicology Program, 15th edition, also lists benzene as “known to be a human carcinogen” based on sufficient evidence of carcinogenicity from human studies.⁴² Many human studies published within the scientific literature have consistently found an association between exposure to benzene and non-Hodgkin lymphoma. However, it has been noted that most studies only contain small numbers of non-Hodgkin lymphoma patients and often have mortality as the measured outcome, limiting the sensitivity of the studies, as many non-Hodgkin lymphomas are either indolent or have a relatively high cure rate, including diffuse large B-cell lymphoma (DLBCL).

A large retrospective cohort study of over 70,000 benzene-exposed Chinese workers, published by the Chinese Center for Disease Control and Prevention-U.S. National Cancer Institute Benzene Study

³⁶ Miligi L, Costantini AS, Benvenuti A, et al. Occupational exposure to solvents and the risk of lymphomas. *Epidemiology*. 2006;17:552-61.

³⁷ Seidler A, Möhner M, Berger J, et al. Solvent exposure and malignant lymphoma: a population-based case-control study in Germany. *J Occup Med Toxicol*. 2007;2:2.

³⁸ Vlaanderen J, Straif K, Pukkala E, et al. Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries. *Occup Environ Med*. 2013;70:393-401.

³⁹ Lynge E, Andersen A, Rylander L, et al. Cancer in persons working in dry cleaning in the Nordic countries. *Environ Health Perspect*. 2006;114:213-9.

⁴⁰ Travier N, Gridley G, De Roos AJ, et al. Cancer incidence of dry cleaning, laundry and ironing workers in Sweden. *Scand J Work Environ Health*. 2002;28:341-8.

⁴¹ IARC. Benzene. *IARC Monog Eval Carcinog Risks Hum*. 2018;120:1-301.

⁴² National Toxicology Program. 15th Report on Carcinogens [Internet]. Research Triangle Park (NC): National Toxicology Program; 2021. *Benzene*: CAS No. 71-43-2.

Group, showed a near 4-fold increase in non-Hodgkin lymphoma (RR = 3.9; CI = 1.5-13).⁴³ A separate large Chinese study published by the same group also found that workers with 10 or more years of exposure to benzene had a significantly increased risk of developing non-Hodgkin lymphoma (RR = 4.2; 95% CI = 1.1-15.9).⁴⁴ A case-cohort study of nearly 25,000 Norwegian men working in the offshore oil industry with presumed benzene exposure showed an elevated adjusted risk for the development of diffuse large B-cell lymphoma (DLBCL) when comparing those who began employment prior to 1980, compared to control subjects without known exposure (HR = 1.32; 95% CI = 0.35-4.62).⁴⁵ A study of two cohorts of male Swedish seamen exposed to cargo vapors from gasoline and other petroleum products containing benzene reported a statistically significant increase in non-Hodgkin lymphoma (OR = 3.3; 95% CI = 1.1-10.6) in those workers with at least one month of an exposure history, including a noted significant exposure-response relation when evaluating all lymphatic and hematopoietic malignancies.⁴⁶ Some authors have suggested that biases in many studies could decrease the sensitivity of finding an association between true benzene exposure and resultant non-Hodgkin lymphoma cases. In a meta-analysis of 22 studies examining varying degrees of benzene exposure, the summary relative risk for non-Hodgkin lymphoma was 1.22 (95% CI = 1.02-1.47), though increased to 1.49 (95% CI = 1.12-1.97) when studies that likely included unexposed subjects within the case group, and further increased to 2.12 (95% CI = 1.11-4.02) when studies based solely on self-reported work history were excluded.⁴⁷ Importantly, a very large meta-analysis, including a search that yielded 2,481 articles and nearly 10,000 patients, reported findings that not only suggested a causal link between benzene and non-Hodgkin lymphoma as a whole, but especially for diffuse large B-cell lymphoma (DLBCL), the subtype of lymphoma Mr. Vidana

[REDACTED],⁴⁸

E. CAMP LEJEUNE SITE STUDIES

For decades, epidemiologic studies have pointed to an association between the consumption of contaminated drinking water, particularly containing chlorinated volatile compounds, and an increased

⁴³ Linet MS, Yin SN, Gilbert ES; Chinese Center for Disease Control and Prevention-U.S. National Cancer Institute Benzene Study Group. A retrospective cohort study of cause-specific mortality and incidence of hematopoietic malignancies in Chinese benzene-exposed workers. *Int J Cancer*. 2015;137:2184-97.

⁴⁴ Hayes RB, Yin SN, Dosemeci M, et al. Benzene and the dose-related incidence of hematologic neoplasms in China. Chinese Academy of Preventive Medicine--National Cancer Institute Benzene Study Group. *J Natl Cancer Inst*. 1997;89:1065-71.

⁴⁵ Stenehjem JS, Kjærheim K, Bråtveit M, et al. Benzene exposure and risk of lymphohaematopoietic cancers in 25,000 offshore oil industry workers. *Br J Cancer*. 2015;112:1603-12.

⁴⁶ Nilsson RI, Nordlinder R, Hörte LG, Järholm B. Leukaemia, lymphoma, and multiple myeloma in seamen on tankers. *Occup Environ Med*. 1998;55:517-21.

⁴⁷ Steinmaus C, Smith AH, Jones RM, Smith MT. Meta-analysis of benzene exposure and non-Hodgkin lymphoma: biases could mask an important association. *Occup Environ Med*. 2008;65:371-8.

⁴⁸ 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;5:e633-e643.

incidence of hematopoietic malignancies.^{49,50,51} A large study of drinking water contamination and leukemia and non-Hodgkin lymphoma incidence was conducted in a 75-town area in New Jersey, comparing cancer incidence in towns with the highest amount of known exposure to TCE and PCE, to towns without detectable exposure to those chemicals.⁵² In this analysis, there was a statistically significant increase in those individuals diagnosed with intermediate—grade non-Hodgkin lymphoma/diffuse large B-cell lymphoma, in both men (RR = 1.59; 95% CI = 1.04-2.43) and women (RR = 1.66; 95% CI = 1.07-2.59) exposed to greater than 5 ppb of TCE in their drinking water.

Based on this earlier work, it is not surprising that studies of individuals based at Camp Lejeune, where high levels of TCE, PCE, and benzene were detected in the water for decades, also corroborate the increase in risk of exposed individuals to the combination of these chemicals with malignancy, including non-Hodgkin lymphoma. A study utilizing guidance from the U.S. EPA and Agency for Toxic Substances and Disease Registry (ATSDR) to calculate the cancer risk to Marines who were exposed to the carcinogens in the drinking water at Camp Lejeune from 1953-1987, found that TCE, PCE, benzene, and vinyl chloride contributed to the known cumulative cancer risk that was seen in these individuals.⁵³ They went on to summarize that the “cancer risk values provide substantial evidence that disease such as liver cancer, bladder cancer, kidney cancer, NHL (non-Hodgkin lymphoma), and multiple myeloma are a high risk to individuals who spent time on the base, especially during the years of greatest contamination in the late 1970s and 1980s.” In a morbidity study of former Marines, employees, and dependents potentially exposed to the contaminated drinking water at Camp Lejeune performed by the ATSDR, although statistically significant results were identified for both Marines and civilian employees for bladder cancer, kidney cancer, and kidney disease, the small increase in risk for lymphoma seen in Camp Lejeune Marines compared to Camp Pendleton Marines was not statistically significant (OR = 1.06; 95% CI = 0.75-1.50).⁵⁴ However, limitations of this study were the relatively small percentage of subjects who responded to the questionnaires sent out, in addition to the fact that the authors did not further subclassify the lymphomas at all in this analysis. A mortality study comparing Camp Lejeune and Camp Pendleton cohorts did not reveal an increased risk of non-Hodgkin lymphoma after adjusting for sex, race, rank, and education with a 10-year lag (HR = 0.81; 95% CI = 0.56-1.18).⁵⁵ However, again, the subtype of non-

⁴⁹ Lagakos SW, Wessen BJ, Zelen M. An analysis of contaminated well water and health effects in Woburn, Massachusetts. *J Am Stat Assoc.* 1986;81:583-96.

⁵⁰ Aschengrau A, Ozonoff D, Paulu C, et al. Cancer risk and tetrachloroethylene (PCE) contaminated drinking water in Massachusetts. *Arch Environ Health.* 1993;48:284-292.

⁵¹ Fagliano J, Berry M, Bove F, Burke T. Drinking water contamination and the incidence of leukemia: an ecologic study. *Am J Public Health.* 1990;80:1209-12.

⁵² Cohn P, Klotz J, Bove F, et al. Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma. *Environ Health Perspect.* 1994;102:556-61.

⁵³ Rosenfeld PE, Spaeth KR, McCarthy SJ, et al. Camp Lejeune marine cancer risk assessment for exposure to contaminated drinking water from 1955 to 1987. *Water Air Soil Pollut.* 2024;235:124.

⁵⁴ Agency for Toxic Substances and Disease Registry (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.

⁵⁵ Bove FJ, Ruckart PZ, Maslia M, Larson TC. Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study. *Environ Health.* 2014;13:10.

Hodgkin lymphoma was not separated out in the analysis, which is extremely important as etiologies for different lymphomas are extremely varied. Also, as this was a mortality study and many non-Hodgkin lymphomas are either indolent or, conversely, cured with therapy, the diagnosis of a prior lymphoma may not have been listed on the death certificate. Both of these would have the tendency to underestimate the actual risk associated with consumption of contaminated water at Camp Lejeune and a non-Hodgkin lymphoma such as diffuse large B-cell lymphoma (DLBCL). In a recent study of personnel and civilian workers stationed or employed at Camp Lejeune during a period when the drinking water was contaminated, positive associations for marginal zone lymphoma and mantle cell lymphoma (two separate non-Hodgkin lymphomas) were seen with Marine/Navy personnel, but not for all non-Hodgkin lymphomas, DLBCL, or follicular lymphoma.⁵⁶ Of note, among civilian workers, the adjusted HR for all non-Hodgkin lymphomas was 1.19 (95% CI = 0.83-1.71), and was even greater for DLBCL (HR = 1.30 (95% CI = 0.73-2.32)). A mortality study done by the same group also evaluating Camp Lejeune and Camp Pendleton subjects did not show a significant increased risk of non-Hodgkin lymphoma, again with the caveat that mortality studies are insensitive to detect associations with cancers, such as some non-Hodgkin lymphomas, with either an indolent clinical course or relatively high remission/cure rates, like DLBCL.⁵⁷

VIII. DISCUSSION OF VIDANA CASE FACTS

According Mr. Vidana's deposition transcript, his exposure to the water at Camp Lejeune occurred from May 12, 1983, until June 30, 1983. During his time at Camp Lejeune, Mr. Vidana testified that, following running, he would shower and "drink 5 gallons of water off the sink." He noted that he "couldn't drink enough water" while at Camp Lejeune. He also testified that he showered "much more" than twice a day because he was criticized for perspiring so much, approximately 3 to 5 times per day on weekdays, and 2 to 3 times on weekends, sometimes for more than 15-20 minutes at a time. Throughout the deposition, [REDACTED]

As detailed previously, there are several known and well-described clinical factors and exposures that would increase one's risk for developing a diffuse large B-cell lymphoma (DLBCL). [REDACTED]

[REDACTED] Additionally, DLBCL can arise in the setting of immune deficiency/dysregulation (e.g., related to HIV infection or immunosuppressive treatment for inflammatory/autoimmune disorders, or after solid-organ or bone marrow transplantation). [REDACTED]

[REDACTED] According to the

⁵⁶ Bove FJ, Greek A, Gatiba R, 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. *Environ Health Perspect.* 2024;132:107008.

⁵⁷ Bove FJ, Greek A, Gatiba R, et al. Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study. *Environ Health.* 2024;23:61.