Exhibit 502

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.

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. *Jose Vidana was diagnosed with Diffuse Large B-cell Lymphoma in October of 2007.*

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.*

4. *Jose Vidana spent time at Camp Lejeune and, over the course of approximately 6 weeks was exposed to water contaminated with chemicals including TCE, PCE, and Benzene.*

5. *The contaminated water to which Mr. Vidana was exposed during his time in Camp Lejeune was as likely as not a cause of his non-Hodgkin lymphoma.*

III. METHODOLOGY FOR ASSESSING CAUSATION OPINIONS

In order to form the opinions that I have detailed in this report, I have followed the methodology that I routinely use in my daily practice as an anatomic and clinical pathologist, employing the same rigorous standards and principles in order to research, examine, evaluate, and interpret the pertinent scientific and medical evidence in this case, an approach that I have also used in the past while performing research and practicing pathology that has been scrutinized and accepted within the peer-reviewed literature. The practice of a successful anatomic and clinical pathologist heavily relies on the constant and repeated incorporation of basic scientific and biological mechanisms of disease, pertinent clinical information, etiological risk factors, including potential causes of a disease process, and epidemiological data regarding diagnostic entities. It is by ultimately combining this information with accurate interpretation of the diagnostic laboratory material that I am able to come to the best diagnosis with the highest degree of medical certainty possible. In order to effectively function at a high-level within the rapidly advancing field of pathology, I not only maintain knowledge of the current, available information within the scientific literature, but also continually increase my proficiency in new data and techniques that can be applied towards improving my diagnostic acumen. In my clinical practice, I regularly review, interpret, evaluate, and incorporate the same types of medical and scientific evidence that I have included as references to this report, including epidemiological studies, meta-analyses, and systematic review. Careful evaluation and consideration of epidemiological data and studies is of paramount importance within my practice, as medicine, and, in particular the field of pathology is not static, but rather is constantly changing and advancing as general knowledge increases and as molecular characterization of diseases expands. It is now known that many pathologic conditions share molecular mutations/signatures, some of which were initially believed to be unique to a particular disease process.[1] Proper identification and distinction of these entities therefore require careful consideration and correlation with clinical, epidemiological, and microscopic features in order to arrive at the correct diagnosis so that the patient can be managed appropriately. It is this practice and practice philosophy that I have utilized and that I have built upon as part of my background, education, and training, in order to remain successful in my field, and is what I have applied towards my opinions that I render in this report.

Most of the articles I evaluated were acquired through my subsequent own independent study that consisted of an extensive utilization of scientific and medical search engines, which led me to various textbooks and journals from around the world. I also reviewed scientific literature and scientific evidence provided to me by Plaintiffs' counsel that was produced in discovery or used in depositions. The literature I have reviewed in assessing this case includes experiments performed using a diverse array of study designs from many laboratories that have reported results and have reached varying conclusions predominantly over the last two-to-three decades with regards to the concepts addressed in this causation analysis. In reviewing the volume and strength of this data, I have considered many variables in order to address the validity and limitations of these studies, including the reliability of the study design, the particular groups studied, the study power and statistical significance in addition to overall and consistent

[1] Dermawan JK, Vanoli F, Herviou L, et al. Comprehensive genomic profiling of *EWSR1/FUS-CREB* translocation-associated tumors uncovers prognostically significant recurrent genetic alterations and methylation-transcriptional correlates. *Mod Pathol*. 2022;35:1055-65.

trends, possible bias, replication of study findings among other researchers, accounting for confounding variables, type and degree of exposure, and biological plausibility. Although occasionally some groundbreaking experiments can result in a major shift in a previously held medical and scientific opinion regarding certain biological mechanisms of disease, it is often important not to place an inordinate amount of weight on a single study, particularly if the results and conclusions from such an analysis do not fit into a generally held concept that is based on an opinion that is both biologically plausible and otherwise supported by the remaining body of the medical and scientific literature. On the other hand, as is often the case within the scientific and medical community, occasional studies focusing on potential and previously studied risk factors for a particular disease that are published, due in combination to a well-designed study method, power, and strength of findings, are often vital in providing strong evidence to support causation with respect to the pathogenic process being evaluated. Therefore, my opinions are based on the totality of the evidence, though, as stated, some studies carry more weight than others, and I independently interpret all studies based on the considerations detailed above.

An important part of my methodology is to consider valid, alternate, and opposing perspectives when assessing the entire body of scientific and medical literature that I have reviewed. I have considered these viewpoints and address them within the body of my report, though ultimately concluding with a high degree of medical certainty that exposure to the water at Camp Lejeune was a significant cause of Mr. Vidana's non-Hodgkin lymphoma.

IV. JOSE VIDANA – CLINICAL SUMMARY

Jose Vidana was born on [redacted] 1963. His past medical/surgical history is significant for mild hypertension, allergies with sinus infections, obesity, and a prior eye surgery as a child. His social history was notable for cigarette smoking consisting of approximately 5 cigarettes per day from the age of 24. He additionally reports occasional alcohol usage. His family history is significant for a sister with breast cancer. He worked primarily as an operations manager for a law firm in Los Angeles prior to his cancer diagnosis.

On October 24, 2007, Mr. Vidana underwent an open lymph node biopsy in his left neck, performed by Dr. Dennis Crockett at USC University Hospital for a preoperative history of an enlarging left-sided neck mass with increasing shortness of breath. He had reportedly initially presented with a nontender mass back in June of 2007, that he noticed while shaving, at which time he believed that it was secondary to his known allergies and history of sinus infections. He then went to an urgent care center in early September 2007, where a health care provider prescribed him a course of antibiotics along with nasal spray. After his neck mass did not resolve, he returned to the same urgent care center and was prescribed a second course of antibiotics. After the mass still failed to resolve, he was referred to an ENT physician for further work-up. He was then seen by Dr. Dun Ha, an ENT specialist on September 17, 2007, at which time a fine needle aspiration (FNA) of the mass was performed. The results of this analysis reportedly showed malignancy, though no further subclassification was provided at that time. He additionally was noted to have a 20-pound weight loss over this same time period. Following the results of the FNA, Dr. Ha referred Mr. Vidana to Dr. Crockett for further tissue biopsy. A frozen section performed at the time of the biopsy was notable for a "small round blue cell tumor," and a lymphoma work-up (tissue sent for flow cytometry) was initiated. Mr. Vidana was admitted for overnight observation of his airway, as he had a known history of obstructive sleep apnea, and did not have any adverse events, thus was discharged the following day on October 25, 2007. Following an extensive pathologic work-up of the tissue procured from the biopsy of the left-sided neck mass, a diagnosis of **"large B-cell lymphoma" (Diffuse Large B-cell Lymphoma; WHO Classification)** was rendered by the pathologist.

Mr. Vidana was eventually admitted following a clinic visit with Dr. Ann Mohrbacher on October 31, 2007, because of increased shortness of breath and a rapidly growing lymphoma with airway

obstruction. He was given Decadron 20 mg IV and it was noted that his breathing significantly improved, with resolution of his shortness of breath. On physical examination he had an oxygen saturation of 94% on room air. He was noted to have a large neck mass extending from the left submandibular area to the anterior auricular region and the posterior left neck. He also was found to have a right submandibular area mass, a right supraclavicular mass, and right inguinal lymphadenopathy. Based on these physical examination findings, his lymphoma was considered to be Stage III, and ultimately underwent CT scans which revealed extensive lymphadenopathy within the neck, with foci of mass encirclement of the proximal internal carotid artery, as well as prominent retroperitoneal, pelvic, and inguinal lymphadenopathy. An echocardiogram showed a normal left ventricular size and ejection fraction. Mr. Vidana was subsequently discharged the following day on November 1, 2007, with plans to start R-CHOP chemotherapy (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone). A bone marrow biopsy performed on November 5, 2007, was essentially unremarkable, without evidence of lymphoma.

On November 7, 2007, Mr. Vidana began his first of 6 cycles of chemotherapy for his diffuse large B-cell lymphoma, and prior to commencement of the third cycle on December 7, 2007, his previously massive left-sided cervical lymphadenopathy was found to have resolved, with improvement of his upper respiratory symptoms. His chemotherapy course was complicated by a severe neutropenia following his 5th cycle of chemotherapy, requiring Neupogen, though his white blood cell count subsequently recovered. He was noted to have radiographic evidence of a near complete response within the nasopharynx and left cervical lymph node chain with questionable radiographic persistent disease within the pelvis and/or possibly right clavicle.

On February 19, 2008, Mr. Vidana was seen by Dr. Paul Pagnini, Assistant Professor of Radiation Oncology, for evaluation of possible consolidated radiation treatment. On physical examination, he was found to have mild left cervical lymphadenopathy/fullness without an appreciable mass (compared to the right side). No submandibular, supraclavicular, axillary, epitrochlear, inguinal, or popliteal lymphadenopathy was appreciated. Based on these findings, he was felt to be a candidate for radiotherapy to the bulky left cervical lymph node area and nasopharynx. He therefore underwent involved field radiation therapy beginning on March 4, 2008, with his last treatment occurring on April 9, 2008, without any reported difficulties. Following the radiation treatments, he received consolidated rituximab therapy in August and September of 2008. After completion of his therapy, he was found to have no evidence of disease on subsequent CT scans of the chest, abdomen, and pelvis, as well as by full-body PET scan. His lymphoma has been in clinical remission since completion of this therapy in 2008, until the present day. However, Mr. Vidana's subsequent medical records since his diagnosis of diffuse large B-cell lymphoma are significant for the development of left leg sciatica, diabetes mellitus, and hypothyroidism, confirmed by the deposition transcript from his internal medicine primary care physician, Dr. Radhika Tulpule, who reportedly saw him regularly for annual visits.

V. DIFFUSE LARGE B-CELL LYMPHOMA:

Large B-cell lymphomas, as detailed in the most recent World Health Organization classification of hematolymphoid tumors, are a subtype of non-Hodgkin lymphoma that represent a wide range of tumors that have varying morphologies, genetic features, and clinical behaviors.[2] Diffuse large B-cell

[2] Ott G, Siebert R, Alaggi R, et al. Large B-cell lymphomas: Introduction. In: WHO Classification of Tumours Editorial Board. Haematolymphoid tumours. Lyon (France): International Agency for Research on Cancer; 2024. . (WHO classification of tumours series, 5th ed.; vol. 11).

lymphoma (DLBCL) NOS represents the most common entity within this subgroup, though is heterogeneous with respect to clinical, morphological, phenotypical, and genotypic features. This new classification specifically recognizes 17 separate and specific entities as within the group of large B-cell lymphomas, outside of the DLBCL-NOS subtype, all separated according to their genetic characteristics, clinical context, and/or site of origin.

Diffuse large B-cell lymphoma (DLBCL) NOS consists of medium- to large-sized B cells that generally have a diffuse growth pattern histologically, and do not meet the diagnostic criteria for any of the specific large B-cell lymphoma malignancies. DLBCL represents the most common type of non-Hodgkin lymphoma,[3] constituting approximately 30% of all adult lymphoma cases, and although the majority of patients present with disease restricted to lymph nodes, a significant minority have tumor confined to an extranodal site at the time of presentation.[4] Although it can be diagnosed at any age, DLBCL more commonly develops with increasing age, peaking in elderly patients.[5]

Although the etiology of diffuse large B-cell lymphoma (DLBCL)-NOS is unknown in the majority of cases, well-known risk factors include a family history of lymphoma, immune deficiency/dysregulation, and viruses (namely Hepatitis C, Epstein Barr Virus, and Human Immunodeficiency Virus).[6] In addition, DLBCL may sometimes present as transformation from an underlying more indolent B-cell lymphoma, such as small lymphocytic lymphoma, follicular lymphoma, and marginal zone lymphoma, among others.[7] Exposure to various environmental and occupational carcinogens has also been associated with the development of various non-Hodgkin lymphomas, including DLBCL.[8,9]

VI. CONTAMINANTS FOUND WITHIN CAMP LEJEUNE WATER

In forming my opinions regarding Mr. Vidana's exposure to carcinogens while stationed at Camp Lejeune, I am relying on a report titled "ATSDR Assessment of the Evidence for the Drinking Water

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[3] The Non-Hodgkin's Lymphoma Classification Project. A clinical evaluation of the International Lymphoma Study Group classification of non-Hodgkin's lymphoma. Blood. 1997;89:3909-18.

[4] Anderson JR, Armitage JO, Weisenburger DD. Epidemiology of the non-Hodgkin's lymphomas: distributions of the major subtypes differ by geographic locations. Non-Hodgkin's Lymphoma Classification Project. *Ann Oncol.* 1998;9:717-20.

[5] Roman E, Smith AG. Epidemiology of lymphomas. *Histopathology*. 2011;58:4-14.

[6] Cerhan JR, Kricker A, Paltiel O, et al. Medical history, lifestyle, family history, and occupational risk factors for diffuse large B-cell lymphoma: the InterLymph Non-Hodgkin Lymphoma Subtypes Project. *J Natl Cancer Inst Monogr.* 2014;2014:15-25.

[7] Cerhan JR, Kricker A, Paltiel O, et al. Medical history, lifestyle, family history, and occupational risk factors for diffuse large B-cell lymphoma: the InterLymph Non-Hodgkin Lymphoma Subtypes Project. *J Natl Cancer Inst Monogr.* 2014;2014:15-25.

[8] Ferri GM, Specchia G, Mazza P, et al. Risk of lymphoma subtypes by occupational exposure in Southern Italy. *J Occup Med Toxicol.* 2017;12:31.

[9] Schinasi L, Leon ME. Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis. *Int J Environ Res Public Health.* 2014;11:4449-527.

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

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[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. TRICHLOROETHYELENE (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

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[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

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[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 IinterLlymph] 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

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[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

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[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 was diagnosed with in October of 2007.[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

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[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, Järvholm 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-

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[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, Mr. Vidana denied exposure to any other hazardous chemicals or solvents while at home or at work. He denied exposure to prior radiation. He also did not have any known family history of immune deficiencies or cancer in his mother or siblings. Mr. Vidana testified that after his last round of chemotherapy, he began having joint pain after riding his motorcycle and was physically exhausted, necessitating him selling his motorcycle, something he stated that he had mentioned to his oncologist. Similarly, shortly after his lymphoma treatment, he stopped fishing, a common hobby of his, as well as golfing, due to significant pain.

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). Specifically, there was no mention in either the left neck incisional biopsy pathology report or within the subsequent bone marrow biopsy report that Mr. Vidana had any evidence of background, pre-existing low-grade lymphoma from which his DLBCL arose from. 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). Not only did Mr. Vidana have no evidence of an immune deficiency/dysregulation or autoimmune disorder, but there was also no evidence of a family history of such a condition in his known relatives. According to the

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[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.

deposition transcript of Dr. Ann Mohrbacher from May 17, 2024, Mr. Vidana was specifically tested for HIV infection (and was found to be negative) and had not undergone prior transplantation, etiologies that were mentioned in her sworn testimony that can be ruled out as contributing causes for his diagnosis of DLBCL-NOS. Mr. Vidana also did not have a history of exposure to ionizing radiation prior to his lymphoma diagnosis, another known risk factor for the development of DLBCL, both according to the scientific literature and the sworn testimony of Dr. Mohrbacher. Finally, as was discussed previously, at the time of Mr. Vidana's cancer diagnosis, DLBCL encompassed a variety of different large B-cell lymphomas that have since been subclassified by the 5th edition of the World Health Organization Classification of Hematopoietic Tumors, many of which are related to underlying etiologies, such as known EBV infection ("EBV-positive diffuse large B-cell lymphoma"), sites of chronic fibrin deposition in confined or acquired anatomic spaces ("fibrin-associated large B-cell lymphoma"), in the setting of serous effusions ("fluid overload-associated large B-cell lymphoma"), and in the setting of longstanding chronic inflammation ("diffuse large B-cell lymphoma associated with chronic inflammation"), none of which apply to Mr. Vidana's case or ultimate diagnosis of DLBCL.

A significant fact to incorporate into the findings in this case in the context of literature assessing non-Hodgkin lymphoma, including DLBCL, risk and exposure to individual chemicals, is the known impact of exposure to numerous chemicals simultaneously that can have a synergistic effect with respect to carcinogenesis. In support of this are the increased risks seen in many of the Camp Lejeune-specific studies, where those individuals exposed to contaminated water containing elevated levels of numerous known carcinogens. In the case of DLBCL, a study examining the spatial patterns of this subtype of non-Hodgkin lymphoma by linking geocoded data on toxic release sites in Georgia over a 10-year period (1988-1998), including TCE, PCE, and benzene release sites, found that DLBCL incidence was significantly associated with distance from these locations, arguing in favor of a combined and synergistic effect exposure to multiple carcinogens would have on the development of malignancy.[58]

In coming to my conclusions in this report, I used a differential etiology process applied to the question whether his exposure to the chemicals in the water at Camp Lejeune is "at least as likely as not" a cause of Mr. Vidana's DLBCL. This methodology determines the possible causes of the DLBCL and reviews Mr. Vidana's medical history, exposure to NHL-causing agents, and family history to determine the presence of one or more of those causes. Here, as discussed above, Mr. Vidana has only one generally accepted risk factor for NHL – exposure to water at Camp Lejeune for sufficient time and at sufficient concentrations to be a cause of his NHL. We know that Mr. Vidana was exposed to the water at Camp Lejeune for more than 30 days and based on his testimony, that was an exposure consistent with the ATSDR report and the Congressional 30 day time frame. There is no evidence of any other cause here—there was no exposure to other chemicals at work or home, radiation, prior infections, or prior germane medical history. In this context, with an absence of evidence of other causes, and with the length and concentration of exposure to the water at Camp Lejeune, I conclude that his exposure to the Camp Lejeune water is "at least as likely as not" a cause of Mr. Vidana's DLBCL.

IX. CONCLUSION

Based on the totality of the literature I have reviewed in conjunction with this case, predominantly consisting of peer-reviewed and published research studies, and in combination with all of the evidence of this case including medical records and deposition testimony, it is my opinion that Mr. Vidana's exposure to the contaminated water at Camp Lejeune containing elevated mean concentrations

[58] Bulka C, Nastoupil LJ, Koff JL, et al. Relations Between Residential Proximity to EPA-Designated Toxic Release Sites and Diffuse Large B-Cell Lymphoma Incidence. *South Med J.* 2016;109:606-614.

of TCE, PCE, and benzene between May 12, 1983, and June 30, 1983, was a at least as likely as not a cause for Mr. Vidana's diffuse large B-cell lymphoma (DLBCL) diagnosed in October of 2007.

All of the opinions I have expressed in this report are based on a combination of my medical training, personal experience as a diagnostic anatomic and clinical pathologist, my frequent interactions with clinical colleagues in the day-to-day management of patients, and my past and ongoing extensive review of pertinent literature in the field. I strongly express these opinions with a reasonable degree of medical certainty.

2/1/2025

Date

Paul J. Michaels, M.D.

Exhibit 1

PAUL J. MICHAELS, M.D.

4000 Beach Loop Road, Bandon, Oregon 97411
PHONE: (512) 808-6711 EMAIL: pauljmichaels@gmail.com

PROFESSIONAL EXPERIENCE

January 2021 – Present

Pathologist at Pathology Consultants Coos Bay, OR

- Member of ~10 pathologist group that operates the largest and most comprehensive anatomic pathology services laboratory within Central and Coastal Oregon.
- One of 2 pathologists responsible for covering Bay Area Hospital, the largest hospital on the Oregon Coast, which serves as a regional referral center.
- Bay Area Hospital
  - Chairman of Pathology and member of Hospital Medical Executive Committee (1/2022-Present)
  - Medical Director for Transfusion Medicine, Hematology, and Cytopathology (1/2021-Present)
  - Member of Cancer Committee
  - Member of Trauma Committee
- Southern Coos Hospital and Medical Center
  - Laboratory Medical Director (1/2021–Present)
- Anatomic pathology responsibilities include frozen sections, gross evaluation and sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, presentation at tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, and evaluation of blood transfusion reactions

February 2013 – December 2020

Pathologist and Shareholder at Clinical Pathology Associates Austin, TX

- Member of ~50 pathologist group that covers several major hospitals in the Central Texas region (including the Austin area and surrounding communities, San Marcos, Waco, and San Antonio)
  - Vice President (2018–2020) and Director of the Operations Committee
- Anatomic pathology responsibilities include frozen sections, ROSE, fine needle aspiration of superficial sites, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, presentation at tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions and RBC antibody serology work-ups, TEG analysis, and evaluation of protein electrophoresis panels
- St. David's Medical Center:
  - Chairman of Pathology (2020):
  - Member of the hospital Medical Executive Committee (2020)
  - Member of Quality Committee (2018–2020)
  - Member of Infection Prevention Committee (2018 –2020)
  - Pathology representative of region-wide St. David's Healthcare Lung Cancer Working Group (2015–2020)
- Laboratory Medical Director for:
  - First Texas Hospital, Houston, Texas (2016-2020)
  - Clinical Pathology Laboratories North Stat Laboratory (2013-2020)
  - Clinical Pathology Laboratories South Stat Laboratory (2013-2020)
- College of American Pathologists (CAP) Inspections:

- Team Leader (June 2014)

July 2006 – January 2013

Pathologist at Laboratory Medicine Consultants/Aurora Diagnostics Las Vegas, NV

- Member of a ~18 pathologist group that covered several hospitals in the southern Nevada and northwestern Arizona region
  - Steering/Executive Committee Member (2009-2013)
- Anatomic pathology responsibilities include frozen sections, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, coverage of outpatient FNA clinic, presentation at numerous tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions, protein electrophoresis and immunofixation analysis, and interpretation of various chemistry, lipid, coagulation, and serologic laboratory test panels
- MountainView Hospital:
  - Laboratory Medical Director (2010-2013)
  - Member of the Medical Executive Committee (2010-2013)
  - Member of the Quality Council (2010-2013)
- Sunrise Hospital and Medical Center:
  - Cancer Conference Coordinator (2007–2009)
  - Cancer Program Activity Coordinator (2007–2009)
- Outpatient Cytopathology Laboratory Director (2007–2010)
- College of American Pathologists (CAP) Inspections:
  - Team Leader (September 2009, August 2010)
  - Team Member (March 2007, September 2010)
- In 2012, was voted a "Top Doctor" in Pathology in Las Vegas, NV by Consumers' Checkbook of Washington, D.C., published in *Vegas Seven* magazine (2/23/2012).
  - Received the most votes of any pathologist in the city

October 2004 – June 2006

Locum Tenens Pathologist at Commonwealth Pathology Partners Salem, MA

- Independently performed autopsies at North Shore Medical Center that occurred during the weekends and holidays
- Prepared and signed-out (cosigned) the autopsy reports with an attending/supervising pathologist

EDUCATION/CLINICAL TRAINING

July 2005 – June 2006

Cytopathology Fellowship, Massachusetts General Hospital/Harvard Medical School

- Included Elective Subspecialty GYN and GI pathology sign-out (1-month each)

June 2001 – June 2005

Anatomic/Clinical Pathology Residency, Massachusetts General Hospital/Harvard Medical School

- Chief Resident, Anatomic Pathology (June 2004 – November 2004)
- Resident Representative for Mentoring, American Society of Cytopathology, Ethics and Conduct Committee (2004–2006)

August 1996 – June 2001

Doctorate of Medicine, University of California, Los Angeles

- Post Sophomore Fellowship in Anatomic/Clinical Pathology, Combined UCLA/Cedars Sinai Program (June 1998 – June 1999)
- Alpha Omega Alpha Honor Society (Elected 2001)

September 1992 – September 1995

Bachelor of Science, University of California, Irvine

- Major in Biological Sciences with a Minor in Microbiology, *Cum Laude*
- Awarded "Excellence in Research" Award
- Phi Beta Kappa Honor Society (Elected 1995)
- UC Regents Scholar (1992–1995)
- Golden Key National Honor Society

ACADEMIC APPOINTMENTS

January 2023 – Present

Clinical Assistant Professor in the Department of Pathology & Laboratory Medicine, Oregon Health & Science University (OHSU)

- Recipient of the OHSU Department of Pathology "Most Innovative Teaching Award," 2022-2023

March 2018 – December 2020

Assistant Professor of Diagnostic Medicine, Dell Medical School, The University of Texas at Austin

- Division Chief of Head and Neck Pathology
- Dell Medical Admissions Applicant Review Committee Member
- Faculty Senate, member

June 2009 – June 2013

Adjunct Associate Professor of Pathology, Touro University of Nevada, College of Osteopathic Medicine

June 2001 – June 2006

Clinical Fellow/Instructor in Pathology, Harvard Medical School

- Focus on teaching medical renal pathology to second year medical students.

CERTIFICATION

October 2006 – Present

American Board of Pathology, Cytopathology (Time Limited, Recertified - March, 2015)

August 2005 – Present

American Board of Pathology, Anatomic and Clinical Pathology (Time Unlimited)

MEDICAL LICENSURE

Oregon (License # MD201747)
California (License # C55645)

PROFESSIONAL ORGANIZATIONS

American Medical Association (AMA)
Oregon Medical Association (OMA)
American Society of Cytopathology (ASC)
American Society of Clinical Pathology (ASCP)
College of American Pathologists (CAP)
United States and Canadian Academy of Pathology (USCAP)

AD HOC REVIEWER

Cancer Cytopathology
American Journal of Clinical Pathology

ACADEMIC PUBLICATIONS

Yeager TS, Stroh BC, El Youssef R, and **Michaels PJ**. Ectopic Prostatic Tissue Involving the Omentum and Presenting with Intussusception and Small Intestinal Obstruction: A Report of a Rare Case with a Review of the Literature. *Human Pathology Reports* 2022;30:300679.

Pusztaszeri M. Wang H, Cibas ES, Powers CN, Bongiovanni M, Ali S, Khurana KK, **Michaels PJ**, and Faquin WC. Fine-needle Aspiration Biopsy of Secondary Neoplasms of the Thyroid Gland: a Multi-institutional Study of 62 Cases. *Cancer Cytopathol* 2015;123:19-29.

Lewis Jr. BA, Zebrowski B, Yumiaco NS, **Michaels P**, and Erling M. Case Report of Paratesticular Liposarcoma with Metachronous Large Renal Cell Carcinoma. *Curr Urol* 2010;4:162-163.

Pitman MB, **Michaels PJ**, Deshpande V, Brugge WR, and Bounds BC. Cytological and Cyst Fluid Analysis of Small (<3 cm) Branch Duct Intraductal Papillary Mucinous Neoplasms Adds Value to Patient Management Decisions. *Pancreatology* 2008;8:277-84.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasms (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. *Cancer* 2006;108:163-73.

Steele DJ and **Michaels PJ**. Case Records of the Massachusetts General Hospital. Weekly Clinicopahtological Exercises. Case 40-2004- A 42-year-old Woman with Long-Standing Hematuria. *N Engl J Med* 2004;351:2851-9.

Michaels PJ, Espejo ML, Kobashigawa J, Alejos JC, Burch C, Takemoto S, Reed EF, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Risk Factors, Hemodynamic Consequences and Relationship to Transplant Coronary Artery Disease. *J Heart Lung Transpl* 2003;1:58-69.

Michaels PJ, Fishbein MC, and Colvin RB. Humoral Rejection in Human Transplantation. *Springer Semin Immunopathol* 2003;25:119-140.

Marchevsky AM, Lau SK, Khanafshar I, Ockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Internet Teleconferencing Method for Telepathology Consultations from Lung and Heart Transplant Patients. *Hum Pathol* 2002;33:410-4.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine, TGF-β, and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. *J Heart Lung Transpl* 2000;20:407-16.

Michaels PJ, Kobashigawa J, Child JS, and Fishbein MC. Chronic Right Sided Myocarditis Mimicking Arrhythmogenic Right Ventricular Dysplasia. *Hum Pathol* 2000;31:618-21.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-Based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. *Lab Invest* 2000;80:37A.

Michaels PJ and Mautz WJ. Effects of Inhaled Ozone and Formaldehyde on Tracheal Epithelial Secretion of Rats Exposed During Rest and Exercise. *Journal of Undergraduate Research in the Biological Sciences* 1995;25:779-90.

PRESENTATIONS

INVITED TALKS:

March 2022

"Cervical Cancer Screening: An Update" at Bay Area Hospital, Coos Bay, Oregon. Invited Grand Rounds Speaker.

November 2019

"Recognition of Germline Mutations in General Surgical Pathology" at University of Texas Medical Branch at Galveston, Department of Pathology. Invited Speaker.

August 2018

"Screening Laboratory Tests: Recent Guidelines from the Newborn Nursery to the Nursing Home" at St. David's Medical Center, Austin, Texas. Invited Grand Rounds Speaker.

May 2018

"Interesting FNA Cases from a Busy Outpatient Clinic" at George Washington University and Health Sciences, Department of Pathology. Invited Speaker.

April 2015

"The Surgical Pathology of Dysphonia" for the Masters Program in Speech and Language Pathology at University of the Pacific, Stockton, California. Invited Speaker.

January 2014

"Sin City Cytology" at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Grand Rounds Speaker.
"Cytology Jeopardy" at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Unknown Conference for Residents.

March 2013

"Confounding Metastatic Breast Cancer Controversy" presented at the 23rd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Planet Hollywood Resort & Casino. Las Vegas, Nevada. Invited Speaker.

March 2012

"Interesting Cases: What Would You Have Done?" presented at the 22nd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Paris Las Vegas Hotel & Casino. Las Vegas, Nevada. Invited Speaker and Panelist.

November 2010

"Cytology From Sin City 2" at Massachusetts General Hospital and Brigham and Women's Hospital, Departments of Pathology, Harvard Medical School. Invited Speaker.

February 2009

"Cytology From Sin City" at Massachusetts General Hospital, Brigham and Women's Hospital, and Beth Israel Deaconess, Departments of Pathology, Harvard Medical School. Invited Speaker.

December 2003

"Thin Basement Membrane Nephropathy and Alport Syndrome" at Massachusetts General Hospital, Clinicopathologic Conference (Published in *N Engl J Med*), Harvard Medical School.

PLATFORM PRESENTATIONS:

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasm (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Annual Meeting of United States and Canadian Academy of Pathology. March 2004. Vancouver, British Columbia, Canada.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. 20th International Society of Heart and Lung Transplantation Annual Meeting. April, 2000. Osaka, Japan.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin Prolongs Rat Survival after Orthotopic Liver Transplantation by Decreasing the

Expression of β2-Glycoprotein-1 and Proinflammatory Cytokines. World Congress of the Transplantation Society. July 12-17, 1998. Montreal, Quebec, Canada.

POSTER PRESENTATIONS:

Michaels PJ, Bounds BC, Brugge WR, Lewandrowski K, Pitman MB. The Clinical Utility of Cyst Fluid Analysis in Conjunction with Cytological Evaluation in the Preoperative Characterization and Subclassification of Pancreatic Mucinous Cysts. 52nd American Society of Cytopathology Annual Meeting. November, 2004. Chicago, IL.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplams (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Massachusetts General Hospital Clinical Research Day. June, 2004. Boston, MA.

Michaels PJ, Kobashigawa J, Espejo ML, Alejos JC, Burch C, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Recent UCLA Experience. Sixth Banff Conference on Allograft Pathology. April, 2001. Banff, Canada.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. 2000 Annual Meeting of United States and Canadian Academy of Pathology. March, 2000. New Orleans, LA.

Kakkis JL, Schmit P, **Michaels PJ**, and Thompson J. Management of Gallstone Disease During Pregnancy in the Era of Laparoscopic Cholecystectomy. The Southwestern Surgical Congress. April, 1999. Coronado, CA.

Kakkis JL, **Michaels PJ**, Ke B, Zhao D, Kato H, Imagawa D, Kupiec-Weglinski JW, and Busuttil RW. Treatment with Pravastatin Ameliorates Rejection and Improves Survival in Liver Transplanted Rats. International Congress on Immunosuppression. December 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Gornbein J, Terasaki P, Imagawa D, Busuttil R. Multivariate Analysis of Risk Factors in 1,008 Orthotopic Liver Transplant Recipients Reveals Significant Influence of Panel Reactive Antibody on Patient and Graft Survival. Annual Meeting of the American College of Surgeons, October 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin-induced Survival in Rat Orthotopic Liver Transplantation is Accompanied by Diminished Expression of β2-Glycoprotein-1 and Proinflammatory Cytokines. American Society of Transplant Physicians. May, 1998. Chicago, IL.

Michaels PJ, Ma J, Zhao D, Imagawa D, Busuttil R, and Kakkis JL. Pravastatin Treatment is Associated with Downregulation of TGF-β and TNF-α in Liver Transplanted Rats. 1997 Short Term Training Program Poster Session. Los Angeles, CA.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Zhao D, Imagawa DK, and Busuttil RW. Analysis of Genetic Modifications in Liver Transplanted Rats Utilizing Messenger RNA Differential display. American Society of Transplant Surgeons. May, 1997. Chicago, IL.

Exhibit 2

PAUL J. MICHAELS, M.D.

BOARD CERTIFIED IN ANATOMIC AND CLINICAL PATHOLOGY, AND CYTOPATHOLOGY

4000 Beach Loop Drive SW
Bandon, OR, 97411

MOBILE — (512) 808-6711
Email: *pauljmichaels@gmail.com*

SCHEDULE OF FEES

1.	**Cognitive work, lab work, records review, meetings**	**$600.00/hour**
2.	**Depositions***	**$600.00/hour**
3.	**Court Testimony****	**$3,000/half day** **$5,500/whole day**
4.	**Overnight**	**As above, plus per diem ($1000.00)**
5.	**Interest**	**All invoices are due and payable within 15 days. Invoices 30 days past due will be charged interest at the rate of 10% per month and will additionally be assessed a $500 late fee.**

**** Depositions are charged at a two hour minimum. Any cancellations less than 48 hours will be charged two hours. Paid by side taking the deposition (unless otherwise agreed upon).***

***** Cancellations of court appearances, etc., occurring less than 48 hours of the event will be billed at 50% of the scheduled rate.***

Exhibit 3

PAUL J. MICHAELS, M.D.

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PRIOR DEPOSITIONS / COURT TESTIMONIES (4 YEAR HISTORY)

DEPOSTIONS	**DATE**
Leslie Coates (Plaintiff) vs. Ethicon, Inc., et al. *Deposed as expert Pathology witness for Plaintiff*	2/6/21
Catalina Torres (Plaintiff) vs. Boston Scientific Corporation *Deposed as expert Pathology witness for Plaintiff*	5/25/21
Heidi McKenna and Andrew McKenna (Plaintiff) vs. Boston Scientific Corporation *Deposed as expert Pathology witness for Plaintiff*	11/8/21
Re: Zantac (Ranitidine) Products Liability Litigation and Defendants Boehringer Ingelheim Pharmaceuticals, Inc., GlaxoSmithKline LLC, Pfizer Inc., Sanofi US Services Inc., Sanofi-Aventis U.S. LLC, and Chattem, Inc. *Deposed as expert Pathology witness for Plaintiff*	5/22/22
Marisol Datil (Plaintiff) vs. C. R. Bard, Inc. *Deposed as expert Pathology witness for Plaintiff*	6/5/22
Re: Zantac (Ranitidine) Products Liability Litigation and Defendants Boehringer Ingelheim Pharmaceuticals, Inc., GlaxoSmithKline LLC, Pfizer Inc., Sanofi US Services Inc., Sanofi-Aventis U.S. LLC, and Chattem, Inc. *Deposed as expert Pathology witness for Plaintiff regarding supplemental report*	10/18/22

COURT TESTIMONIES	
Teri Freeman (Plaintiff) vs. Ethicon *Testified as expert Pathology witness for Plaintiff*	9/13/22