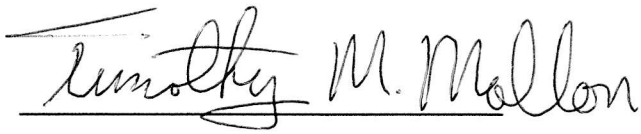
Exhibit 404

Specific Causation Report of Timothy M. Mallon, M.D., M.P.H., MS. For Ms. Jacquiline Tukes

Prepared by:



Timothy M. Mallon, M.D., M.P.H., M.S.
Occupational and Environmental Medicine
Adjunct Assistant Professor of Preventive Medicine
Uniformed Services University
Bethesda, MD 20814

Consultant Address:
6508 Folded Leaf Square
Columbia, MD 21044, USA

Email: mallonti03@gmail.com

Table of Contents

I. Summary of Opinions

This report summarizes my medical expert opinion regarding the specific causation of Ms. Jacqueline Tukes' kidney cancer and the causal relationship between exposure to the chemicals in the water at the Camp Lejeune military base, including trichloroethylene (TCE), tetrachloroethylene (PCE), benzene, and vinyl chloride, and the development of Ms. Tukes' kidney cancer. My opinions are based on my professional education, training and experience, knowledge of the pertinent scientific and medical literature reasonably relied upon by others in my profession and the documents cited in this report. I am qualified to evaluate the scientific literature and to render opinions about exposure to these substances causing kidney cancer. I hold all of my opinions in this report to a reasonable degree of medical and scientific certainty. I reserve the right to amend or supplement my report should new information become available.

Based on my general causation report on December 9, 2024, and the facts of this case, I conclude to a reasonable degree of medical and scientific certainty that Ms. Tukes' kidney cancer was more likely than not caused by the toxic water at Camp Lejeune. This exceeds the "at least as likely as not" standard at issue in this litigation.

II. Expert Qualifications

I am a Medical Doctor, board-certified in Preventive Medicine (Occupational Medicine). I hold a bachelor's degree in biology from Clarkson University. I earned a master's degree in public health from Johns Hopkins University School of Public Health in 1985; a master's degree in environmental health from CUNY Hunter College in 1986; and a Master of Science in Natural Resource Policy and Management from the University of Michigan, Graduate School of Natural Resources in 1987. I attended medical school at the Syracuse Upstate Health Science Center in Syracuse, New York in 1991.

Currently, I hold an adjunct Assistant Professor position in Preventive Medicine at Uniformed Services University in Bethesda, MD, and I consult for the Veteran's Evaluation Services and the Health and Human Services Federal Occupational Health Program. Prior to my retirement from military service in 2016, I was a full professor in Preventive Medicine at the Uniformed Services University from July 2004 to July 2016.

For over six years (January 2013 – August 2019), I led a team of investigators studying the association between certain diseases and exposures to burn pit smoke in Iraq and Afghanistan. This team included researchers from the University of Rochester, Clarkson University, Emory University, Uniformed Services University, and the Armed Forces Health Surveillance Agency. We completed a health assessment of 200 service members exposed to burn pit smoke in Iraq and Afghanistan which generated over 30 publications in the peer-reviewed literature and won several awards, including grants from NIEHS, and the Department of Defense's Defense Health Agency, and earned recognition by the American College of Occupational and Environmental Medicine.

I was awarded a Lifetime Achievement Award for Leadership in Academic Medicine and Research in 2019 and the Army Surgeon General's Academic Excellence Award "the A-Designator" as the Residency Director in Occupational Medicine at the Uniformed Services University.

I served as the specialty editor for The Textbook of Military Medicine in Occupational and Environmental Medicine, the specialty editor of three supplements to the Journal of Occupational and Environmental Medicine in Workers Compensation Programs, and editor of two supplements on Burn Pit Exposures in Iraq and Afghanistan in 2016 and 2019.

I have authored or co-authored over 46 journal articles and written 23 book Chapters for the Textbook of Military Medicine and the Clinics of North America. I also served on the American Board of Preventive Medicine and the Accreditation Council for Graduate Medical Education Residency Review Committee in Preventive Medicine.

My training, expertise, and service have included work specific to environmental exposures and associated cancers. I trained specifically in toxicology, environmental health, environmental epidemiology, and cancer epidemiology as part of my coursework at Johns Hopkins University and CUNY. This included collaboration on epidemiologic studies of Agent Orange exposure, Non-Hodgkin's Lymphoma, and soft tissue Sarcoma. I also served on the Advisory Board for several Residencies in Aerospace and Occupational and Environmental Medicine for the Air Force at Brook Air Force Base, Johns Hopkins Occupational and Environmental Medicine Residency Program in Baltimore Maryland, and the Navy School of Aerospace Medicine and Occupational Medicine in Pensacola, Florida.

I have taught over 200 residents in Occupational and Environmental Medicine as the Occupational and Environmental Medicine Residency Program Director at the Uniformed Services University in Bethesda, MD from 2005 to 2016. A copy of my curriculum vitae is attached as Exhibit 1.

III. Causation Standard

I have reviewed the Camp Lejeune Justice Act of 2022 (CLJA),[1] which I understand to be the governing statute for the causation standard in this case. The CLJA requires that marines or family members bringing claims under the Act "show one or more relationships between the water at Camp Lejeune and the harm," by "produc[ing] evidence showing that the relationship between exposure to the water at Camp Lejeune and the harm is – (A) sufficient to conclude that a causal relationship exists; or (B) sufficient to conclude that a causal relationship is at least as likely as not."[1]

"As likely as not" is a standard that is less rigorous than a "more likely than not" standard. I am familiar with these terms and how the terms are applied in the sciences of environmental science, toxicology, epidemiology and other sciences dealing with these same issues.

I also reviewed the "ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases" dated January 13, 2017.[2] The

ATSDR Report used four categories to classify the strength of the evidence for a causal relationship between the chemicals in the water at Camp Lejeune and various harms.[2] The "Sufficient" and "Equipoise and Above" categories of this classification scheme employ the same language as the Camp Lejeune Justice Act: 1. Sufficient: "the evidence is sufficient to conclude that a causal relationship exists." 2. Equipoise and Above: "the evidence is sufficient to conclude that a causal relationship is *at least as likely as not*, but not sufficient to conclude that a causal relationship exists."[2] The authors of the ATSDR describe how, in their view, each of these categories can be met. For example, for "equipoise and above evidence for causation," ATSDR states:

"Equipoise and above evidence for causation: The evidence is sufficient to conclude that a causal relationship is at least as likely as not, but not sufficient to conclude that a causal relationship exists. This category would be met, for example, if:

> "1. The degree of evidence from human studies is less than sufficient but there is supplementary evidence from animal studies and/or mechanistic studies that supports causality, **or**
>
> "2. A meta-analysis does not provide convincing evidence (e.g., the summary risk estimate is close to the null value of 1.0, i.e., ≤1.1), or if the meta-analysis observes a non-monotonic exposure-response relationship) but there is at least one epidemiological study considered to be of high utility occurring after the meta-analysis has been conducted, in which an association between the exposure and increased risk of the disease of interest has been found and in which chance and biases can be ruled out with reasonable confidence.
>
> "3. A meta-analysis has not been conducted, but there is 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 has been found and in which chance and biases can be ruled out with reasonable confidence."[2]

It is my opinion that these classifications are consistent with the sciences for which they apply (i.e., they are consistent with how the sciences of epidemiology, toxicology, and other related sciences apply these standards).

Based on my years of medical and epidemiological training and expertise, I am familiar with the term "equipoise," and find ATSDR's definition of "equipoise and above" or "at least as likely as not" to be appropriate in this case. The explanations by ATSDR for how each category of classification can be met are similarly appropriate for this case and based on sound scientific principles and methodology. Moreover, I have reached my opinion in this case to a reasonable degree of medical and scientific certainty under a "more likely than not standard," which surpasses the "at least as likely as not" standard. I make clear throughout where each of my opinions are expressed under the "more likely than not" or "at least as likely as not" standard.

IV. Methodology Employed

The methodology I used to form my opinions in this case aligns with the standard practices that I and other experts utilize when conducting similar analyses. Specifically, my approach included the following as stated in my general causation report, with additional methodology for these specific causation opinions:

- Conducting PubMed searches of peer-reviewed scientific literature examining associations between TCE, PCE, benzene, and/or vinyl chloride and kidney cancer.

- Searching the Cochrane database for systematic reviews and meta-analyses.

- Reviewing and analyzing reports from national and international agencies, such as the International Agency for Research on Cancer (IARC), the Agency for Toxic Substances and Disease Registry (ATSDR), the National Toxicology Program (NTP), and the United States Environmental Protection Agency (EPA). This included a thorough review and evaluation of the studies cited within these reports.

To contextualize my findings, and to help with my analysis using a differential diagnosis, I evaluated these studies and reports using the Bradford Hill[3] viewpoints, including strength of association, consistency, specificity, temporality, dose-response, plausibility, coherence, experiment, and analogy. While not every Bradford Hill viewpoint needs to be satisfied to establish causality, they serve as a valuable framework for causation determinations. In this report, I assess both the presence and strength of each Bradford Hill viewpoint and compare their relative significance to formulate my causation opinions. This assists in terms of what weight to give the causal association of the water at Camp Lejeune and Ms. Tukes' kidney cancer.

Numerous epidemiological studies relevant to the association between TCE, PCE, Vinyl Chloride, and Benzene and kidney cancer are available. In both my general causation report and this report, I identify and examine meta-analyses, cohort studies, case-control studies, ecologic/water-contamination studies, reports from national and international agencies, and Camp Lejeune-specific studies related to contaminants and kidney cancer. It is standard practice among experts in my field to consider data from each of these categories when conducting a causality assessment.

I also assess any relevant toxicology and mechanistic data that provide additional relevant information to this causal analysis.

After concluding that the water at Camp Lejeune causes kidney cancer, including the four main toxins at issue, I then looked to the specifics of Ms. Tukes' case and whether the toxins she was exposed to caused her kidney cancer. I reviewed the medical records and deposition testimony provided. I examined other case-specific material that was provided to me regarding Ms. Tukes' history and exposure to the water at Camp Lejeune. I examined all of the potential risk factors for kidney cancer and analyzed whether those risk factors were relevant to Ms. Tukes. I then utilized a differential diagnosis approach for evaluating which of the risk factors were most relevant for Ms. Tukes' kidney cancer.

V. Discussion

A. Ms. Tukes History at Camp Lejeune and Medical History

1. Ms. Jacqueline Tukes completed a Short Form Complaint dated 11/06/2023 that noted she lived at Tarawa Terrace from December 1985 to January 1987 which was 13 months. In addition to this time on base, Ms. Tukes' testimony indicates she spent June 18, 1985 to July 18, 1985 in temporary housing on Hadnot Point where she was exposed to Benzene. Then from July 19 to December 17, 1985, she lived in Sherwood Mobile Home Park, a Trailer Park across the street from Hadnot Point. Ms. Tukes was exposed to the chemicals on Camp Lejeune while living in the trailer park for five months because she would often go to Camp Lejeune to shop and eat and do other activities as well. During this 5-month time she was exposed to the toxins in the water because she would drink the water while on base. In total, Ms. Tukes was exposed for approximately 19 months to solvents in the drinking water at Camp Lejeune. These solvents, even at lower levels during the time she was at Hadnot Point in 1985, acted to continually assault her kidneys with toxins that are known to cause kidney cancer. A longer time-period of exposure weakens the body's ability to fight the carcinogenic and mutagenic nature of these toxins.

2. Ms. Tukes deposition indicates that Ms. Jacqueline Tukes earned her certification as a Certified Nurse Assistant (CNA) and was working as a CNA prior to her cancer diagnosis. Ms. Tukes is married to a former Marine Corp service member, and they were exposed to the water at Camp Lejeune for approximately 19 months.

3. Ms. Tukes Deposition:

Ms. Tukes stated the following in her first deposition: In the morning, Ms. Tukes drank water with her breakfast and could drink two or three cups. (51:12-22). About four times a week she had mixed orange juice from concentrate with water and drank that with her breakfast. (59:1-14). Every once and a while, she boiled water from the faucet to have tea with breakfast, but she had tea more often in the afternoon. (51:25-52:7). Ms. Tukes mixed a pitcher of Kool-Aid almost every day and always kept a pitcher in the fridge, drinking much of the Kool-Aid herself. (52:8-15, 52:24-53:4). She made pitchers of lemonade four or five times a week and shared those pitchers with her husband and son. (53:7-18). With lunch, she drank at least two glasses of Kool-Aid, tea, or lemonade. (59:23-60:15). Some days, she would go to meet her husband for lunch on base, and on these days, she would drink a cup of water from the concession stand. (50:3-7). Ms. Tukes says that, since there was no bottled water back then, when she was on base, she drank from the water fountain. (49:14-19). When she was at the hospital during her pregnancy, she drank the water there. (49:20-22). She also drank water from the water fountains at the Commissary and the Exchange where she would do her shopping. (55:23-57:8). She remembers the Camp Lejeune water being the "nastiest tasting water." (65:24-66:9).

Ms. Tukes cooked three meals a day every day. (57:18-24). In the morning, she boiled smoked sausage with water and made grits with water for breakfast. (58:7-23). She also cooked with water every time she made dinner and commonly boiled her vegetables, rice, and meat in water for dinner. (60:16-61:6).

Ms. Tukes handwashed dishes after every meal in hot water that she left running the whole time. (61:13-15, 106:11-23). She did her own laundry at her house, possibly every day, and washed her husband's military uniforms. (50:9-25). She also mopped her floors with hot water with a type of mop from which she had to wring out the water. (62:14-22).

Because Ms. Tukes was not working while at Camp Lejeune, she spent most of her days on base. (39:10-15). During the summer, she would fill up a personal pool with hose water for her son to play in outside of their house. (40:3-6, 41:5-17). She also fished at the Camp Lejeune ponds and went to the Camp Lejeune beach where she drank water and rinsed sand off at the water fountains. (64:12-65:14).

Ms. Tukes' family shared one bathroom with no windows. Tukes Dep. 38:19-39:1. Ms. Tukes took hot showers for up to 15 to 20 minutes twice a day, replacing one of the showers with a bath about twice a week. Tukes Dep. 54:2-21, 55:1-7. She also brushed her teeth up to six or seven times a day with lukewarm water. Tukes Dep. 107:4-13, 20-25.

This testimony from Ms. Tukes evidences the substantial nature of her exposure. She was continuously being exposed to the toxins in the water at Camp Lejeune during her day-to-day activities, indicating a substantial frequency of exposure to these chemicals. She was exposed to the toxins for an extended period, namely, 6 months at Hadnot Point during 1985 and thirteen months at Tarawa Terrace from December of 1985 to January of 1987. The intensity of her exposure was significant given the levels of the toxins in the water and how often she was exposed on a daily basis.

4. Ms. Jacqueline Tukes was a patient of Dr. David M Stevens, NH Camp Lejeune and she was being worked up for a cause of her hypertension when a CT renal arteriogram noted a 1.6-centimeter mass in the right kidney that was suspicious for carcinoma. Ms. Tukes had a partial nephrectomy on the right on 8/20/2010 and she had partial nephrectomies on the left in 2018 and 2019. Ms. Tukes subsequently had a right total nephrectomy in 2022 and a left total nephrectomy in 2023. Ms. Tukes started on dialysis in 2023 right after her surgery and successfully had a kidney transplant in July 2024. This is consistent with her medical records.

a. Right Kidney: Ms. Tukes was first referred for evaluation to determine the cause of her hypertension by Dr. David M Stevens, NH Camp Lejeune. During the work-up, she had a CT renal arteriogram on 6/4/2010 and Dr. Gregory Hall prepared a CT Report that showed a 1.6 cm right renal mass in the right lower pole of the kidney and a simple cyst in the left kidney. Dr. David Stevens referred Ms. Tukes to urological surgery on 6/18/2010. Dr. Mathew Nielsen, MD wrote a surgical procedure note dated 8/21/2010 and indicated Ms. Tukes had a right partial nephrectomy on 8/20/2010 for renal cell carcinoma. Dr. Andrew Larrimore completed a surgical pathology report dated 8/23/2010 and indicated that Ms. Tukes had a histological cell type of renal cell carcinoma with clear cell subtype.

Ms. Tukes had a right total nephrectomy on 5/23/2022 following a recurrent kidney cancer in the right kidney. The surgical pathology report by Dr. Nathan Pulkingham dated 5/23/2022 indicated there were several foci of cancer throughout the kidney that were cancerous and necessitated removal. The tumor is a papillary type but demonstrates fairly diffuse clear-cell changes. Dr.

Pulkingham felt that the tumor was a clear-cell papillary variant of renal cell carcinoma.

b. Left Kidney: Ms. Tukes had a left partial nephrectomy in 2018 and again in 2019 for kidney cancer. Dr. Roc McCarthy wrote a Medical Encounter note dated 5/9/2018 and indicated that Ms. Tukes had three small cancer foci in the left kidney and the surgical pathology report indicated that she had clear cell papillary renal cell carcinoma. Dr. McCarthy wrote a Medical Encounter note dated 4/3/2019 and indicated that Ms. Tukes underwent a left partial nephrectomy for a small recurrent tumor on 3/14/2019 and the surgical pathology report indicated that the tumor cell type was a clear cell renal cell carcinoma similar to the other kidney cancers removed on the left and right. Ms. Tukes had a follow-up CT Screening examination on 1/25/2023 and Dr. McCarthy saw Ms. Tukes on 2/7/2023 noting there was a 1.4 cm left renal mass and made plans for a left total nephrectomy on June 12, 2023. Dr. McCarthy indicated that Ms. Tukes left nephrectomy also was positive for a renal cell carcinoma.

c. Ms. Tukes had a dialysis catheter inserted in her radial forearm but there was a problem with functionality so a central catheter was inserted into her chest and the central catheter was utilized while Ms. Tukes was on dialysis.

d. Ms. Tukes was worked up for a Kidney transplant and she was fortunate to be placed on the list awaiting a donor and it was not long after she received a transplanted kidney.

5. Dr. Nagesh Jayaram (oncologist) in his deposition stated that because of the apparent family history of kidney cancer, they did genetic testing, but the results indicated there was no genetic component to Ms. Tukes kidney cancer. Dr. Jayaram indicated that when genetic testing is negative, this rules out a genetic predisposition for kidney cancer. Further, Dr. Jayaram indicated that he thought Ms. Tukes kidney cancer was caused by her exposure to contaminated drinking water at Camp Lejeune. When asked why he believed the water at Camp Lejeune caused Ms. Tuke's kidney cancer, Dr. Jayaram indicated that he was familiar with the medical literature that shows a strong link between solvent exposure at Camp Lejeune and the development of kidney cancer. Further, Dr. Jayaram stated he was treating or had treated five other people who had been exposed to contaminated drinking water at Camp Lejeune and had subsequently developed kidney cancer.

6. Katherine Garbarini, MS, Certified Genetics Counselor (CGC), gave a deposition on June 20, 2024, and stated she was a master's level trained and board-certified geneticist who worked for UNC Health as a genetic counselor in 2018 when Ms. Tukes had her genetic testing performed.

a. Ms. Garbarini ordered genetics testing for Ms. Tukes on 8/8/2018 after doing an assessment of her family history and review of the medical record. Her note dated 8/12/2018 indicated Ms. Tukes is a 53-year-old African American female who has a personal history of renal cell cancer. Ms. Garbarini noted that Ms. Tukes mother had metastatic disease at the time of diagnosis of her cancer and it was unclear whether the kidney cancer was the primary cancer. Ms. Garbarini wrote, after doing a pedigree of Ms. Tukes maternal and paternal family, that there is no clear family history of kidney cancer to suggest a particular inherited pre-disposition for renal cell cancer.

b. Ms. Garbarini wrote a letter to Ms. Tukes dated 11/26/2018 forwarding the results of her genetic testing and advised that the test results were negative for genetic changes known to be associated with hereditary renal cell cancer. She wrote that there were no pathogenic mutations in any of the genes associated with kidney cancer. She stated that it is still possible there could be a mutation that current technology cannot detect, but the chance of that is small. Ms. Garbarini stated in her deposition that current testing can detect 99% of the genetic changes associated with kidney cancer risk and so that served as her basis that the risk was very small. Ms. Garbarini wrote in her letter that there were two variants of uncertain significance for the PMS2 gene and the SMARCA4 gene. Neither of these two variants is associated with kidney cancer.[4,5]

c. Thomas Winder, PhD, Geneticist, wrote a laboratory report dated 4/10/2020 that was an addendum to the prior genetic testing for Ms. Tukes. He indicated there was a change in the classification of the results of the SMARCA4 gene from uncertain significance to likely benign significance. Ms. Garbarini wrote Ms. Tukes a letter on 4/10/2020 providing her with the updated addendum to the laboratory results and providing her with the updated results. The genetic testing laboratory results were amended for the PMS2 gene on 9/6/2022 and the results changed from indeterminant to likely benign. Julianne O'Daniel, MS, CGC, Certified Genetic Counselor UNC wrote a letter to Ms. Tukes on 9/13/2022 and advised her of the updated results. She goes on to say this is good news and confirms the initial suspicion that these indeterminant results are natural variation among people and have nothing to do with kidney cancer risk and that no further testing was indicated.

d. Ms. Garbarini was asked during her deposition whether someone with negative genetic testing and a family history of kidney cancer was still at an increased risk of developing kidney cancer. Ms. Garbarini stated the chance Ms. Tukes has a predisposition to kidney cancer for hereditary reasons is very small. Dr. Jayaram, clinical oncologist, testified that when genetic testing is negative in his practice, this rules out a genetic predisposition for kidney cancer. Dr. McCarthy testified to the same.

7. Dr. Irvine Allen, PhD, wrote an analysis of the two genetic variants following Ms. Tukes genetics testing. In his opinion, the cumulative findings from the genetic testing demonstrates that it is more likely than not that Ms. Tukes' RCC is not directly caused by an inherited or congenital genetic mutation. This is based on the genetic screening panel results from Ms. Tukes testing at UNC Chapel Hill. Dr. Allen stated that Ms. Tukes was also negative for other genetic changes that can lead to diseases evaluated by this genetic screening panel, which includes diseases such as Von Hipple Lidow that are known to be causative for RCC. Ms. Tukes had variants of unknown significance in the PMS2 gene and the SMARCA4 gene that are "likely benign" and do not directly cause RCC. The PMS2 gene alteration is associated with a mismatch repair deficiency and results in an increase in cancer risk when a person is exposed to carcinogens. So while Ms. Tukes had heterozygous (changes in one of the two copies of each gene) for SMS2 and SMARCA4, these changes in the gene do not increase the occurrence of kidney cancer; rather, they require carcinogen exposure to initiate the cancer development. Thus, according to Dr. Allen, it is as likely as not that these mutations contribute to an increase in the susceptibility of Ms. Tukes' to overall cancer development following exposure to carcinogens

and at doses that would not ordinarily lead to increased tumor development in individuals without these mutations. In short, Dr. Allen's report states that Ms. Tukes was, given her congenital genetic alterations, more susceptible to cancer when exposed to lower doses of carcinogens than she otherwise would have been without the genetic alterations. This is significant in the assessment of Ms. Tukes' case. It means the chemicals in the water Ms. Tukes was exposed to had a greater impact than the same level of chemicals would have had on another person or on Ms. Tukes if she did not have these altered genes.

B. Chemicals in the Water at Camp Lejeune Associated with Kidney Cancer Causation

1. The Tarawa Terrace treatment plant provided drinking water to the housing area and key samples were taken from February 1985 onward. The water in the Tarawa Terrance Water Treatment Plant was contaminated with TCE, PCE, and VC. PCE levels peaked at 215 ppb in February 1985. Water modeling[6,7] for Tarawa Terrace using the Techflow Model showed a range of PCE concentrations from 2.10 ppb in July of 1985 to 8.28 ppb in January 1987. The below chart depicts the levels of toxins in the water at Tarawa Terrace during the time Ms. Tukes lived there:

Exposure Dates	TCE (ug/l-M)	PCE (ug/l-M)(TechFlowMP Model)	PCE (ug/l-M)(MT3DMS Model)	VC (ug/l-M)	BZ (ug/l-M)
12/18/1985-12/31/1985	0.16	3.58	8.27	0.76	0.00
1/1/1986-1/31/1986	0.18	3.95	8.85	0.82	0.00
2/1/1986-2/28/1986	0.19	4.24	9.42	0.83	0.00
3/1/1986-3/31/1986	0.24	5.40	12.14	1.01	0.00
4/1/1986-4/30/1986	0.22	4.93	10.83	0.89	0.00
5/1/1986-5/31/1986	0.23	5.25	11.56	0.91	0.00
6/1/1986-6/30/1986	0.25	5.61	12.28	0.92	0.00
7/1/1986-7/31/1986	0.26	5.97	13.06	0.94	0.00
8/1/1986-8/31/1986	0.28	6.36	13.84	0.96	0.00
9/1/1986-9/301986	0.30	6.75	14.61	0.97	0.00
10/1/1986-10/31/1986	0.31	7.12	15.42	0.99	0.00

11/1/1986-11/30/1986	0.33	7.52	16.21	1.00	0.00
12/1/1986-12/31/1986	0.34	7.89	17.03	1.01	0.00
1/1/1987-1/8/1987	0.36	8.28	17.85	1.03	0.00
Totals	**3.65**	**82.85**	**181.37**	**13.04**	**-**

2. The current U.S. EPA maximum contaminant levels (MCLs) for TCE, PCE, and benzene are 5 ppb, established in 1989 for TCE and benzene and in 1992 for PCE. The MCL for Vinyl Chloride is 2ppb.[2]

3. Ms. Tukes was present at Hadnot Point from June to December of 1985 as she lived there for a month and also would frequently visit when she lived off base. She was exposed to Benzene in the water during these times from. From December 1985 through January 1987, Ms. Tukes was exposed primarily to the concentrations above from TT during the time she lived there. She would sometimes go to Hadnot Point for lunch or to shop and would be exposed to the Hadnot Point concentrations of the chemicals during those visits.

C. Ms. Tukes' Genetic Susceptibility to Exposure to Toxins at Low Levels

Dr. Allen's report details the significance of the genetic testing performed on Ms. Tukes at UNC Chapel Hill in 2018.

First, based on Dr. Allen's report, it is clear Ms. Tukes' kidney cancer was not hereditary in nature. This is consistent with the testimony of Ms. Garbarini, the records from UNC Chapel Hill and Mrs. Tukes' treating physicians deposed in the case.

Second, Dr. Allen's report states that the two genes found to be altered in Ms. Tukes' genetic profile made her more susceptible to exposures to carcinogens, such as TCE, PCE, VC and Benzene, at lower levels then she otherwise would have been without these genetic mutations.

I independently reviewed Ms. Tukes exposure to the toxins in the water at Camp Lejeune, her medical history and all of the other documents noted in this report and came to the conclusion Ms. Tukes kidney cancer was caused by the water at Camp Lejeune without Dr. Allen's opinions. However, knowing Ms. Tukes' was more susceptible to environmental carcinogens, such as her exposure at Camp Lejeune, makes my opinion that much stronger.

D. Ms. Tukes' Exposure to the Water at Camp Lejeune was Substantial and Exceeded a De Minimis Level

Ms. Tukes lived at Tarawa Terrace for 13 months. Ms. Tukes stated in her deposition she drank at least 2 glasses of water with meals. Ms. Tukes testified that when she went for walks and attended to her son playing outside, and when she walked to the commissary and to the base exchange, she would get thirsty and increase her water consumption quite a bit.

Ms. Tukes had six months of Benzene exposure at Hadnot Point when she stayed in temporary housing for the month of June 1985, and from July to December1985 when she shopped, ate and socialized on Hadnot Point during her stay in Sherwood Trailer Park.

Ms. Tukes was exposed daily through all three routes of potential exposure: ingestion, inhalation and dermal exposures. I will not repeat the deposition testimony of Ms. Tukes, other than to say that her exposure to the water at Camp Lejeune was substantial in terms of the amount of the exposure (see prior total chemicals exposed), the duration of the exposure (19 month time period), the frequency of the exposure (see description of how often she was exposed to the chemicals daily) and the intensity of the exposures (see levels in the water at Camp Lejeune during the time period Ms. Tukes was present). The intensity of the exposure is also derived from the epidemiology and other studies showing the levels at which a causal relationship existed between the toxins at issue in this case and kidney cancer.

I have made my determination that Ms. Tukes' exposure was substantial based on the above information and details. However, I have also reviewed the calculated cumulative ingestion charts created by Plaintiff's expert Kelly Reynolds. The extent of Ms. Tukes' ingestion is seen in these charts:

		Chart 1: 1L	Chart 2: ATSDR CTE	Chart 3: ATSDR RME	Chart 4: Deposition Estimates
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)
TCE	3.65	100	107	271	259
PCE (ug/l-M)(TechFlow MP Model)	82.85	2,280	2,437	6,142	5,875
PCE (ug/l-M)(MT3DMS Model)	181.37	4,989	5,335	13,443	12,858
VC	13.04	361	386	974	931
BZ (only at HP)	60.00	678	373	939	898

Using the TechFlow model and assuming the ingestion statistics in Ms. Tukes' deposition, Ms. Tukes was exposed to 5,875 ppb of PCE through ingestion alone. This is a substantial exposure.

Ms. Tukes would have also had exposure through inhalation and dermal routes of absorption. Therefore, the numbers in these charts are, in reality, even higher.

As seen in the charts, not only was Ms. Tukes exposed to substantial quantities of PCE, she was also exposed to TCE, VC and Benzene. These chemicals are all known carcinogens linked to kidney cancer. The TCE, VC and Benzene would have acted additively or synergistically with the PCE to cause Ms. Tukes' kidney cancer.

When the numbers above are viewed in the context of Dr. Allen's report, and the understanding Ms. Tukes was more susceptible to exposure to environmental carcinogens at lower levels, the numbers detailed in the charts should be interpreted as even more dangerous and substantial. The fact Ms. Tukes was more susceptible to environmental carcinogens, like the ones listed in the charts, makes the impact of each exposure in the chart greater. The fact that Ms. Tukes' was more susceptible to these toxins at lower levels makes the causal relationship between her exposure and her kidney cancer even stronger.

E. Non-Camp Lejeune Personal Risk Factor Analysis for Ms. Tukes' Kidney Cancer

1. Ms. Tukes has few personal risk factors for developing kidney cancer. She was 45 years old when she developed kidney cancer, so she was not at increased risk due to her age.

2. Ms. Tukes is African American, so she was at a small increased risk of kidney cancer due to her ethnicity compared to Caucasian women in the general population.[8]

3. Ms. Tukes was slightly overweight during the time of her first diagnosis of kidney cancer. Obesity can be a cause of kidney tumors. Beebe-Dimmer JL et. (2012) examined BMI risk for kidney cancer, and after adjustment for demographic variables—including age, education, hypertension status, family history of renal cancer, and smoking history–noted that African American women like Ms. Tukes were found to have lower overall risk for kidney cancer based on BMI.[9] They noted for BMI within the 25-29 category, the risk ratio was only 0.8 (95% CI: 0.4 to 1.9) and for BMIs in the 30 to 34.9 (obese category), there was a risk ratio of 0.9 (95%CI: 0.4 to 2.1). Thus, ethnic and gender differences markedly reduced Ms. Tukes risk related to her being overweight and having a lower risk of kidney cancer according to this study. Ms. Tukes risk from being overweight was not significantly elevated. Further, Ms. Tukes' BMI fluctuated between 26.96 and 31.98. These are not significantly elevated BMIs that would materially alter the differential diagnosis in this case. Given that Ms. Tukes' BMI changed over the course of the 13 years she was developing kidney tumors and having them removed, it is unlikely that her BMI was related to her development of kidney cancer. BMI is a known risk factor for kidney cancer, but is usually thought of as a softer risk factor, especially when there is another known cause of kidney cancer in the differential.

4. Ms. Tukes is a female, so she was not at increased risk due to her gender.

5. Ms. Tukes was a non-smoker, and she did not drink alcohol, so she was not at increased risk due to lifestyle issues. Concurrent cigarette smoking increases the risk of renal failure and kidney cancer, however, Ms. Tukes was a lifetime non-smoker, so she was not at increased risk of

kidney cancer due to smoking.

6. Ms. Tukes was diagnosed with hypertension around time she was diagnosed with kidney cancer. She had heightened blood pressure for a time before her original diagnosis in 2010. Therefore, Ms. Tukes was at slightly elevated risk due to her having hypertension. However, Ms. Tukes' blood pressure was ultimately controlled over the course of the 13 years Ms. Tukes continued developing kidney tumors (from 2010 – 2023). For a significant part of that time her blood pressure was within normal limits. This makes it less likely that her kidney tumors were being caused by hypertension. Given the fact Ms. Tukes hypertension was well controlled and she still continued to develop kidney cancer, it is unlikely that hypertension was responsible for causing her kidney cancers.

7. People with a strong family history have higher chance of developing kidney cancer. This risk is highest for people who have a brother or sister with the cancer. According to the medical records for Ms. Tukes, it was never confirmed that the cancer her mother had was a primary kidney cancer. What was allegedly known at the time was that there was cancer in her kidney, but Ms. Tukes was not aware there was any pathology showing it was primary kidney cancer as opposed to a metastasis. Ms. Tukes tested negative for genetic changes in her DNA that would represent a hereditary renal cell carcinoma. Ms. Garbarini, the Genetics Counselor at UNC, stated in her encounter note on 8/8/2018 there was not a clear pattern for a family history of kidney cancer for Ms. Tukes. Further, Dr. Allen's report makes clear that Ms. Tukes' kidney cancer was not hereditary.

8. Many studies have suggested that workplace exposure to substances can increase the risk for kidney cancer. Some of these substances are cadmium, herbicides, organic solvents and lead. Ms. Tukes did not have a history of workplace exposures to the hazards listed above, other than Camp Lejeune water, so she was not at increased risk of kidney cancer due to workplace hazards.

9. The medical literature on Crohn's disease indicates that individuals who have longstanding crohn's disease are at increased risk of developing kidney cancer. Ms. Tukes did not have a history of crohn's disease, nor did she have a family history, so she was not at increased risk.

10. Rhabdomyolysis is not a risk factor for kidney cancer. The Defendants assert that Ms. Tukes had a genetic predisposition for Rhabdomyolysis but there is no association between rhabdomyolysis and kidney cancer. Ms. Tukes was at increased risk for rhabdomyolysis due to the intense summer heat in North Carolina where she lived and her gender. There is no association between rhabdomyolysis and an increased risk of kidney cancer in the medical literature.

F. Camp Lejeune Water Contamination Related Risks

1. Total Volatile Organic Compounds Exposure Risk and Kidney Cancer

a. The chemicals in the water at Camp Lejeune, namely TCE, PCE, VC and Benzene, in combination, have been shown to be very dangerous to humans and create an increased risk of kidney cancer.

b. Bove et. al. (2014a)[10] and (2014b)[11] conducted retrospective cohort mortality studies of Marines/Navy personnel and civilian workers at Camp Lejeune. Bove et. al. (2014a) reported in Table 6 that TVOC exposures between 1 and 4600 ug/L-months fall into the low exposure category for kidney cancer risk. Ms. Tukes' TVOC exposure fit into the low exposure category which had a RR of 1.42.[10]

c. The Bove (2024)[12] cancer mortality study compared Marines and Navy personnel to Camp Pendleton personnel and noted a significantly increased risk of death from kidney cancer with a SMR of 1.22 (95% CI: 1.03, 1.45) (Table S2). The civilian worker analysis noted an elevated SMR for kidney cancer death of 1.49 (95% CI: 0.76, 2.92).[12] The study also analyzed kidney cancer risk relative to exposure duration, measured by time spent on base.[12] For personnel with low duration of exposure (1–2 quarters), the risk ratio was 1.33 (95% CI: 0.95-1.86) (Table S6). For medium-term exposure (2-7 quarters), the risk ratio was 1.23 (95% CI: 0.88–1.72) (Table S.6).[12] These findings suggest that even the short exposure durations on base during the 1972–1985 period was linked to a higher risk of death due to kidney cancer. Ms. Tukes was not on base during that time but she was on base being exposed to these chemicals for over 7 quarters of time. She was also more susceptible to these chemicals because of her genetic alterations. This study, therefore, provides very relevant information to this analyses.

d. Bove et al. (2024)[13] conducted a cancer incidence study on Camp Lejeune personnel that noted Camp Lejeune Marines/Navy personnel had an elevated adjusted standardized incidence ratio (SIR) (see Table 3) for renal cell carcinoma, NOS of 1.12 (95% CI: 0.94 to 1.34).[13] These findings suggest that individuals exposed to contaminated water at Camp Lejeune during 1975–1985 were at above equipoise elevated risk of developing kidney cancer.[13] The study also analyzed kidney cancer risk relative to exposure duration, measured by time spent on base. The category for low duration was 1-6 quarters.[13] The medium duration of exposure was 7-10 quarters on base, and the high duration of exposure was over 10 quarters.[13] Ms. Tukes was not on base during that time but she was on base being exposed to these chemicals for over 7 quarters of time. She was also more susceptible to these chemicals because of her genetic alterations. This study, therefore, provides very relevant information to this analyses.

2. Perchloroethylene Exposure and Kidney Cancer Risk

a. Bove et. al. (2014a)[10] noted that PCE increased the kidney cancer risk. In Bove's assessment, the medium exposure category cutoff was 155 ug/liter-months.[10] Ms. Tukes exposure to PCE was 82.85 ug/L-months which falls in the middle of the low exposure category.[10] Using Bove 2014a's table 7, the low PCE exposure category had an odds ratio of 1.40 (95% CI: 0.54 to 3.58).[10] Ms. Tukes had a markedly elevated risk of kidney cancer due to her PCE exposure based on these results.

b. Aschengrau A, Ozonoff D, Paul C, et al.[14] noted an elevated kidney cancer risk with total PCE exposure in the range of 27-44 mg (90^{th} percentile) in water entering a household at Cape Cod. Ms. Tukes' total cumulative PCE exposure would have been similar to the levels found by

Aschengrau that showed increased risk ratios for kidney cancer in that study when you take into account her exposure through ingestion, inhalation and dermal exposure routes.[14]

c. ATSDR (2018)[15] performed a morbidity study of Marines and Navy personnel who were assigned to Camp Lejeune and compared the results with personnel stationed at Camp Pendleton. The study found that there was an increased risk for cancers, including kidney cancer, as a result of exposure to water contaminated with perchloroethylene (PCE).[15] The morbidity study of Marines and Navy personnel stationed at Camp Lejeune, found an elevated Odds Ratio for kidney cancer of 1.31 (95% CI: 0.86, 1.99) compared to Camp Pendleton.[15] For high residential exposures (≥90th percentile) to PCE, the OR was even higher at 1.79 (95% CI: 1.02, 3.12).[15] The Odds Ratio for high PCE exposure was 2.01 (95% CI: 1.29, 3.13) and a monotonic exposure-response relationship was observed.[15] The OR for Civilian employees at Camp Lejeune as compared with those at Camp Pendleton was 1.52 (95% CI: 0.69, 3.35) for kidney cancer.[15] The OR for high workplace exposures (≥90th percentile) to PCE among civilian employees at Camp Lejeune compared with those at Camp Pendleton was 13.92 (95% CI: 5.09, 38.10).[15] For the analysis internal to Camp Lejeune civilian employees, the OR for high PCE exposure was 41.5 (95% CI: 10.2, 169.23), and a monotonic exposure-response relationship was observed.[15] These findings support Ms. Tukes' contention that her solvent exposure at Camp Lejeune, particularly PCE, caused her kidney cancer. While Mrs. Tukes' levels may not have been as high as some of the people in this study, the study shows PCE causes kidney cancer and we know Mrs. Tukes was more susceptible to exposures at lower levels given her genetic alterations.

d. There is evidence of a causal relationship between PCE exposure and kidney cancer in Mandel et. al. (1995)[16] who found a relative risk of 1.4 (95% CI: 1.1-1.7) in their study of drycleaners. Anttila et. al. (1995)[17] found an increased cancer risk of 1.82 (95% CI: 0.22-6.56).[17] Calvert GM et al. (2011)[18] found in a cohort of dry cleaners an elevated risk in PCE exposed workers who had an elevated SMR of 1.35 (0.16-4.89). Callahan et. al. (2019)[19] performed a cohort study of drycleaners and noted the kidney cancer deaths were elevated with an SMR of 1.1 (95% CI:0.6-1.9). They found an exposure–response relationship for kidney cancer from medium exposure with a risk ratio of 4.1 (95% CI = 0.7, 22.5) to high exposure with a risk ratio of 24.4 (95% CI: 2.9, 201.6) (Ptrend = 0.004) in a 20-year lagged analysis.[19] Ruder et. al. (2001)[20] conducted a cohort study of dry-cleaners and found for PCE exposed workers, the SMR was 1.73 (95% CI: 0.21-6.25) for kidney cancer. Christensen et al. (2013)[21] conducted a case-control study and found for any exposure a 1.6-fold increased risk (95% CI: 0.3-9.4) and the risk ratio for >90% cumulative exposure was 3.1-fold increased risk (95%CI: 0.4-24) of kidney cancer. Karami S. et. al. (2012)[22] conducted a case control study of 1217 cases and 1235 controls of workers in the dry-cleaning industry, primarily exposed to PCE. They found an elevated risk of renal cell carcinoma with an odds ratio of 2.0 (95% CI = 0.9-4.4).[22] When the duration of exposure was considered, exposure over 5 years increased risk 2.5-fold (95%CI: 0.4-14.4).[22] Delahunt et al. (1995)[23] conducted a case-control study of New Zealand dry cleaning workers and noted an increase in kidney cancer of 1.92 (95% CI: 0.27-13.89).

e. It is important to note that the EPA recently enacted a rule banning PCE products and uses of PCE in the workplace. EPA's rule was enacted, in part, due to their statements that PCE was known to cause kidney cancer and also cause kidney cancer at low levels.

f. In summary, the epidemiological evidence of an association between Ms. Tukes' PCE exposure and kidney cancer exists and is supported by different epidemiological studies, animal data and mechanistic effects. In my opinion, the analysis of the currently assembled evidence, using the Bradford Hill framework, makes it more likely than not that Ms. Tukes PCE exposure is a cause of her kidney cancer (and therefore also exceeds the "standard of at least as likely as not" prescribed by the Camp Lejeune Justice Act.). This is especially true when it is taken into account that Ms. Tukes' had genetic alterations making her susceptible to cancer at low levels of exposure.

3. TCE Exposure and Kidney Cancer Risk

a. Ms. Tukes had exposure to TCE at Tarawa Terrace for 13 months. The levels of TCE at Tarawa Terrace during that time were less than 1 ug/L-month.

b. As stated in my general causation report, it is clear that TCE is a cause of kidney cancer. I incorporate my general causation report statements as to the causal relationship between TCE and kidney cancer into this report.

c. Ms. Tukes' exposure to TCE, although minimal, would have contributed additively or synergistically to her risk of kidney cancer.

4. Vinyl Chloride Exposure and Kidney Cancer Risk

a. As stated in my general causation report, VC is a cause of kidney cancer. I incorporate my general causation report statements as to the causal relationship between VC and kidney cancer into this report.

b. This VC exposure would have contributed additively or synergistically to her risk of kidney cancer.

5. Benzene Exposure at Hadnot Point, Camp Lejeune and Kidney Cancer Risk

a. Ms. Tukes had Benzene exposure at Hadnot Point when she stayed in temporary housing on Hadnot Point for the month of June 1985. She also had exposure to benzene from July to December 1985 when she would visit the base. She was exposed when she would have lunch with her husband at Hadnot Point when she was living at Tarawa Terrace as well. The Benzene concentration at the time Ms. Tukes was exposed to water at Hadnot Point ranged from 2 to 4 ug/L and the average concentration was 3 ug/L. She was exposed to this water for the full 19 months and her cumulative exposure to Benzene was 60 ug/L-months.

b. This exposure would have contributed additively or synergistically to her risk of kidney cancer.

c. Bove 2014a used a threshold between low and medium exposure of 45 ppb/L-months. Thuse, given Mrs. Tukes prolonged exposure over time at Hadnot Point to Benzene, her exposure given

the concentrations in the water at those times would place her in the medium category.

VI. Additive Effects of TCE, PCE, Benzene and Vinyl Chloride on Kidney Cancer Risk

When considering the carcinogenic potential of simultaneous exposure to two or more known carcinogens, one may reasonably and scientifically anticipate that the carcinogens increase risk of cancer in an additive fashion-which is typically the default assumption when regulators assess chemicals that act through a common mode of action. Adding the effects of TCE, PCE, Benzene and Vinyl chloride in Kidney cancer risk would be a reasonable approach to assess the total risk kidney cancer, since these chemicals have similar breakdown products and have similar mechanisms of action and have been shown to be genotoxic by causing damage to DNA and to cause chromosomal malformations. As such, in my opinion, it is reasonable to apply an additive approach to carcinogens with a common mode of action and conclude that the combined risk is more likely than not to be at least additive.

VII. Differential Diagnosis Methodology for the Risk Factor Analysis to Determine the Etiology of Ms. Tukes Kidney Cancer

There were a number of sources used to develop a list of personal risk factors for kidney cancer. For example, the Mayo Clinic Fact Sheet on Kidney Cancer[24] was reviewed. Each of these personal risk factors was considered as potentially contributing to the cause of Ms. Tukes kidney cancer. It is common practice in environmental medicine to conduct a differential diagnosis process where we proceed step by step in considering each potential risk factor to see if the plaintiff had one of the listed risk factors for kidney cancer. If the plaintiff does have the risk factor, an analysis of the relative contribution of this risk factor to the cause of the individual's kidney cancer is conducted. Then, once all the personal risk factors are considered, a weighing of all the personal risks is done by adding the individual risks together to come up with a summative personal risk estimate. The individual's environmental risks are considered in a similar fashion and are summed to develop a summative environmental risk estimate. Finally, the personal summative risk estimate is compared with the summative environmental risk estimate. A weighing of the relative personal risks and environmental risks provides a good indicator whether the kidney cancer was due to environmental risks or personal risks.

The personal risk factors for kidney cancer are well documented in the medical literature and are noted above in this report. As noted previously, Ms. Tukes had virtually no other significant risk factor for the development of her kidney cancer other than her exposure to the toxins at Camp Lejeune. For purposes of the differential diagnosis, these risk factors will be briefly stated again below with an analysis of why they are not significant. Following this, an analysis of all of Ms. Tukes environmental risks are summarized from the sections above and the risks are noted together to develop a summative environmental risk estimate. Finally, the personal risks will be analyzed and compared with the summative environmental risk estimate to see if personal risks or environmental risks outweigh the other.

Ms. Tukes was slightly overweight when she was diagnosed with Kidney cancer. She would have been at slightly increased risk due to her weight. However, this is usually not a significant risk factor and Ms. Tukes was not significantly overweight. Ms. Tukes weight fluctuated over

time, yet she continued to be diagnosed with new primary RCC tumors. This suggests her slightly increased weight prior to and following her first diagnosis of kidney cancer in 2010 was not relevant as a cause of that cancer, otherwise, she would have had a reduction in the development of cancer when her BMI or weight changed.

Ms. Tukes was a lifetime non-smoker and non-drinker, so she was not at increased risk of kidney cancer due to these risk factors. Ms. Tukes was 45 in 2010 when she was first diagnosed with kidney cancer, so she was not at increased risk of developing kidney cancer due to her age. Kidney cancer is about twice as common in men, so Ms. Tukes was not at increased risk due to her gender. Ms. Tukes is African American, so she had a slightly increased risk of kidney cancer due to her ethnicity. This represents a low risk.

Ms. Tukes did have high blood pressure around the time she was diagnosed with her first kidney cancer. This is usually not a very significant risk factor, even though there is some association between hypertension and kidney cancer. In particular, for Ms. Tukes, it is unlikely her high blood pressure caused or contributed to the development of her kidney cancer. During the time period of 2010 through 2023, there were significant periods of time when her hypertension was within normal limits, but she continued to develop new primary RCC. This suggests hypertension was not likely the cause of her cancers, otherwise, she would have likely stopped developing kidney cancers if that was truly the cause.

Ms. Tukes did not have a confirmed family history of Kidney cancer. It was not confirmed her mother had a primary kidney cancer rather than metastatic disease. Ms. Tukes tested negative for a genetic predisposition for kidney cancer. This makes it very highly unlikely that her kidney cancer was hereditary. Mrs. Tukes' treating physicians deposed in the case seem to agree with this point.

Ms. Tukes environmental exposure risk outweighed the very little to no risk posed by her personal risk factors. In my general causation report I cite a number of levels at which the epidemiology literature indicates that the toxins in the water at Camp Lejeune were known to cause kidney cancer. Ms. Tukes met several of these levels and had similar exposure to others. Therefore, the risk of kidney cancer due to her exposure to these toxins, at the levels she was exposed to, was substantial. This is especially true given Ms. Tukes' genetic susceptibility to environmental exposures of known carcinogens.

Ms. Tukes exposure to PCE at Tarawa Terrace for 13 months increased her risk of Kidney cancer substantially and significantly. Her exposure to additional levels of TCE, VC and Benzene, both during those 13 months and also prior that at Hadnot Point, likely contributed to this exposure additively or synergistically.

Given there are no other significant potential risk factors for Ms. Tukes development of kidney cancer, other than a very slightly elevated risk due to her hypertension and weight, it is clear Ms. Tukes' exposure to the toxins at Camp Lejeune was a substantial contributing factor and cause of her kidney cancer. I am able to conclude based on this differential diagnosis that Ms. Tukes' kidney cancer was more likely than not caused by exposure to the water at Camp Lejeune.

The report of Dr. Allen discussed the role of Ms. Tukes two genetic variants PMS2 and SMARCA4 and explained how these variants made her more susceptible to cancer following carcinogen exposure. Neither of these two variants are related to her development of her kidney cancer. Ms. Tukes genetic variants do increase her susceptibility for developing other cancers due to the loss of transcription control and reduced DNA repair capability. Dr. Allen noted that these genetic abnormalities found on Ms. Tukes' testing make it at least as likely as not that Ms. Tukes was more susceptible to exposure to these toxins at lower levels than what would normally cause cancer. This adds weight and strength to my conclusion that the Camp Lejeune water was the cause of Ms. Tukes' kidney cancer.

VIII. Response to Defendants Supplemental Answers to Interrogatories

The defendant notes that alternative explanations exist for the cause of the plaintiffs kidney cancer to include that she had a family history of kidney cancer, e.g., Ms. Tukes' mother was diagnosed with renal cancer and a cousin died of kidney cancer. There are two points to be made that serve to refute this contention. First, Ms. Tukes mother was not confirmed to have kidney cancer of primary origin. Second, the medical records and genetic testing from UNC, the testimony from Ms. Garbarini and the report of Dr. Allen, all support that Ms. Tukes' cancer was not hereditary.

The Defendant stated that the Plaintiff's exposure to water at Camp Lejeune may not have been significant enough to cause the alleged illness or injury. The combined additive TVOCs, TCE, PCE, VC, and Benzene amount to a substantial and significant exposure. Ms. Tukes had few personal risk factors for developing kidney cancer, so it is more likely than not that Ms. Tukes kidney cancer risk was caused by or due to her solvent exposures at Camp Lejeune. This is especially true given that according to Dr. Allen's report, Ms. Tukes was more susceptible to exposure to these toxins at lower levels.

The Defendant argued that the length of time (latency period) between exposure to the water at Camp Lejeune and the onset of the Plaintiff's illness or injury may indicate an alternative cause or that the cause of the Plaintiff's illness or injury is idiopathic. Ms. Tukes kidney cancer developed 23 years after exposure at Camp Lejeune ended. There is medical literature on latency periods relating to solvent exposure and kidney cancer noting that latency periods generally exceed 20 years.[25] So, Ms. Tukes kidney cancer development is well within the normal range of latency period for kidney cancer. The treating oncologist stated that Ms. Tukes kidney cancer was caused by Ms. Tukes exposure to contaminated water at Camp Lejeune.

IX. Summary Medical Opinion for TCE, PCE, Benzene and Vinyl Chloride Exposure and Kidney Cancer Risk

The levels of toxins in the water at Camp Lejeune were at levels that were hazardous to humans generally and known to cause kidney cancer. This is assessed given all of the literature cited above, including importantly, the literature analyzing the exact exposure of interest at Camp Lejeune. It also includes the body of other epidemiology literature showing the levels of the different toxins known to be hazardous to humans and to cause kidney cancer. Mrs. Tukes had

no other significant risk factors for the development of her multiple and unique kidney cancer presentation. The small risk factors she did have, such as hypertension for a time and fluctuating BMI, were so small in comparison to known exposures to carcinogens like the ones in the water at Camp Lejeune.

Therefore, it is my opinion that to a reasonable degree of medical certainty, the kidney cancers Ms. Tukes developed were more likely than not caused by her exposure to the contaminated drinking water at Camp Lejeune. This exceeds the "at least as likely as not" standard required in this case. This is especially true given her susceptibility to exposure to chemicals at low levels.

X. Damages

The damages Ms. Tukes suffered as a result of her kidney cancer are listed below. For example, Ms. Tukes medical course was complicated by numerous medical procedures (surgeries) to treat her kidney cancer and the residual effects of her kidney cancer treatments. Ms. Tukes went through numerous surgeries from the time of her first diagnosis of kidney cancer to the present.

In addition to the five surgeries to remove her kidney cancers, she required two surgeries to put in dialysis catheters to facilitate her dialysis that was required because she had both kidneys removed due to cancer. Furthermore, Ms. Tukes required an additional surgery for her transplant to place the new kidney in her right flank when a kidney donor was found. Furthermore, Ms. Tukes underwent dialysis while awaiting a kidney transplant. Additionally:

1. The harms and injuries and damages suffered by Ms. Tukes that are described in this report are permanent.

2. The treatment and care Ms. Tukes has received and is now receiving is reasonable and medically necessary.

3. The Plaintiff is not expected to live a normal life expectancy given her current age of 60.

4. The medical billing relating to Mrs. Tukes' kidney cancer diagnosis, the surgery to remove his kidneys and the follow up treatment related to her kidney cancer was reasonable and medically necessary.

References Consulted

[1] Camp Lejeune Justice Act of 2022, https://www.congress.gov/117/plaws/publ168/PLAW-117publ168.pdf.

[2] 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. 2017:1-150.

[3] Bradford Hill, A. The Environment and Disease: Association or Causation? President's Address January 14, 1965. *Proceedings of the Royal Society of Medicine*. 1965:295-300.

[4] Cancer Institute New South Wales Fact Sheet: Facts for people and families with a constitutional mismatch repair deficiency. 2022. https://www.eviq.org.au/cancer-genetics/consumer-information/4057-facts-for-people-and-families-with-a-constitu

[5] Cancer Institute New South Wales:SMARCA4-associated rhabdoid tumor predisposition syndrome-risk management. 2023. https://www.eviq.org.au/cancer-genetics/paediatric/risk-management/3742-smarca4-associated-rhabdoid-tumour-predisposi.

[6] Masila M, Sautner J, et al. Analyses of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water at Tarawa Terrace and Vicinity, U.S. Marine Corps Base Camp Lejeune, North Carolina: Historical Reconstruction and Present-Day Conditions Chapter A: Summary of Findings. Atlanta, GA: Agency for Toxic Substances and Disease Registry; 2007. Accessed January 1, 20, 2025. https://www.atsdr.cdc.gov/camp-lejeune/php/data-research/tarawa-terrace/index.html.

[7] Maslia M, Suarez-Soto R, Sautner J, et al. *Analyses and Historical Reconstruction of Groundwater Flow, Contaminant Fate and Transport, and Distribution of Drinking Water Within the Service Areas of the Hadnot Point and Holcomb Boulevard Water Treatment Plants and Vicinities, U.S. Marine Corps Base Camp Lejeune, North Carolina -- Chapter A: Summary and Findings. Atlanta, GA: Agency for Toxic Substances and Disease Registry.*; 2013. Accessed April 24, 2024. https://www.atsdr.cdc.gov/camp-lejeune/php/data-researchhadnot-point-a/index.html#cdc_report_pub_study_section_1-chapter-a-summary-and-findings.

[8] Sims JN, Yedjou C, et al. Racial Disparities and Preventive Measures to Renal Cell Carcinoma. *Int. J. Environ. Res. Public Health*. 2018; 15. doi. 10.3390/ijerph15061089.

[9] Beebe-Dimmer J, Colt JS, et al. Body Mass Index and Renal Cell Cancer: The Influence of Race and Sex. *Epidemiology*. 2012;23(6):821-828. doi:10.1097/EDE.0b013e31826b7fe9.

[10] Bove FJ, Ruckart PZ, et al. Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study. *Environmental Health*. 2014. http://www.ehjournal.net/content/13/1/10.

[11] Bove FJ, Ruckart PZ, et al. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environmental Health*. 2014. http://www.ehjournal.net/content/13/1/68.

[12] Bove FJ, Greek A, 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. *Environmental Health*. 2024. https://doi.org/10.1186/s12940-024-01099-7.

[13] Bove FJ, Greek A, et al. Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study. *Environmental Health Perspectives*. 2024;132(10). https://doi.org/10.1289/EHP14966.

[14] Aschengrau A, Ozonoff D, et al. Cancer Risk and Tetrachlorethylene – contaminated Drinking Water in Massachusetts. *Archives of Environmental Health: An International Journal*. 2010;48(5):284-292.

[15] Agency for Toxic Substances and Disease Registry. Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune. 2018:1-126.

[16] Mandel JS, McLaughlin JK, et al. International renal-cell cancer study. IV. Occupation. *Int. J. Cancer*. 1995;61(5):601-605. doi: 10.1002/ijc.2910610503.

[17] Anttila A, Pukkala E, et al. Cancer Incidence among Finnish Workers Exposed to Halogenated Hydrocarbons. *JOEM*. 1995;37(7):797-806.

[18] Calvert GM, Ruder AM, et al. Mortality and end-stage renal disease incidence among dry cleaning workers. *Occup. Environ. Med*. 2011;68:709-716. doi:10.1136/oem.2010.060665.

[19] Callahan CL, Stewart PA, et al. Extended mortality follow-up in a cohort of dry cleaners. *Epidemiology*. 2019;30(2):285-290. doi:10.1097/EDE.0000000000000951.

[20] Ruder A, Ward E, et al. Mortality in dry-cleaning workers: An update. *Am. J. Ind. Med*. 2001;39(2):121-132. doi: 10.1002/1097-0274(200102)39:2<121::aid-ajim1000>3.0.co;2-h.

[21] Christensen KY, Vizcaya D, Richardson H, Lavoué J, Aronson K, Siemiatycki J. Risk of selected cancers due to occupational exposure to chlorinated solvents in a case-control study in montreal. *J Occup Environ Med*. 2013;55(2):198-208. doi:10.1097/JOM.0b013e3182728eab.

[22] Karami S, Colt JS, et al. A case-control study of occupation/industry and renal cell carcinoma risk. *BMC Cancer*. 2012. http://www.biomedcentral.com/1471-2407/12/344.

[23] Delahunt B, Bethwaite PB, et al. Occupational risk for renal cell carcinoma. A case-control study based on the New Zealand Cancer Registry. *Br. J. Urol*. 1995;75(5):578-582. doi:

10.1111/j.1464-410x.1995.tb07410.x.

[24] Mayo Clinic Fact Sheet on Kidney Cancer. Aug. 17, 2024. https://www.mayoclinic.org/diseases-conditions/kidney-cancer/symptoms-causes/syc-20352664.

[25] National Toxicology Program. Report on Carcinogens Monograph on Trichloroethylene (RoC Monograph 05). 2015:1-236.

TIMOTHY MALLON'S CV EXHIBIT 1

CURRICULUM VITAE

I. PERSONAL DATA

Name: Timothy M Mallon, MD, MPH, FACOEM
Address: 6508 Folded Leaf Square, Columbia, MD 21044
E-Mail/Tel#:mallonti03@gmail.com/ 443-370-9267

II. EDUCATION

Year	Degree	Type of Degree / Institution
1977	BPS	Bachelor Professional Studies Clarkson University Potsdam, NY
1986	MS	Resource Policy and Management School of Natural Resources University of Michigan
1987	MS	Environmental Health Hunter College City University of Ney York

III. POST GRADUATE EDUCATION

Year	Position	Type of Degree / Institution
1991	Medical Student	Doctor of Medicine Upstate Medical University Syracuse, NY
1992	Resident Year 1	Internship in Internal Medicine Tripler Army Medical Center, Honolulu, HI
1995	Resident Year 2	Master of Public Health School of Hygiene & Public Health Johns Hopkins, Baltimore, MD
1996	Resident Year 3	Occ. & Env. Medicine Residency US Army Public Health Center Edgewood Area-APG, Gunpowder, MD

IV. ACADEMIC APPOINTMENTS

Year	Position	Institution
2016	Professor/Adjunct Professor	Uniformed Services University
2012	Associate Professor	Uniformed Services University
2004	Assistant Professor	Uniformed Services University
1996	GPM Residency Faculty	Madigan Army Medical Center
1995	Teaching Fellow	Johns Hopkins University School of Medicine

V. CURRENT POSITIONS

Veterans Evaluation Services **Occupational Medicine Contract Consultant** **Oct 2017 to Present**

Duties/Accomplishments Hours per work- 30
-Review Camp Lejeune & Agent Orange cases for service members and Veterans
-Review Gulf War Injury Claims, PACT Act TERA Claims and write medical opinion for VBA
-Address causal connection between exposure and related health outcomes

Montgomery County Retirement **Occupational Medicine** **Oct 2017 to Present**

Duties/Accomplishments Hours per work- 5
-Review disability cases for Montgomery County employees
-Apply standards of medical fitness for police, firefighters, other employees who are injured
-Advise management regarding whether medical documentation supports ongoing disability

Federal Occupational Health Bethesda, MD **Occupational Medicine Contract Consultant** **Oct 2016 to Present**

Duties/Accomplishments Hours per work- 10
- Review ADA and FMLA cases for medical employability determinations
- Review preventive medicine informational booklets for technical accuracy
- Review respirator questionnaires and make recommendations for respirator wear

Department of Prev. Med. and Biostatistics, USU **Adjunct Professor** **July 2016 to Present**

Duties/Accomplishments: Hours per week 10
- Serve as mentor to colleagues in PMB on current research projects
- Serve as Specialty Editor for the Textbook of Military Medicine

VI. PRIOR POSITIONS HELD

Health Research Sys Admin. Comp. Injury Countermeasures **Occupational Medicine Consultant** **April 2022 to May 2023**

Duties/Accomplishments Hours per work- 10
-Review claims for injuries related to COVID-19 vaccinations
-Make determination of whether injury exists and causation
-Prepare recommendations for program director & legal review

Brown and Brown Physician Disability Associates **Occupational Medicine Consultant** **April 2017 to Oct 2021**

Duties/Accomplishments Hours per work- 5
-Review disability cases for multiple insurance companies
-Apply standards of medical fitness for workability for injured/ill employees
-Advise management regarding whether medical documentation supports ongoing disability

Prior Jobs (Cont)	Duty Title	Dates
Department of Prev. Med. and Biostatistics, USU	**Professor and OEM Residency Director**	**July 2012 to June 2016**

Duties/Accomplishments: Hours per week 80

- Serve as Residency Director of the OEM Residency Program at USU,
- Selected, trained, and mentored OEM physicians for the Department of Defense.
- Revised training and assessment of residents to document ACGME competencies.
- Oversaw the training of 25 military OEM physicians from the US and Canada.
- Led efforts nationally among residency directors to implement ACGME Milestones, developed Milestones Translation Tools and shared best practices.
- Led ACOEM President's Task Force on Recruiting physicians to OEM
- Prepared the residency for an accreditation site visit, received maximal accreditation.
- Preceptor for Occupational medicine residents and medical students. Served as course director for four occupational medicine courses.
- Authored 8 book chapters and 45 peer reviewed journal articles
- Invited to speak at national meetings including the AOHC, APHA, and ACPM conferences and presented multiple poster and oral presentations.
- Chaired Residency Advisory Committees for OEM Residencies at Madigan and Pensacola and served on Committees for Dayton, Johns Hopkins, Walter Reed & USUSU Prev. Medicine

Dept of Preventive Med. and Biostatistics USU	**Vice Chair for Prev Med.**	**July 2010 to June 2012**

Duties/Accomplishments: Hours per week: 40

- Led efforts to support for Medical School Curriculum Reform, enlarged role of Public Health.
- Led the PMB leadership committee, providing policy guidance to the Chair
- Served on the Medical School Student Promotions Committee for the University.
- Served as Chair of the Preventive Medicine Leadership Committee.
- Participated on the Medical School Curriculum Committee representing the Department.

US Army Surgeon General's Office	**Consultant in OEM**	**July 2008 to June 2012**

Duties/Accomplishments: Hours per week 30

- Served as subject matter expert & consultant to Army Surgeon General.
- Provided OEH consults to 130 OH clinics worldwide.
- Recommended to Human Resources Command assignments/deployments of OEM physicians.
- Developed OH Improvements that focused on Staffing, Training, Credentialing & performance.
- Validated workload, staffing requirements successfully obtained $54.5 million for OH Program.
- Developed Army web-based OHP checklist to track OH clinic performance.
- Updated DoD OEM physician credentialing requirements for OH providers.
- Served as Chair of DoD OEM Working Group: Updated OH Surveillance Manual (DoD 6055.05M) that provides guidance on meeting federal law and regulations from OSHA.
- Led efforts to develop a DoD Biomarker Policy; developed DoD process/outcome performance measures for OH Program execution.
- Led VA and DoD efforts to revise the DoD Post Deployment Health Assessment DD FORM 2796 to better capture soldier deployment OEH health exposure concerns.

- Linked deployment exposures in Defense Medical Surveillance System with health outcomes.

Dept of Preventive Med. and Biostatistics | **OEM Residency Director** | **July 2004 to June 2010**

Duties/Accomplishments: Hours per week: 40
- Serve as Residency Director of the OEM Residency Program at USU,
- Selected, trained, and mentored OEM physicians for the Department of Defense.
- Revised training and assessment of residents to document ACGME competencies.
- Obtained additional training starts and successfully recruited best DoD physicians to the field.
- Doubled the size of the residency from eight to sixteen residents a year each year.
- Oversaw the training of sixty military OEM physicians from the US and Canada.
- Led residency programs nationally in implementing the Milestones and was commended by the National Capital Consortium and ACGME for these efforts
- Prepared the residency for two ACGME site visits and received the maximal accreditation.
- Preceptor for Occupational medicine residents and medical students. Served as course director for four Occupational medicine courses.
- Authored a book chapter and 13 peer reviewed journal articles and was invited to speak at national civilian medical meetings including the American OH Conference and Federal Occupational Health Conference and presented multiple poster and oral presentations.

Army Center for Public Health, Aberdeen Proving Grounds, Gunpowder, MD | **Director, OEM** | **August 2000 to July 2004**

Duties/Accomplishments:
- Served as Director of the OEM Directorate at the US Army Public Health Command.
- Provided oversight of Army OEH worldwide technical consultations.
- Supervised staff of 32 and managed a budget of over $3.9 million
- Developed policy and programs to reduce injuries, illnesses; lower FECA costs; obtained $1 million for pilot project demonstrated medical case managers effective and achieved a 4:1 ROI.
- Developed policy and advised commanders on FHP measures related to CRBN threats.
- Primary author of "Occupational Health" for the 2005 revision of DA Pamphlet 40-11.
- Assessed the quality of Army worker's compensation, OH, and NBC surety programs.
- Developed OH templates for the electronic medical record, AHLTA.
- Standardized OH business practices world-wide as proponent for the MEDCOM commander.
- Coordinated OH support for Pentagon and World Trade Center response: developed exposure guidelines for contaminants to support consequence management and building re-entry
- Oversaw health assessments of 8000 soldiers who deployed to the WTC, Pentagon.

Madigan Army Med. Ctr, Fort Lewis WA | **Chief & Region Consultant Occ. & Env. Med. Service** | **July 1996 to August 2000**

Duties/Accomplishments: Hours per week: 50
- Served as Western Region Medical Cmd. OEM Consultant, oversaw delivery of care in eleven OH clinics in six states from Alaska to Southern California, supported 60,000 personnel.
- Significantly improved patient care, customer satisfaction, and OH clinic utilization.
- Standardized region respiratory protection, blood borne pathogens, latex allergy programs.
- Served as Chief of OEM at Madigan Army Medical Center, provided OM services for 10,000 employees and 40,000 active, guard and reserve solders.

- Supervised staff of 39 providers, His, OHNs and support staff.
- Updated Blood Borne Pathogen, Latex & Infection Control Programs, commended by Joint Commission for model infection control and disaster response programs.
- Chaired the Infection Control, member Hospital Executive, QA/QI and Safety Committees.
- Overhauled tuberculosis (TB) surveillance program to meet JC and OSHA requirements.
- Enhanced chemical response capability through training and exercises.
- Developed and implemented a region wide heat injury prevention plan.

Patterson Army Cmty Hospital, Fort Monmouth, NJ **Chief of Prev. Medicine** **July 1992 to June 1994**

Duties/Accomplishments: Hours per week: 45

- Oversaw delivery of PM services for five installations in NY and NJ;
- Supervised 4 MDs, 4 Industrial Hygienists, 7 nurses, 7 medics, 6 staff.
- Served on PACH Executive Committee, Chair of Infect Control Cmtte, hospital QA/QI.
- Upgraded OH services in region by organizing, updating SOPs and QI programs for PM that resulted in accessible, high quality care and commended by Joint Commission.
- Provided oversight of disease, injury prevention programs at two OH clinics, acute care clinic.
- Ensured workplace IH monitoring conducted that guided worker medical surveillance programs, obtained $250K for IH regional support.
- Provided Installation Commanders advice on community health, safety, lead poisoning and TB prevention and control plans, Travel Medicine, Post Deployment Surveillance for soldiers.

VII. CERTIFICATION AND LICENSURE

	Date
American Board of Preventive Medicine	
Board Certification in Occupational Medicine	16 Jan 1997

License: Maryland

VIII. MEMBERSHIP IN SCIENTIFIC SOCIETIES\PROFESSIONAL ORGANIZATIONS.

Association of Military Surgeons of the United States
American College of Occupational and Environmental Medicine (ACOEM)
American College of Preventive Medicine (ACPM)

IX. FUNDED GRANTS

Title	Role	Funded (amount)	Grant Period
Exposure biomarkers & health outcomes in Iraq and Afghanistan, funded by DoD/NIEHS	PI	$4,650,000	9/8/2013 to 7/31/2019

X. PRIOR TEACHING ACTIVITIES

PMO 973	OEM Journal Club- Co-course Director
PMO 542	Clinical Occupational / Environmental Medicine- Co-course Director
PMO 655	Safety and Injury Prevention- Course Director
PMO 642	Clinical PM Services and Selected Topics in OEM- Co-course Director
PMO 558	Intro to Preventive / Occ. Medicine Residencies- Co-course Director
PMO 549	Toxicology - Course lecturer

XII. **OTHER PROFESSIONAL ACTIVITIES**

Specialty Editor, Textbook of Military Medicine in Occupational Medicine 2019
Special Editor J. Occupational and Environmental Medicine Supplement December 2019
- Deployment Environmental Exposures, Metabolomics, Inflammatory and microRNA
- Biomarkers and Health Outcomes Related to Burn Pits in Iraq and Afghanistan. 2016
- Federal Workers Compensation Programs, Published March 2015.
Editor, Mil. Med. Supplement July 2011, Hazardous Exposures in Military Populations.
Reviewer, Military Medicine on Preventive and Occupational Medicine topics, 2007 to present.

XIII. **CLINICAL ACTIVITIES**

Staff occupational medicine physician, Federal Occupational Health, Bethesda, MD.
Staff Occupational Medicine Physician Walter Reed Army Medical Center 2004 to 2016

XIV. **COMMITTEES** (national advisory, professional societies, hospitals)

Member, Residency Review Committee for Preventive Medicine, Accreditation Council for Graduate Medical Education- 7/2016 to 7/2019
Member, American Board of Preventive Medicine- 7/2016 to 7/2019

XV. **HONORS AND AWARDS**

ACOEM Award for Leadership in Academic Medicine and Research, April 2016
Delta Omega Public Health Honor Society as USU Faculty 2015
Defense Superior Service Medal 2016
US Army Surgeon General's "A-Designator" Award for Academic Excellence
Recipient, Military Order of Medical Merit
Fellow, American College of Occupational and Environmental Medicine.

XVI. **BIBLIOGRAPHY**

Publications (Peer Reviewed)

Krahl PL., Mallon TM., Gaydos JG. Response to Letter to the Editor Regarding Commentary "Hazardous Non-combat Exposures in the Department of Defense". December 2022 Military Medicine 188(5-6). DOI:10.1093/milmed/usac421

Thomas RJ. Krahl PL., Mallon TM., Gaydos JG. Preparedness of Military Public Health for Epidemic and Pandemic Recognition and Response. October 2022. Military Medicine 188(2) DOI: 10.1093/milmed/usac323.

Krahl PL., Mallon TM., Thomas RJ, Gaydos JG. The Future of Military Occupational and Environmental Medicine in the Department of Defense Journal of occupational and environmental medicine. September 2021 DOI: 10.1097/JOM.0000000000002384.

Krahl PL., Mallon TM., Gaydos JG. Hazardous Non-Combat Exposures in the U.S. Department of Defense. June 2021 Military Medicine 187(10). DOI: 10.1093/milmed/usac166

Forging a Healthy and Productive Navy Civilian Workforce. Krahl PL., Thomas RJ., Mallon TM., Gaydos JC. July 2021 US Naval Institute Proceedings Vol. 147/7/1,421.

Krahl PL., Thomas RJ., Gaydos JC., Mallon T.M. Training Occupational & Environmental Medicine (OEM) Residents at the Uniformed Services University, JOEM, Vol 63: Number

5, May 2021.

Krahl PL., Mallon TM., Thomas RJ, Gaydos JG. Training Occupational and Environmental Medicine (OEM) Residents at the Uniformed Services University. The Medical Journal. US Army Center of Excellence. Oct-Dec 2020: pp 4-58.

Mallon, Timothy M.; Krahl, Pamela K.; Haines, Kevin M. Jr.; Use of Biomarkers to Assess Environmental Exposures and Health Outcomes in Deployed Troops. Journal of Occupational and Environmental Medicine. 61:S1-S4, December 2019.

Krahl, Pamela L.; Benchoff, Edward; Mallon, Timothy. Advances in Comprehensive Exposure Assessment: Opportunities for the US Military. Journal of Occupational and Environmental Medicine. 61:S5-S14, December 2019.

Smith, Matthew Ryan; Uppal, Karan; Walker, Douglas I.; Utell, Mark J.; Hopke, Philip K.; Mallon, Timothy M.; Krahl, Pamela L.; Rohrbeck, Patricia; Go, Young-Mi; Jones, Dean P. Environmental Chemicals Altered in Association With Deployment for High Risk Areas. Journal of Occupational and Environmental Medicine. 61:S15-S24, December 2019.

Go, Young-Mi; Smith, Matthew R.; Walker, Douglas I.; Uppal, Karan; Rohrbeck, Patricia; Krahl, Pamela L.; Hopke, Philip K.; Utell, Mark J.; Mallon, Timothy M.; Jones, Dean P. “Metabolome-Wide Association Study of Deployment to Balad, Iraq or Bagram, Afghanistan”. Journal of Occupational and Environmental Medicine. 61:S25-S34, December 2019.

Smith, Matthew Ryan; Woeller, Collynn F.; Uppal, Karan; Thatcher, Thomas H.; Walker, Douglas I.; Hopke, Philip K.; Rohrbeck, Patricia; Mallon, Timothy M.; Krahl, Pamela L.; Utell, Mark J.; Go, Young-Mi; Jones, Dean P. “Associations of Benzo(ghi)perylene and Heptachlorodibenzo-p-dioxin in Serum of Service Personnel Deployed to Balad, Iraq, and Bagram, Afghanistan Correlates With Perturbed Amino Acid Metabolism in Human Lung Fibroblasts”. Journal of Occupational and Environmental Medicine. 61:S35-S44, December 2019.

Thatcher, Thomas H.; Woeller, Collynn F.; Thakar, Juilee; Khan, Atif; Hopke, Philip K.; Smith, Matthew Ryan; Uppal, Karan; Walker, Douglas I.; Go, Young-Mi; Jones, Dean P.; Krahl, Pamela L.; Mallon, Timothy M.; Sime, Patricia J.; Phipps, Richard P.; Utell, Mark J. “Analysis of Post deployment Serum Samples Identifies Potential Biomarkers of Exposure to Burn Pits and Other Environmental Hazards of Exposure to Burn Pits and Other Environmental Hazards”. Journal of Occupational and Environmental Medicine. 61:S45-S54, December 2019.

Khan, Atif; Thatcher, Thomas H.; Woeller, Collynn F.; Sime, Patricia J.; Phipps, Richard P.; Hopke, Philip K.; Utell, Mark J.; Krahl, Pamela L.; Mallon, Timothy M.; Thakar, Juilee. “Machine Learning Approach for Predicting Past Environmental Exposures From Molecular Profiling of Post-Exposure Human Serum Samples”. Journal of Occupational and Environmental Medicine. 61:S55-S64, December 2019.

Thakar, Juilee; Thatcher, Thomas H.; Smith, Matthew Ryan; Woeller, Collynn F.; Walker, Douglas I.; Utell, Mark J.; Hopke, Philip K.; Mallon, Timothy M.; Krahl, Pamela L.; Rohrbeck, Patricia; Go, Young-Mi; Jones, Dean P.; Uppal, Karan. “Integrative Network Analysis Linking Clinical Outcomes With Environmental Exposures and Molecular Variations in Service Personnel Deployed to Balad and Bagram”. Journal of Occupational and Environmental Medicine. 61:S65-S72, December 2019.

Smith, Matthew Ryan; Walker, Douglas I.; Uppal, Karan; Utell, Mark J.; Hopke, Philip K.; Mallon, Timothy M.; Krahl, Pamela L.; Rohrbeck, Patricia; Go, Young-Mi; Jones, Dean P. "Benzo[a]pyrene Perturbs Mitochondrial and Amino Acid Metabolism in Lung Epithelial Cells and Has Similar Correlations With Metabolic Changes in Human Serum". Journal of Occupational and Environmental Medicine. 61:S73-S81, December 2019.

Woeller, Collynn F.; Thatcher, Thomas H.; Thakar, Juilee; Cornwell, Adam; Smith, Matthew R.; Jones, Dean P.; Hopke, Philip K.; Sime, Patricia J.; Krahl, Pamela; Mallon, Timothy M.; Phipps, Richard P.; Utell, Mark J. "Exposure to Heptachlorodibenzo-p-dioxin (HpCDD) Regulates microRNA Expression in Human Lung Fibroblasts". Journal of Occupational and Environmental Medicine. 61:S82-S89, December 2019.

Bedno SA.,Forst L. Mallon TM Systems Evaluation of Occ. Injuries and Illness in the US Army. JOEM, Volume 61, Number 6, June 2019.

Gaydos JC., Mallon TM., Rice WA. "The U.S. Army OEM Residency at Aberdeen Proving Ground, MD: 1960-1996." Mil Med. 2016 Nov;181(11):e1637-e1643.

Mallon TM., Rohrbeck P. Haines KM., Baird CP., Gaydos JC., "Introduction to Department of Defense Research on Burn Pits, Biomarkers, and Health Outcomes Related to Deployment in Iraq and Afghanistan". JOEM Supplement, Volume 58: Number 8S. August 2016.

Walker DI., Mallon TM, Hopke PK, Uppal K, Go YM, Rohrbeck PI, Pennell KD, Jones DP. "Deployment-Associated Exposure Surveillance with High-Resolution Metabolomics". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Woeller CF., Thatcher TH. Twist DV., Pollock SJ., Croadsdell A. Kim N., Hopke PK., Xia X. Thakar J., Mallon TM., Utell MJ. Phipps RP. "Detection of serum microRNAs from Department of Defense Serum Repository: correlation with cotinine, cytokine and polycyclic aromatic hydrocarbon levels." JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Walker DI., Pennell KD., Uppal K., Xia X., Hopke PK., Utell MJ., Phipps RP., Sime PJ., Rohrbeck PI., Mallon TM., Jones DP. "Pilot Metabolome-Wide Association Study of Benzo(a)pyrene in Serum from Military Personnel". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Liu KH, Walker DI., Uppal K., Tran VL., Rohrbeck PI., Mallon TM., Jones DP. "High-resolution metabolomics assessment of military personnel." JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Go YM. Walker DI., Uppal K., Rohrbeck PI., Mallon TM., Jones DP. "Metabolic Pathways and Networks Associated with Tobacco Use in Military Personnel". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Accardi, CJ., Walker DI. Uppal K., Quyyumi AA. Rohrbeck PI., Pennell KD. Mallon TM., Jones DP. "High-Resolution Metabolomics for Nutrition and Health Assessment of Armed Forces Personnel". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Xia X., Carroll-Haddad A., Brown N., Utell MJ., Mallon TM., Hopke PK. "Polycyclic Aromatic Hydrocarbons and Polychlorinated Dibenzo-p-Dioxins/Dibenzofurans in Microliter Samples of

Human Serum as Exposure Indicators." JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Dalgard CL., Polston KF., Sukumar G., Wilkerson MD., Mallon TM., Pollard HB. "MicroRNA Expression Profiling of the Armed Forces Health Surveillance Branch Cohort for Identification of "Enviro-miRNAs". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Woeller CF., Thatcher T., Twisk DV., Pollock S., Croadsdell A., Xia X., Hopke PK., Thakar J., Sime P., Mallon TM,. Utell MJ., Phipps RP. "MicroRNAs as Novel Biomarkers of Deployment Status and Exposure to PAHs, Dioxins and Dibenzofurans". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Masiol M. Mallon TM., Haines, KM., Utell MJ. Hopke, PK. "Airborne Dioxins, Furans, and Polycyclic Aromatic Hydrocarbon Exposure". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Masiol M. Mallon TM., Haines, KM., Utell MJ. Hopke, PK. "Source Apportionment of Airborne Dioxins, Furans and Polycyclic Aromatic Hydrocarbons at A U.S. Forward Operating Air Base During the Iraq War". JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Rohrbeck PI. Hu Z., Mallon TM. "Assessing Health Outcomes after Environmental Exposures Associated with Open Pit Burning in Deployed U.S. Service Members." JOEM Supplement, Volume 58: Number 8S. August 15, 2016.

Mancuso, J. Mallon, T. Gaydos, J. "Needs Group Capabilities Assessment for the Department of Defense (DoD) Serum Repository", Military Medicine, Vol. 180, pp 13-24. October Supplement 2015, available at http//publications.amsus.org/doi/pdf/10.7205/MILMED-D-14-00732.

"Hauret, K., Bedno, S., Loringer, K., Kao, D., Mallon, T., Jones, B. "Epidemiology of Exercise- and Sports-Related Injuries in a Population of Young, Physically Active Adults: A Survey of Military Service Members". Am J. Sports Med. 2015; 43(11):2645-53.

Mallon, TM., Grizzell, TL. Hodgson, MJ. "Federal Workers' Compensation Programs: Department of Defense and Veterans Health Administration Experiences". Journal of Occupational & Environmental Medicine: March 2015 - Volume 57 - Issue - p S1–S3. doi:10.1097/JOM. 0000000000000367, Federal Workers' Compensation Supplement.

Mallon, TM., Grizzell, TL., Nelson, CL.Hodgson, MJ. "Federal Workers' Compensation Program Basics". Journal of Occupational & Environmental Medicine: March 2015 - Volume 57 - Issue – p S4–S11. doi: 10.1097/JOM. 0000000000000407. Federal Workers' Compensation Supplement.

Mallon, TM., Grizzell, TL., Nelson, CL. "Managing Federal Workers' Compensation Injuries and Costs". Journal of Occupational & Environmental Medicine: March 2015 - Volume 57 - Issue - p S12–S19. doi: 10.1097/JOM. 0000000000000410. Federal Workers' Compensation Supplement.

Nelson, CJ. Bigley, DP. Mallon, TM. "Analysis of New Workers' Compensation Claims in the Department of Defense Civilian Workforce, 2000–2012". Journal of Occupational &

Environmental Medicine: March 2015 - Volume 57 - Issue - p S20–S26 doi: 10.1097/JOM.0000000000000368. Federal Workers' Compensation Supplement.

Mallon, TM. Cherry, SE. “Investigating the Relationship Between Worker Demographics and Nature of Injury on Federal Department of Defense Workers' Compensation Injury Rates and Costs From 2000 to 2008”. Journal of Occupational & Environmental Medicine: p S27–S30 .doi: 10.1097/JOM.0000000000000416. Federal Workers' Compensation Supplement.

Mallon, TM. Ortiz, JM. Candler, WH. Rogers, G. Hilburn, R. J. “Investigation of an Outbreak of Anemia Cases at an Army Trinitrotoluene Munitions Production Plant From 2004 to 2005 and Subsequent Surveillance 2005–2013”. Mil. Med. Nov 2014, Vol. 179, No. 11, pp. 1374-1383.

Freeman RJ, Jankosky C, Olsen CH, Mallon T. “Occupational effect on the occurrence of idiopathic venous thromboembolism”. Mil Med. 2012 Oct;177(10):1217-22. PMID 23113451

Bedno, S. Soltis, M. Mancuso, J, Burnett, D. Mallon, T. “The In-Service Examination Score as a predictor of Success on the American Board of Preventive Medicine Certification Examination”, Am J. Prev. Med 2011; 41 (6): 641-644.

“Progress in Implementing recommendations of the National Academy of Sciences Reports: protecting those who serve: strategies to protect the health of deployed US Forces”, Mallon, T. J Mil Med, 7 Suppl. July 6, 2011.

Bedno SA. Hauret, K. Loringer, K. Kao TC. Mallon, TM. Jones, B. “Effects of Personal and Occupational Stress on Injuries in a Young, Physically Active Population: A Survey of Military Personnel”. Military Medicine Nov 2014, Vol. 179, No. 11, pp. 1311-1318.

Cloeren M, Mallon TM, Firestone LM, Burch HC. “Contract Case Managers Prove Cost Effective In Federal Workers”, Mil Med. 2008 Mar;173(3):253-8.

Mallon TM., Parker D. Balmer J. et. al. Chapter 5 US Army Occ. Health Programs and Services in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. Pages 55-69.

Heller JM, Mallon TM, Baird CP, et. al. Chapter 6, History of Army Occ Health, 1991 2015 in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. Pages 71-111.

Mallon TM. Chapter 7, Army Occupational Health Program Management in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. . pp 113-126.

Cameron J. L. Nelson, Sheryl A. Bedno, and Timothy M. Mallon. Chapter 9 Federal Workers’ Compensation Programs in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp 139-151.

Timothy M. Mallon, Cameron J.L. Nelson, David P. Deeter, and Janet M. Ruff. Chapter 10 Medical Surveillance in Textbook of Military Medicine, Occupational Health and the Service

Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp.153-170.

Bedno, S., Loringer, K., Mallon, T. Injury Occurrence from Participation in Sports, April 2008 Status of Forces Survey. US Army Public Health Center Technical Report. August 2011.

Health Hazards to Healthcare Workers, Melissa A. McDiarmid, Kevin P. Michaels, Marlene Sanchez, Gary E. Herr, Michael J. Testa, Daniel J. Caldwell, and Timothy M. Mallon, Chapter 11 in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp 171-198.

Respiratory Protection. Timothy M. Mallon, Cameron J. L. Nelson, Tifani Gleason, Brandon Gardner, Gregory J. Martin, and John P. Barrett. Chapter 13, in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp.219-234.

Beryllium, Dale Harman, Nicole S. Chavis, and Timothy M. Mallon, Chapter 23 in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. , pp 505--519

Explosives and Propellants. Timothy M. Mallon, Peter G. Matos, W. Scott Monks, Raul A. Mirza, and Donald T. Bodeau, Chapter 28, in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp 561-592.

Mallon TM. Phipps RP. Thakar J. Woeller C. et. al. Chapter 29 Biological Monitoring in Textbook of Military Medicine, Occupational Health and the Service Member Mallon TM (Editor). Office of the Surgeon General, US Army Borden institute, San Antonio, Texas, 2019. pp 593-616.

Bedno, S., Loringer, K., Tzu-Cheg,K., Hauret,K., Mallon, T. Jones B. “Injury Occurrence from Participation in Sports, Exercise, and Recreational Activities Among Active Duty Service Members, Descriptive Analysis of Data from the April 2008 Status of Forces Survey of Active Duty Members. US Army Public Health Command Technical Report. August 2011.

Cloeren M, Mallon T. “Managing Workers Compensation Costs in the Military Setting: The Army’s Story”. Clin Occup Environ Med. 2004. May; 4(2):vii, 323-39. Review.

Textbook Chapters

Mallon TM., Parker D. Balmer J. et. al. Chapter 5 US Army Occ. Health Programs and Services Pages 55-69, in in Occ. Health & the Service Member, TM Mallon, editor, 632 pages, 2019.

Heller JM, Mallon TM, Baird CP, et. al. Chapter 6, History of Army Occ Health, 1991-2015, pp 71-111 in Occupational Health & the Service Member, TM Mallon, editor, 632 pages, 2019.

Mallon TM. Chapter 7, Army Occupational Health Program Management. pp 113-126. in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

Nelson CJL., Bedno SA., Mallon TM. Chapter 9 Federal Workers' Compensation Programs. Pages 139-151, in Occ. Health & the Service Member, TM Mallon, editor, 632 pages, 2019.

Mallon TM. Nelson CJL. Deeter DP. Ruff JM. Chapter 10 Medical Surveillance. pp 153-170, in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

Mcdiarmid MA. Michaels KP. Sanchez M. Herr GE. Testa MJ. Caldwell DJ. Mallon TM. Chapter 11 Health Hazards to Healthcare Workers. pp. 171-198, in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

Mallon TM. Nelson CJL., Gardner B. Martin GJ. Barrett JP. Chapter 13 Respiratory Protection. pp. 219-234, in Occupational Health & the Service Member, TM Mallon, editor, 632 pages 2019.

Dale Harman D. Chavis NS. Mallon TM. Chapter 25 Beryllium. pp. 505-519. in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

Mallon TM. Matos PG. Monks WS. Mirza RA. Bodeau DT. Chapter 28 Explosives and Propellants. pp 561-592, in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

Mallon TM. Phipps RP. Thakar J. Woeller C. et. al. Chapter 29 Biological Monitoring, pp 593-616, in Occupational Health and the Service Member, TM Mallon, editor, 632 pages, 2019.

TIMOTHY MALLON'S STATEMENT OF COMPENSATION

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DIVISION

IN RE:	)	
	)	
CAMP LEJEUNE WATER LITIGATION	)	
	)	
This Document Relates to:	)	Case Nos.:
	)	
ALL CASES	)	7:23-CV-897
	)	
DAVID DOWNS	)	7:23-CV-01145-BO
	)	
DAVID WILLIAM FANCHER	)	7:23-CV-00275-BO-BM
	)	
ALLAN WAYNE HOWARD	)	7:23-CV-00490-BO
	)	
FRANK W. MOUSSER	)	7:23-CV-00667-BO-RN
	)	
JACQUELINE JORDAN TUKES	)	7:23-CV-01553-BO-BM

PLAINTIFFS' DESIGNATION AND DISCLOSURE OF PHASE III EXPERT WITNESSES WITH RESPECT TO KIDNEY CANCER

TIMOTHY M. MALLON'S STATEMENT OF COMPENSATION

Pursuant to Fed. R. Civ. P. 26(a)(2)(B)(vi), Plaintiffs provide the following statement of compensation: Timothy M. Mallon charges $650 per hour for work on the present matter.