Exhibit 386

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Weill Cornell Medicine | NewYork-Presbyterian

Specific Causation Expert Report: Frank W. Mousser

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Signed by:
Joseph J. Del Pizzo, MD
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February 3, 2025

Re: Frank W. Mousser
DOB: 5/12/1963

I am writing this letter in response to your request to provide a medical expert evaluation of the records of Frank W. Mousser with respect to his diagnosis of transitional cell upper tract carcinoma of the kidney and the potential causal relationship to exposure to trichloroethylene (TCE) and other volatile organic compounds including perchloroethylene (PCE), vinyl chloride (VC) and benzene from contaminated water at United States Marine Corp Base Camp Lejeune in North Carolina.

I. My Background

I am a physician, licensed and in good standing to practice medicine in the State of New York. I received my medical education from the Albert Einstein College of Medicine and completed internships and residencies in general surgery and urologic surgery at the University of Maryland School of Medicine in Baltimore. I completed a fellowship in Minimally Invasive Urologic Surgery and Laparoscopic/Endourology at New York-Presbyterian Hospital. I received my board certification by the American Board of Urology in 2003 and have maintained my certification through the maintenance of certification program. I have been Attending Urologist at New York Presbyterian Hospital since 2000. I have also been the Vice Chairman of the Department of Urology at New York Presbyterian Hospital/Weill Cornell Medicine since 2012. I currently hold the position of the E. Darracott Vaughan Distinguished Professor of Urology and Professor of Urology in Surgery at Weill Cornell Medicine. In addition to my teaching duties, I maintain a clinical practice as the Director of the Advanced Minimally Invasive Kidney Donor Program, including many patients with renal masses treated and diagnosed as transitional cell carcinoma of the kidney. For further information concerning my qualifications, please see my curriculum vitae, attached to this report.

II. Records and Materials

During this evaluation, I reviewed and relied on the documents and materials in the attached document entitled materials considered list.

III. Causation Standard

The statute at issue in this case states that there are two ways to meet the causation burden:

> "(2) Standards – To meet the burden of proof described in paragraph (1) a party shall produce 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 a causal relationship is at least as likely as not."

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These standards for causation are defined in science and medicine as either (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" that a causal relationship exists.[1]

Using these parameters, the ATSDR (2017) in its assessment of the evidence, utilized differing causality standards in the context of assessing the causal relationship between the toxins in the drinking water at Camp Lejeune and different diseases, including kidney cancer. Specifically, ATSDR outlined the following causality standards:

"Sufficient evidence for causation: the evidence is sufficient to conclude a causal relationship exists. This category would be met, for example, if:

> "1. There is sufficient evidence from human studies in which chance and biases (including confounding) can be ruled out with reasonable confidence, **or**
>
> "2. There is less than sufficient evidence from human studies but sufficient evidence in animal studies and strong evidence that the agent acts through a relevant mechanism in humans."[1]

"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 bias 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."[1]

This is consistent with how I perform this type of causation analysis, how I interpret these standards and this language in the applicable literature and how reasonable physicians in my field apply the same and similar standards.

This report details standard methodology to determine causation of Mr. Mousser's transitional cell carcinoma of the kidney with consideration of the at least "as likely as not" standard.

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IV. Methodology

I relied on peer reviewed scientific literature pertaining to kidney cancer/upper tract urothelial carcinoma ("UTUC") risk associated with exposure to trichloroethylene (TCE), perchloroethylene (PCE), vinyl chloride (VC) and benzene. including occupational and environmental exposure. In evaluating the causal relationship between exposure to these organic compounds and kidney cancer, several meta-analyses considered to be of high utility as well as epidemiologic evidence were reviewed. As appropriate, such evidence will be cited during the course of this report.

As part of my methodology for this causation analysis I specifically looked to the Bradford Hill considerations, which are often employed to assess whether an observed or proposed association is causal. I analyzed each of the factors as support for my conclusion that Mr. Mousser's kidney cancer was to a reasonable degree of medical certainty caused, by his exposure to the toxins in the water at Camp Lejeune.

Further, as part of my causation analysis, I utilized a differential diagnoses methodology for determining the etiology of Mr. Mousser's kidney cancer. As part of this methodology, I considered the potential risk factors that exist for transitional cell carcinoma, determined which of those potential risks had any possible relevance to Mr. Mousser and finally made a determination as to whether those risk factors were causally related to Mr. Mousser's kidney cancer.

Ultimately, I have concluded that the water at Camp Lejeune was contaminated with significant levels of trichloroethylene (TCE) and other volatile organic compounds including perchloroethylene (PCE), vinyl chloride (VC) and benzene. There is scientific evidence to support causality of each toxin to kidney cancer, including UTUC, using the at least as likely as not standard or equipoise. Epidemiologic studies of both environmental water contamination and occupational exposure provide evidence that the level of exposure at Camp Lejeune to these toxins was sufficient to cause kidney cancer. Therefore, it is my opinion to a reasonable degree of medical and scientific certainty that exposure to the contaminated water at Camp Lejeune is more likely than not the cause of Frank Mousser's kidney cancer. This exceeds the "at least as likely as not" standard required in this case. Further, it is my opinion Frank Mousser was exposed to a substantial amount of the toxins at issue in this case. He was exposed for a substantial duration of time, exposed to a substantial intensity of the toxins and exposed to the toxins at a substantial frequency.

V. Outcome of Interest/ Urothelial Carcinoma of the Renal Pelvis

The outcome of interest in this analysis consists of the development of kidney cancer/UTUC following exposure to the Camp Lejeune water system. Urothelial carcinoma of the renal pelvis or upper tract is a cancer located in the kidney. It has some properties that are different from renal cell carcinoma, the more common form of kidney cancer. While histologically similar to bladder tumors, the majority of epidemiology studies include these cancers with renal cell carcinoma. In reviewing the General Causation Expert Report of Benjamin Hatten, M.D, M.P.H, Dr. Hatten states "most authors that do not separately analyze urothelial tumors include them with kidney cancers, and the measures of association in studies that include urothelial/renal pelvis cancers are similar to studies that do not include urothelial cancers (see appendix 1: table). Furthermore, in studies that directly compare urothelial/renal pelvis cancers to other kidney cancers, the measures of association are similar (Lynge 1997; Raaschou-Nielsen 2003). Urothelial/renal pelvis cancers occur in the kidney. The kidney cancer epidemiological

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studies apply for purposes of this causation analysis. All four of the toxins at issue cause upper tract urothelial carcinoma." This is consistent with my review of the literature and support my opinions in this case. As a result, kidney cancer epidemiology will be used in the causation analysis involving urothelial carcinoma of the upper tract.

VI. Medical History

Frank W. Mousser was a member of the armed services, USMC from July 1982 through January 1988 and was stationed at Camp Lejeune from October 1982 through September 1986. Except for periods of deployment, he lived at the French Creek Barracks supplied by the Hadnot Point water distribution system.

From November 1983 through February 1984, he experienced several episodes of gross painless hematuria necessitating hospitalization and urologic evaluation. The initial episode occurred while on deployment in Beirut, Lebanon, where he was transferred to Italy and after undergoing an intravenous pyelogram and cystoscopy, he was diagnosed with cystitis and treated with antibiotics. A second hospital admission in February 1984 included another cystoscopic evaluation of the bladder, which revealed resolution of his cystitis.

Mr. Mousser was evaluated for gross hematuria again in 2013, and for microscopic hematuria and lower urinary tract symptoms in 2017. Urine cytology at the time revealed reactive atypical cells, and both a CT scan of his abdomen/pelvis and cystoscopy of the bladder were within normal limits.

In July 2020, he began to experience right flank pain and gross hematuria. He underwent nephrology evaluation, and given proteinuria and prior negative urologic work-ups, his clinical picture was initially thought to be suggestive of primary or secondary "loin pain hematuria syndrome" due either to thin basement membrane disease or IgA nephropathy.

However, CT scan performed on August 25, 2020 at Kerville VA medical center revealed a 3.7 centimeter hypo-enhancing mass involving the collecting system of the upper pole in his right kidney. He was again evaluated by urology and on September 3, 2020, a right ureteral catheterization was performed by Dr. Andrew Rockwood which confirmed the presence of the renal mass in his renal pelvis. Brushing sampling from this mass revealed the presence of malignant cells of urothelial origin. He was referred to Dr. Ahmed Mansour Elkenany at Audie L. Murphy Memorial Hospital/VA Medical Center. On October 20, 2020, he underwent a right robotic nephroureterectomy to surgically remove his right kidney, ureter and a cuff of his bladder, with concomitant retroperitoneal lymph node dissection and instillation of intravesical gemcitabine. Surgical pathology revealed a superficial, non-invasive (pTa) high grade transitional cell carcinoma of the right kidney. Since that time, he has undergone surveillance imaging and cystoscopic evaluation of the bladder with no evidence of recurrent or metastatic disease.

With the diagnosis and treatment of his kidney cancer and chronic kidney disease, Mr. Mousser has suffered from insomnia and anxiety. He was diagnosed with adjustment disorder with mixed anxiety and depression. He had behavioral health follow up with psychologist Dr. Janet Mueller and was referred to his primary care physician for psychotropic medication, including escitalopram (changed to sertraline in December 2021) and trazadone. He was ultimately referred to psychiatry as Dr Mueller felt the medication wasn't effective for him.

His other medical history includes hypertension, hypercholesterolemia, coronary artery disease (coronary bypass graft bypass surgery in 2016), overactive bladder, obstructive sleep apnea and chronic back pain from lumbar disc disease.

VII. Factual History

During his deposition, Mr. Mousser testified that he was stationed at Camp Lejeune as a field artillery battery man from October 1982 through September of 1986 other than periods of annual leave and several deployments. He lived at the French Creek Barracks supplied by the Hadnot Point water distribution system and testified that he showered primarily at the Barracks within an open shower area with individual shower heads. He showered at least once per day, more often on certain days based upon level of physical training. Also, his three meals per day were on base in the dining hall. He also testified that he drank primarily water each day supplied at the base, including at the dining facility, in the training field, and at the French Creek Barracks. He testified that he would have two to three cups of coffee each morning, two glasses of water or Kool-Aid/lemonade with each meal and testified that when he was training, he would drink 1.5-2 canteens of water a day during training drills, and half a canteen on non-training days.

These facts provide the basis for the opinion that Mr. Mousser's exposure was substantial. The facts indicate Mr. Mousser was frequently and constantly exposed to the toxins at issue during his day-to-day life. The amount of exposure described above and described in the remainder of Mr. Mousser's deposition and documents was substantial. It clearly was causally related to his kidney cancer and exceeded the levels that are known to cause kidney cancer.

In addition, he discussed the long-term emotional impact that his transitional cell carcinoma diagnosis has had on himself and his wife, Heather Mousser. He has difficulty dealing with his cancer diagnosis, is afraid of contracting cancer again and expresses anger at his exposure to contaminated water at Camp Lejeune. Both he and his wife testified that this has caused him to isolate himself, which has affected their marriage and his work relationships, leading to his retirement from the car dealership industry.

Mr. Mousser had his initial consultation with Dr. Janet Mueller in 2021. Dr Mueller testified that Mr. Mousser continuously expressed grief, sadness and anger due to his cancer diagnosis, its impact on his health, and his exposure to contaminated water at Camp Lejeune. She diagnosed him with adjustment disorder, a stress reaction to a situation that can cause anxiety or depression, or both. Dr Mueller discussed Mr. Mousser's relationship distress with his wife, which he attributed to the health changes with him after his cancer diagnosis as well as her concern for his behavioral health. Dr Mueller also detailed his passive suicidal ideation during their therapy sessions, consistent with his major depressive disorder. She ultimately referred him to specialty care/psychiatry as his medical therapy was not effective and she characterized him as having severe depression.

Mr. Mousser testified that he has no known exposure to other chemical, solvents, heavy metals, pesticides or microplastics.

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VIII. Kidney Cancer Risk Associated with TCE

The International Agency for Research on Cancer (IARC) classifies Trichloroethylene (TCE) as a human carcinogen, specifically citing "sufficient evidence in humans for the carcinogenicity of trichloroethylene. Trichloroethylene causes cancer of the kidney."[2] In addition, available evidence has provided a cohesive database supporting TCE as a known kidney carcinogen. This has been demonstrated in both human and animal studies, with mechanistic data suggesting that the carcinogenic effect of TCE results from its metabolism into genotoxic and cytotoxic intermediates that target the kidney and cause DNA strand breaks and mutations in tumor suppressor genes. The relationship between TCE exposure and kidney cancer risk has been documented in direct occupational exposure as well as residential chronic exposure at low to moderate doses. A study examining kidney cancer risk associated with historic groundwater contamination revealed the 50th-75th percentile of estimated exposure over a 15 year period was associated with an increased risk of kidney cancer with adjusted odds ratio (OR) of 1.78 95% confidence interval (CI) compared to <50th percentile.[3] In this study, the maximum measured groundwater TCE levels varied widely, with estimated TCE exposure levels generally ranging from 0-27.6 ug/L.[3]

Another study providing epidemiologic evidence supporting the association between TCE and renal cell carcinoma risk examined occupational TCE exposure in several European countries.[4] TCE exposure was categorized into one of three levels ranging from 0-<27ug/m^3, 27-270 ug/m^3 and >270 ug/m^3, with almost all TCE exposure occurring at least 20 years before disease onset.[4] For TCE exposure, ORs were significantly elevated for all exposure indices (OR = 1.63-2.34).[4] In addition, this study examined the association between TCE exposure and renal cell carcinoma risk after stratification by GSTT1 genotype, which revealed significant associations among subjects exposed to TCE with an active genotype (OR 1.88; 95% CI) but not among GSTT1 nulls (OR 0.93, 95% CI).[4] The findings of this study support the genotoxic mechanism believed to be causative in the development of renal cell carcinoma in these cases. A follow up analysis examined the association between TCE exposure and subtypes of clear cell renal cell carcinoma, with clear cell B subtypes demonstrating a statistically significant elevated measure of association (OR 3.09).[5]

Additional studies include Karami et al (2012) which also demonstrated that TCE can cause kidney cancer, as the authors performed a meta-analysis of 9 cohort studies which resulted in an overall elevated relative risk of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer.[6] Another meta-analysis included 23 studies: 16 cohort and 7 case-control.[7] This study demonstrated significantly elevated measures of association across all studies (RR 1.42), in only case-control (RR 1.33), and in only studies with well documented exposure assessment (RR 1.34).[7]

In addition to these references, there is literature directly relating to the toxins in the water at Camp Lejeune that supports the causal association between TCE and kidney cancer. Bove et. al. 2014a specifically studied the toxins in the water at Camp Lejeune and found associations between the Camp Lejeune water with all the chemicals at issue (TVOCs) and also individual chemicals.[8] Bove et. al. 2014a found a monotonic exposure response for TVOCs at Camp Lejeune relating to kidney cancer with RR of 1.42 (low exposures), 1.44 (medium exposures) and 1.54 (high exposures).[8] The supplemental tables in this study specifically detail HR for cumulative exposures to TCE for the individuals exposed at Camp Lejeune.[8] The HR for cumulative exposures to TCE were 1.54 (low exposures), 1.21 (medium exposures) and 1.52 (high exposures).[8]

There were additional causal relationships found between the toxic water at Camp Lejeune/TCE in the water at Camp Lejeune and kidney cancer. For example, Bove 2024 (both cancer incidence and cancer mortality) support a causal association for individuals exposed to the water at Camp Lejeune and kidney cancer.[9,10]

Finally, just recently, the EPA gave public notice of a final rule change completely banning TCE in the United States.[11] In the public notice of EPA's ban of TCE, the EPA and its spokespeople specifically listed the connection between TCE and kidney cancer as a reason for the need for the ban.[11] In its notice and rule, it cited Camp Lejeune's water contamination as an example of how TCE can cause cancers, including kidney cancer, at low levels.[11]

I have read the general causation report of experts Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of TCE and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

Of note, Dr. Hatten's report states,

Given that non- parenchymal upper urinary tract (renal pelvis and ureter) shares blood supply from the renal artery with the body of the kidney and that urine flows from the kidney through the renal pelvis and ureter it is at least as likely as not that urothelial cancers share the described carcinogenic mechanism with kidney cancers.

IX. Kidney Cancer Risk Associated with PCE, VC and Benzene

The IARC has classified both vinyl chloride (VC) and benzene as known human carcinogens and PCE as "probably carcinogenic to humans."[2,12] Available epidemiologic data is consistent with toxicological evidence of PCE's carcinogenicity.

a. PCE

Mechanistically, PCE is thought to induce kidney cancer via genotoxicity, oxidative stress leading to DNA strand breaks and mutations, and direct cellular cytotoxicity. Epidemiologic studies involving PCE exposure demonstrate an association with kidney cancer. Aschengrau *et al*. reviewed the cancer risk experienced by a cohort of individuals exposed to PCE via contaminated water supplies on Cape Cod, Massachusetts.[13] Following this discovery, the Massachusetts Department of Health observed "elevations in cancer mortality" in affected areas.[13] This population was then matched to population-based controls to define the risk of cancers for the Cape Cod cohort.[13] The authors found that any PCE exposure (OR 1.23) and low PCE exposure (OR 1.36) demonstrated elevated measures of association with kidney cancer in an analysis not accounting for latency.[13]

The 2018 ATSDR Morbidity Study of Marines and civilians at Camp Lejeune found there was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14] ORs were ≥1.5 for both TCE and PCE in Marines and for TCE/PCE in civilian employees.[14] In addition, an occupational case-control study published after the ATSDR Assessment reported an OR of 3.0 (95% CI: 0.99, 9.0) for kidney cancer among those with high PCE exposure intensity and high cumulative exposure after excluding those with ≥50% probability of TCE exposure.[15]

Many studies examining PCE exposure in occupations involve the dry-cleaning industry. For example, an elevated measure of association (SMR 1.41) for kidney cancer mortality was reported in a cohort study of dry cleaner union members who worked in PCE exposed shops for at least a year prior to 1960 with up to a 20-year latency period.[16]

Further, the EPA just enacted a rule banning PCE products and in that rule used as a basis that PCE is causally associated with kidney cancer and that PCE can cause kidney cancer at low levels.

b. Vinyl Chloride

Mechanistically, vinyl chloride is thought to induce kidney cancer via oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts. A DNA adduct is a segment of DNA that is chemically bonded to a cancer-causing chemical, inducing carcinogenesis.

There are epidemiologic studies involving vinyl chloride exposure that demonstrate an association with kidney cancer. Hu et el al (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to vinyl chloride, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to vinyl chloride, the adjusted OR was 2.0 (95% CI = 1.2–3.3).[17] In addition, Bove at al (2014a) found an elevated measure of association (HR 1.55) for kidney cancer deaths of military personnel stationed at Camp Lejeune compared to Camp Pendleton with at least low exposure to vinyl chloride.[8] Bove et al (2014a) found significantly increased HR at low, medium and high levels of exposure to VC; 1.66 (low exposure), 1.61 (medium exposure) and 1.51 (high exposure).[8]

c. Benzene

Mechanistically, benzene is thought to induce kidney cancer via its metabolites inducing oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts.

There are epidemiologic studies involving benzene exposure that demonstrate an association with kidney cancer. The Hu study (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to benzene, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to the specific chemical, the adjusted OR was 1.8 (95% CI = 1.2–2.6).[17] Another occupational study of benzene exposure and kidney cancer was published by Greenland et al (1994). [18] This case-control study of benzene exposure in transformer manufacturing workers in Massachusetts found an OR of kidney cancer with benzene exposure of 4.29 (95% CI 1.33-13.8).[18] In addition, Seyyedsalehi et al (2024) performed a meta-analysis of 29 studies and found an association between occupational benzene exposure and kidney cancer, with an OR 1.20 (95% CI 1.03-1.39).[19]

I have read the general causation report of expert Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of PCE, VC and Benzene and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

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X. Epidemiology Specifically Related to Upper Tract Urothelial Carcinoma

I also reviewed epidemiological studies that specifically consider upper tract urothelial carcinoma either under the category of kidney cancer or separately from kidney cancer.[20,21,22,23,24] As stated above, many studies specifically list UTUC under the category of kidney cancer. For example, Zhao et al. conducted a cohort study of workers employed at an aerospace company between 1950 and 1993 who were exposed to TCE.[20] The authors constructed a job exposure matrix to assess exposure to TCE.[20] The authors classified cumulative exposure based on job title, and their time employed at each job title, as low, medium, high, or none.[20] The authors included malignant neoplasm of the renal pelvis—i.e. upper tract urothelial carcinoma—under the general category of kidney cancer.[20] A significant relationship between kidney cancer mortality and incidence was found at the high TCE exposure level with estimated risk ratios of 2.03 (95% CI: 0.50, 8.32) and 4.90 (95% CI: 1.23, 19.6), respectively.[20] At the medium TCE exposure level, there was an elevated risk of kidney cancer mortality and incidence with estimated risk ratios of 1.43 (95% CI: 0.49, 4.16) and 1.87 (95% CI: 0.56, 6.20), respectively.[20]

Similar results were reached by Pesch et al. when studying the relationship between chlorinated solvents and urothelial carcinoma.[21] In this study, upper tract urothelial carcinoma was considered under the category of urothelial carcinomas.[21] Long exposure to the job task of metal degreasing—which was considered an occupational setting with TCE exposure—had a significant relationship with incidence of urothelial carcinoma with an odds ratio of 2.3 (95% CI: 1.4-3.8).[21] An elevated risk of urothelial carcinoma was found even when the authors categorized exposure based on estimated exposure to select substances.[21] When assessing for select substance exposure using a job-task exposure matrix, the authors found an odds ratio of 1.8 (95% CI: 1.1-3.1) for substantial exposure to PCE and an odds ratio of 1.8 (95% CI: 1.2-2.7) for substantial exposure to TCE.[21]

Studies that categorized upper tract urothelial carcinoma separately reached similar results.[22,23] Raaschou-Nielsen et al. categorized renal pelvis cancer under the category of kidney cancer, but also separately analyzed the renal pelvis cancers alone. This study found an elevated risk of renal pelvis cancer for men exposed to TCE for at least three months with a SIR of 1.2 (95% CI: 0.81, 1.84).[22] The SIR for renal pelvis cancers was identical to the overall kidney cancer SIR, which was also 1.2 for men. Lynge et al. analyzed renal pelvis cancers separately from kidney cancer and found a significant relationship between both renal pelvis cancer and kidney cancer (analyzed separately) with benzene exposure for all Nordic countries: SIR of 2.0 (95% CI: 1.0-3.7) for renal pelvis cancer and SIR of 1.3 (95% CI: 1.0-1.7) for kidney cancer.[23]

In sum, renal pelvis cancer or UTUC has similar risk profiles when considered in the category of kidney cancer or when analyzed separately.[20,21,22,23,24] This supports the analyses used above and throughout this report.

XI. Impact of TCE, PCE, VC and Benzene Exposure from Camp Lejeune

The Agency for Toxic Substances and Disease Registry (ATSDR) has completed and reviewed several epidemiological studies and meta-analyses to determine if personnel and civilians were at increased risk for certain health effects from exposure to this contaminated water.[1] The evidence from the methodological studies establishes that exposure to the levels of the toxins in the drinking water at Camp Lejeune are causes of kidney cancer.[1] All meta-analyses that evaluated epidemiological studies of high utility were based on reports from agencies mandated to evaluate the health risk of the chemicals, including the IARC (2014), EPA (2011) or NTP (2015).[2,25,26] Interpretation of the findings in meta-analyses published and reviewed in the scientific literature for

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TCE exposure and kidney cancer outline the magnitude of the adjusted Hazard Ratio (HR) between 1.2 and 1.4 across multiple studies, the precision of the effect estimates (CI>95%) and examine the impacts of unmeasured potential confounders and exposure misclassification on the HR estimate.[7,27] As noted, other studies in the literature have linked exposure to PCE, VC and benzene to the development of malignancies, including kidney cancer.

Based upon these studies and a literature review of occupational and environmental studies, the ATSDR report assessed the strength of the evidence supporting the causality of kidney cancer from TCE exposure.[1] The conclusion was that sufficient causal evidence exists linking TCE exposure and kidney cancer.[1] There was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14]

There is additional epidemiologic literature relating specifically to the water at Camp Lejeune finding a causal relationship with kidney cancer/UTUC, including Bove 2014a, Bove 2014b, the ATSDR 2018 mortality study, the 2024 Bove mortality study and the 2024 Bove cancer incidence study.[8,9,10,14,28]

XII. The Levels of the Toxins in the Water at Camp Lejeune

ATSDR conducted historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP) distribution systems.[1] Median estimates from the HP distribution system during peak years for TCE was 366ug/L (range 0-783ug/L), PCE 15ug/L (range 0 to 39ug/L) and VC 22ug/L (range 0 to 67ug/L), all of which exceed the EPA's listing of the maximum contaminant level (MCL) for the volatile organic compounds in drinking water in the United States.[1] These values are 5 ug/L for TCE, PCE and benzene; 2 µg/L for vinyl chloride. In addition, the estimated drinking water concentrations of benzene consistently exceeded the current 5 ug/L MCL. This median estimate of TCE within the drinking water also exceeds median values observed to be associated with an increased risk of kidney cancer in several epidemiologic studies referenced within this report.[1]

There are three known exposure pathways from contaminated water: ingestion, inhalation and dermal absorption. Each pathway contributes to level of chemicals within the body, their known biological effects, and therefore to the overall cancer risk.

In reviewing the General Causation Expert Report of Benjamin Hatten, M.D, M.P.H, Dr. Hatten states "Given that the exposure of interest is water contaminated with multiple culprit compounds, the body of literature that directly examines the Camp Lejeune population exposed to the contaminated water system best answers the question of what levels of exposure are associated with kidney cancer." I agree with this statement, and it supports my opinions in this matter as to the causal connection between the camp Lejeune water and Mr. Mousser's kidney cancer.

Exposures to TCE, PCE, benzene and vinyl chloride at Camp Lejeune occurred simultaneously. TCE and PCE are Camp Lejeune water contaminants with a sufficient body of evidence for causation of kidney cancer, with non-monotonic exposure-relationships evident in studies involving Camp Lejeune.[8] Benzene and vinyl chloride are Camp Lejeune water contaminants with a body of evidence that meets the as likely as not standard for causation of kidney cancer. Therefore, an exposure to these compounds that is demonstrably hazardous to humans at Camp

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Lejeune and is causally associated with kidney cancer is the lowest cumulative exposure category that demonstrates an elevated measure of association.

The RR for the cumulative exposure of each individual chemical as it was causally related to kidney cancer were as follows:

PCE: 1.40 (low exposures), 1.82 (medium exposures) and 1.59 (high exposures)[8]

VC: 1.66 (low exposures), 1.61 (medium exposures) and 1.51 (high exposures)[8]

Benzene: 1.31 (low exposure), 1.38 (medium exposures) and 1.36 (high exposures)[8]

TCE: 1.54 (low exposure), 1.21 (medium exposures) and 1.52 (high exposures)[8]

Dr. Hatten also states "the most relevant evidence for on-base exposures is a monotonic exposure-response relationship with TVOC rather than any individual component exposure (Bove 2014a). Thus, the lowest exposure category to cumulative TVOC with a monotonic dose-response provides evidence of a low level of Camp Lejeune water that is hazardous to human health and a known cause of kidney cancer." I agree with this statement and Dr. Hatten's report supports my opinions in this matter.

In Bove (2014a) the classification for low, medium and high exposures were:

TVOCs: >1 – 4600 ug/L-months (low exposure), >4600 – 12,250 ug/L-months (medium exposures) and >12,250 - 64,016 ug/L-months (high exposure)[8]

TCE: >1 – 3,100 ug/L-months (low exposure), >3,100 – 7,700 ug/L-months (medium exposure), >7,700 – 39,745 ug/L-months (high exposure)[8]

PCE: >1 – 155 ug/L-months (low exposure), >155 – 380 ug/L-months (medium exposure), >380 – 8,585 ug/L-months (high exposure)[8]

Vinyl chloride: >1 – 205 ug/L-months (low exposures), >205 – 500 ug/L-months (medium exposures), >500 – 2,800 ug/L-months (high exposures)[8]

Benzene: 2 – 45 ug/L-months (low exposures), >45 – 110 ug/L-months (medium exposures) >110 - 601 ug/L-months (high exposures)[8]

Mr. Mousser would have met the criteria for the high exposure category for each individual chemical at issue. For TVOC exposure, he was at the very upper end of the medium exposure, and just below the high exposure category.

The Camp Lejeune literature also analyzed exposure by time duration on base. A duration-based intensity of exposure is also supported by the Camp Lejeune literature with a monotonic exposure response evident.[10] The lowest duration category in the monotonic exposure-response finding that demonstrates an elevated measure of

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association is a level that is hazardous to human health and a known to cause kidney cancer. This is the "low" duration group with 1-5 quarters on base (HR 1.36).[10] Mr. Mousser would have been on base for multiples of this time period and would have been in the medium exposure category for this duration based assessment.

Dr. Hatten states in this report "To summarize, if an individual was present at Camp Lejeune and exposed to the levels of the chemicals above, this individual would have been exposed to levels of the water at Camp Lejeune that are hazardous to humans generally and are known to cause kidney cancer."

There were other levels shown in the literature that causally connect the toxins at issue in this case and kidney cancer. These were shown in the general causation reports for Drs. Hatten and Bird as well as cited elsewhere in this report. I will not repeat all these levels in this section, but all should be noted to be relevant to this analysis.

XIII. Specific Causation: TCE, PCE, VC and Benzene Exposure and Frank W. Mousser's Urothelial Cell Carcinoma of the Renal Pelvis

There are risk factors linked to the development of urothelial cell carcinoma of the renal pelvis. Those include tobacco use, exposure to chemicals and dyes used in manufacturing such as plastics, textiles and rubber, exposure to coal, tar, and asphalt, Balkan endemic nephropathy (BEN), genetic conditions such as Lynch syndrome, a history of transitional cell carcinoma of the bladder, use of cancer treating drugs cyclophosphamide and ifosfamide, and excessive use of phenacetin (a pain medication that hasn't been sold in the United States since 1983). An association between occupational risk factors and kidney cancer has also been established in several epidemiologic studies.[6] Occupations that have been linked to kidney cancer include the agricultural, dry cleaning and mechanical industries.

We employ scientific evidence, to attempt to ascertain whether exposure to the known carcinogens in the Camp Lejeune water was the cause of the Mr. Mousser's kidney cancer. Based upon the review of Mr. Mousser's medical records, his time stationed at Camp Lejeune, and review of the scientific and epidemiological evidence, it is my opinion that it is more likely than not that his exposure to the contaminated water at Camp Lejeune was the cause of his kidney cancer.

The following factors support my opinion:

(1) ATSDR historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP) distribution during the relevant times indicate that Mr. Mousser was exposed to water with TCE, PCE, Vinyl chloride and Benzene contamination levels exceeding carcinogenic levels observed in epidemiologic studies demonstrating an increased risk of kidney cancer associated with occupational or groundwater contamination TCE exposure, as well as PCE, VC and benzene exposure.[1]

(2) Frank W. Mousser was stationed at the French Creek Barracks for 891 days from October 18, 1982 through September 7, 1986, not including time away from the base for several deployments. During his time at camp Lejeune he lived at the French Creek Barracks supplied by the Hadnot Point water distribution system. The soldiers and civilian personnel at Camp Lejeune typically experienced multiple routes of exposure. In his deposition testimony, Mr. Mousser stated that he continued to eat and

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hydrate on the base daily, as well as shower there a minimum of once per day for a minimum of ten minutes, even when he lived off base. Scientific studies have demonstrated all three routes of exposure are significant in contributing to overall cancer risk. Further and significantly, Bove and ATSDR studied civilians who lived off base and worked on base, for example, in Bove 2014b, there were significantly elevated risks for kidney cancer seen in the epidemiology for these individuals as well even though they spent time living off base.[28]

(3) The ATSDR water modeling I have reviewed indicates that the levels of TCE, PCE, VC and Benzene in the water at Hadnot Point from October 1982 through September 1986 were as follows in ug/L-months:

Exposure Dates	Total Days	TCE (uG/L)	PCE (ug/L)	VC (ug/L)	Benzene (uG/L)
10/18/82-10/31/82	14	138	6	9	9
11/1/82-11/26/82	26	706	34	55	10
12/20/82-12/31/82	12	721	35	56	8
1/1/83-12/31/83	31	389	19	30	8
4/19/83-4/30/83	12	372	18	29	10
5/1/83-5/30/83	30	449	22	36	8
6/11/83-6/30/83	20	546	27	45	7
7/1/83-7/31/83	31	618	30	51	7
8/1/83-8/31/83	31	659	32	54	9
9/1/83-9/30/83	30	543	26	45	9
10/1/83-10/17/83	17	134	5	9	10
2/11/84-02/29/84	19	560	27	47	8
3/1/84-3/31/84	31	587	28	50	7
4/1/84-4/30/84	30	400	18	33	12
5/1/84-05/31/84	31	491	23	42	10
6/1/84-6/30/84	30	471	22	41	7
7/1/84-7/30/84	31	507	24	45	7
8/1/84-8/31/84	31	539	26	48	8
9/1/-9/5, 9/21/84-9/30/84	15	443	21	39	8
10/1/84-10/31/84	31	94	3	6	8
11/1/84-11/30/84	30	639	31	59	8
12/2-12/2, 12/15-12/31/84	19	43	2	4	2
1/1/85-1/22/85	22	324	16	31	4
8/9/85-8/31/85	23	0	0	0	3
9/1/85-9/19/85	19	0	0	0	3
10/11/85-10/31/85	21	0	0	0	3
11/1/85-11/30/85	30	0	0	0	3
12/1/85-12/31/85	31	0	0	0	3
1/1/86-1/12/86	12	0	0	0	3
2/27/86-2/28/86	2	0	0	0	3
3/1/86-3/31/86	31	0	0	0	3

4/1/86-4/30/86	30	0	0	0	4
5/1/86-5/31/86	28	0	0	0	3
6/1/86-6/30/86	27	0	0	0	3
7/1/86-7/31/86	28	0	0	0	3
8/1/86-8/31/86	28	0	0	0	3
9/1/86-9/7/86	7	0	0	0	3
Total	891	10,373	495	864	227

The median level of these contaminants in the water during this time period was 324 ug/L for TCE, 16 ug/L for PCE, 29ug/L for VC and 7 ug/L for benzene. This median estimate of TCE within the drinking water exceeds median values observed to be associated with an increased risk of renal carcinogenesis in several occupational and environmental exposure based epidemiologic studies, including several referenced within this report.

(4) As stated, the scientific literature supports that the most relevant evidence for on-base exposures is a monotonic exposure-response relationship with TVOC rather than any individual component exposure.[8] Thus, the lowest exposure category to cumulative TVOC with a monotonic dose-response provides evidence of a low level of Camp Lejeune water that is hazardous to human health and a known cause of kidney cancer. Frank Mousser during his time at Camp Lejeune, was exposed to the levels of the chemicals listed above, and both his exposure levels to the individual toxins as well as total volatile organic compounds are hazardous to humans generally and are known to cause kidney cancer. (HR 1.44).[8] For example, Mr. Mousser was likely exposed to the following amounts of the four primary chemicals in the water at Camp Lejeune: TCE: 10,373 ug/l-M, PCE: 495ug/l-M, VC: 864 ug/l-M and benzene: 227 ug/l-M. Mr. Fancher's TVOC's place him in the medium exposure group of >4600 – 12,250 ug/L-months.[8] The RR for the medium exposure group in this monotonic response relationship was 1.44.[8] Mr. Mousser is in the high exposure category for each individual chemical and at the very high end of the medium exposure category for TVOC exposure. In addition, based upon the duration-based intensity of exposure supported by the Camp Lejeune, Mr. Mousser can be categorized within the medium group that is known to cause kidney cancer (6-22 quarters, HR 1.36).[10] Mr. Mousser was exposed to the water at Camp Lejeune for approximately 891 days over approximately 30 months. Mr. Mousser's exposure to the chemicals in the water at Camp Lejeune was at levels found to be causally related to kidney cancer in many other studies and citations as shown above and in the general causation reports of Drs. Hatten and Bird.

(5) Mr. Mousser was found to have a right renal mass subsequently diagnosed as urothelial cell carcinoma of the renal pelvis at age 57, which a sufficient latency period after his exposure to TCE, PCE, VC and benzene, in the contaminated water at Camp Lejeune. This is consistent with studies in the scientific literature examining kidney cancer risk associated with historic groundwater contamination of estimated TCE exposure over a 15 year period. Many studies referenced in this report utilized significant latency periods (10-20 years) to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior to the diagnosis of kidney cancer.[8,28] Two of these studies even conducted sensitivity analyses with up to 20-year lags without substantive changes in results.[8,28]

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XIV. Differential Diagnosis as to Cause

Consideration of risk factors for urothelial cell carcinoma of the renal pelvis is performed in the analysis of a likely cause.

1. Unmodifiable risk factors
    a. Family history/Genetic syndromes
        i. Lynch syndrome is an inherited genetic disorder that increases the risk of developing certain types of cancer, particularly colorectal cancer. The lifetime risk of developing urothelial cell carcinoma in these patients is approximately 5-10%.

2. Modifiable risk factors
    a. Tobacco use
        i. Cigarette smoke contains many carcinogens such as polycyclic aromatic hydrocarbons
    b. Occupational/environmental exposures
        i. Exposure to chemicals and dyes used in manufacturing such as plastics, textiles and rubber.
        ii. TCE, PCE, vinyl chloride, and benzene are all known to cause upper tract urothelial carcinoma
    c. History of transitional cell carcinoma of the bladder
        i. A person with a history of bladder cancer has a slightly increased risk of developing transitional cell carcinoma (TCC) in the kidney, with studies showing a lifetime risk range of 1% to 4%.
    d. Balkan endemic nephropathy
        i. A type of interstitial nephropathy that results in end stage kidney disease characterized by xanthochromia of palms and soles (Tanchev's sign), early and absence of hypertension or proteinuria. These patients have a high incidence of urothelial carcinoma of the kidney.
    e. Cancer treating drugs cyclophosphamide and ifosfamide,
    f. Excessive use of phenacetin (a pain medication that hasn't been sold in the United States since 1983)

In the history and medical records provided as well as deposition testimony, Mr. Mousser did not have a history exposure to other chemicals, solvents, heavy metals, pesticides or microplastics, other than his known exposure to the Camp Lejeune water system. He did not have a family history of kidney or bladder cancer and had several prior cystoscopies that showed no evidence of bladder tumor. He has no relevant family history of genetic syndromes such as Lynch syndrome. He did provide a brief history of tobacco use (0.5-1.0 pack/week) while in the USMC but quit thirty years prior to his diagnosis of urothelial cell carcinoma of the renal pelvis. There is testimony that Mr. Mousser smoked daily (of unknown quantities) in 2012 while working at a car dealership. Mr. Mousser disputes this and says that, although he did smoke a very limited amount, it was only 1 or 2 days a week and only a couple of cigarettes each time. Studies demonstrate a strong dose-dependent increase in risk associated with numbers of cigarettes smoked per day and a substantial reduction in risk for long-term former

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smokers.[29] Furthermore, the potential smoking that did take place in 2012 would not have made his already overall limited smoking history any more significant. This is true under either factual scenario (daily of unknown but limited quantities vs. a couple of cigarettes a week). He has no other known exposure to environmental toxins such as herbicides or pesticides. There is no evidence that this would offset the contribution of his known exposure in the contaminated water at Camp Lejeune with multiple toxins known to be causally associated with kidney cancer: TCE, PCE, VC and Benzene. Mr. Mousser had exposure to these toxins over a thirty-month time period at Camp Lejeune.

I have analyzed all of the potential risk factors and the Camp Lejeune water contamination is the most likely cause of Mr. Mousser's kidney cancer. In addition to risk factors I have felt to be relevant, in an effort to be complete, I have also reviewed Defendants' supplemental answers to interrogatories for causal relationships Defendants have raised as potentially causing Mr. Mousser's kidney cancer. I have rejected all of those as well, as shown below.

XV. Substantial Exposure

When determining whether a person's exposure to a toxic chemical is substantial versus de minimis, it is important to look to the amount of the exposure, the duration of the exposure, the frequency of the exposure and the intensity of the exposure. For Mr. Mousser, each of these factors indicates a substantial exposure.

For example, Mr. Mousser was on base for a total of approximately 891 days. This is two and a half year's worth of just time on base. This extended duration of time is substantial and markedly exceeds anything that could be considered de minimis.

The levels of the chemicals in the water were of a substantial intensity. These levels have been shown in the literature to be incredibly hazardous and known to cause kidney cancer.

Mr. Mousser was exposed daily through multiple routes of exposure. He was exposed by ingesting the chemicals, as stated above and in his deposition, through inhalation in the showers and through other activities in which there would have been steam from the water, and dermally as Mr. Mousser came in contact with the water repeatedly throughout his day on his skin.

To a reasonable degree of medical certainty, it is more likely than not that Mr. Fancher's exposure to the water at Camp Lejeune was substantial.

Given the significantly strong correlation between the water at Camp Lejeune and kidney cancer, including at the levels that existed during the time Mr. Mousser was present at Camp Lejeune, it is more likely than not that Mr. Mousser's kidney cancer was caused by the drinking water at Camp Lejeune.

My opinion that Mr. Mousser had substantial exposure is based upon Mr. Mousser's deposition, the concentrations in the water at the time Mr. Mousser was exposed and corresponding documents from Mr. Mousser's file that detail this exposure. However, I also reviewed exposure charts provided to me from Plaintiff's expert Kelly Reynolds. Dr. Reynolds' charts support my opinions that Mr. Mousser had substantial exposure to the toxins at Camp Lejeune and is consistent with these opinions. The charts detail a reasonable estimated dose of ingestion exposure for Mr. Mousser. Dr. Reynolds' charts are found below:

		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition	Chart 4 Deposition/FM
	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 deposition exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
TCE	10,373	267,296	1,160,828	788,229	1,771,027
PCE	495	12,752	55,380	37,604	84,491
VC	864	22,391	97,241	66,029	148,356
BZ	227	5,595	24,298	16,499	37,071

Ingestion of these levels of TCE alone represent a substantial exposure. However, what must be noted is that these charts only relate to the exposure for ingestion. We know Mr. Mousser was exposed to the toxins in the water through inhalation and dermal exposure as well. While the numbers in this chart are indicative of a very significant and substantial exposure in and of themselves, these numbers are only a part of the full exposure we know Mr. Mousser experienced during his time at Camp Lejeune. Exposure to hundreds of thousands of ppb of TCE is substantial and known to cause kidney cancer. When the multiple tens of thousands of ppb of PCE, VC and Benzene are added to this equation, it is without doubt that these chemicals were related to Mr. Mousser's diagnosis of kidney cancer.

I use these charts to add weight to the differential diagnosis analyses above and to opine Mr. Mousser's exposure was substantial.

XVI. Response to the Government's Answers to Interrogatories

I have reviewed the supplemental answers of the government to interrogatories issued by the Plaintiffs. These answers specific several causes that the government thinks may be causally related to the kidney cancer Mr. Mousser developed. I have reviewed each of these potential arguments and reject each as detailed below:

1. The Government claims the exposure to the chemicals at Camp Lejeune may not have been sufficient to have caused Mr. Mousser's kidney cancer. In addition, the Government claims that the length of time between exposure and diagnosis of kidney cancer may indicate an alternative cause of Mr. Mousser's kidney cancer. This argument lacks merit because of the discussion above with regards to the levels of his exposure to both the individual toxins as well as total volatile organic compounds over a thirty-month period. That exposure was hazardous to humans generally and known to cause kidney cancer. This argument also lacks merit because many studies referenced in this report utilized significant latency periods (10-20 years) to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior to the diagnosis of kidney cancer.[8,28] Two of these studies even conducted sensitivity analyses with up to 20-year lags without substantive changes in results.[8,28] Analysis of these study designs using the

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Bradford Hill factors provides evidence for causation that accounts for the principle of temporality, referring to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause.

2. The Government claims Mr. Mousser has a history of cigarette smoking and that may be relevant to his kidney cancer diagnosis. This argument lacks merit because studies demonstrate a strong dose-dependent increase in risk associated with numbers of cigarettes smoked per day and a substantial reduction in risk for long-term former smokers.[29] Mr. Mousser had a very limited smoking history (1 pack over seven to ten days) over thirty years prior to his diagnosis of urothelial cell carcinoma of the renal pelvis. I have also factored in the relevant smoking history from Mr. Mousser's time at the car dealership in 2012. Mr. Mercer states that Mr. Mousser smoked "daily" but was unsure of the frequency. Mr. Mousser says that his smoking in 2012 was limited to only 1-2 days a week and only a couple of cigarettes each time. Under either scenario, it does not change the outcome of this analysis. First, there is no evidence that Mr. Mousser smoked at such a quantity to significantly increase his risk of UTUC. Second, the amount that Mr. Mousser is alleged to have smoked would be relatively insignificant compared to the extremely substantial exposure he had at Camp Lejeune to four carcinogens known to be causally related to kidney cancer.

3. The Government claims Mr. Mousser may have had an exposure to burn pits while in the military and that may be relevant to his kidney cancer diagnosis. This argument lacks merit because while Mr. Mousser testified that soldiers at Camp Lejeune may have been called upon to work at a burn pit, he does not recall any significant time or exposure at a burn pit during his entire time at Camp Lejeune. Even if he was exposed to a limited number of burn pits, this would be very minimal compared to the very significant exposure we know occurred at Camp Lejeune.

4. The Government claims various VA examiners have found that many of Mr. Mousser's conditions are not related to his water exposure at Camp Lejeune or resulting injuries. Specifically, VA opinions relating to sleep apnea, diabetes, erectile dysfunction and hypertension not being related to the Camp Lejeune water. This argument lacks merit because, as detailed in this report, Mr. Mousser had a significant exposure to the toxins in the Camp Lejeune water system that are known to cause kidney cancer and these other conditions are not related to kidney cancer/UTUC.

5. The Government claims that Mr. Mousser's BMI indicated he was "overweight." This argument lacks merit because obesity is not a recognized risk factor for urothelial cell carcinoma of the renal pelvis. In addition, the medical records do not detail a BMI or weight at the time of kidney cancer diagnosis that met the criteria for obesity. In fact, it would have only been slightly elevated.

XVII. Bradford Hill Factors

Multiple studies reviewed demonstrate an association between exposure to the contaminated Camp Lejeune water system and kidney cancer among Marines and civilians.[8,9,10,14,28] The Bradford Hill considerations are

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employed for a structured analysis to determine whether this association with Mr. Mousser is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Mousser's kidney cancer.

a. Strength of Association

Strength of association is demonstrated by statistical significance. Multiple studies discussed in this analysis demonstrate elevated measures of association between the Camp Lejeune water system that Frank Mousser was exposed to and kidney cancer.[8,9,10,28]

b. Consistency

Consistency refers to studies being done in different populations yielding similar results. Multiple cohort[8,9,10,28] and case control[14] studies reached similar conclusions, providing consistent evidence between an association between exposure to the water system at Camp Lejeune and kidney cancer.

c. Exposure-Response

Studies referenced in this report have demonstrated a monotonic exposure-response relationship between increased TVOC exposure and duration at Camp Lejeune.[8,28] This was a consistent finding despite varied methods of determining exposure within these studies. Frank Mousser, during his time at Camp Lejeune, was exposed to the levels of the chemicals listed above, and both his exposure levels to the individual toxins as well as total volatile organic compounds are hazardous to humans generally and are known to cause kidney cancer.

d. Temporality

Temporality refers to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause. Significant latency periods (10-20 years) were used in studies referenced in this report to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior the diagnosis of kidney cancer. [8,28] Mr. Mousser was diagnosed with renal cell carcinoma at age 57 which is 34 years after the last exposure to the contaminated water at Camp Lejeune.

e. Biological Plausibility

This refers to the concept that a correlation between exposure and a disease process is causal based upon epidemiologic evidence. As discussed, TCE, PCE, vinyl chloride and benzene, all contaminants found in the water at Camp Lejeune, all meet the "as likely as not" standard for causation of kidney cancer. TCE and PCE have well documented mechanisms of kidney carcinogenesis, and vinyl chloride and benzene are both known carcinogens with biologically plausible mechanisms for causation of kidney cancer. The totality of the scientific evidence reviewed meets the biologic plausibility standard for Mr. Mousser's exposure to the Camp Lejeune water and kidney cancer.

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f. Analogy

Frank Mousser's exposure to these toxins in the Camp Lejeune water system are analogous to other contaminated water systems that have been studied for association with kidney cancer, including two systems referenced in this report.[3,13] In addition, there is ample evidence of occupational exposures involving TCE, PCE, vinyl chloride and benzene that provide analogous evidence of causation to kidney cancer.

g. Specificity

The consideration of specificity is limited given that fact that the contaminants in the Camp Lejeune water system are known to cause other adverse health outcomes, including cancer in other organs. In addition, there are other unmodifiable and modifiable known risk factors to kidney cancer. As stated, Mr. Mousser did not have evidence of any specific risk factor documented at the time of his diagnosis at age 57, and his only known exposure was to the contaminants in the Camp Lejeune water system.

h. Coherence

The contaminants in the Camp Lejeune water system are known carcinogens, and literature reviewed includes mechanistic, human and animal studies that provide coherent data demonstrating the association between exposure to the water at Camp Lejeune and the development of kidney cancer.

i. Summary

When the abundant scientific and epidemiologic evidence that directly examines the Camp Lejeune water exposure and the development of Mr. Mousser's kidney cancer is considered through the Bradford Hill analysis, it is my conclusion that the exposure is more likely than not a cause of kidney cancer. Given Frank Mousser's known exposure to the Camp Lejeune water system, the levels found at Camp Lejeune during the relevant time period, and his lack of other risk factors, it is more likely than not to be the cause of his kidney cancer. This analysis helps put weight behind the causal relationship between the water at Camp Lejeune and Mr. Mousser's kidney cancer for purposes of the differential diagnosis and causal relationship.

XVIII. Mr. Mousser's Injuries

I will talk about Mr. Mousser's harms as a result of his kidney cancer, including the medical treatment he required after his kidney cancer diagnosis, the surgery to remove his kidney, his CKD and other medical issues relating to and following his kidney cancer. Additionally:

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

2. The treatment and care Mr. Mousser has received and is now receiving is reasonable and medically necessary.

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3. The medical billing relating to Mr. Mousser's kidney cancer diagnosis, the surgery to remove his kidney and the treatment related to his chronic kidney disease was reasonable and medically necessary.

XIX. Conclusion

In conclusion, given my specific causation assessment, including the medical history of the client, the mechanistic data, and the scientific literature and significant amount of epidemiological evidence reviewed and discussed, it is my opinion to a reasonable degree of medical certainty, that environmental exposure to TCE, PCE, VC and benzene in the water at Camp Lejeune is more likely than not to have constituted the cause to Mr. Mousser's kidney cancer diagnosis.

Sincerely,

Joseph Del Pizzo, MD

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JOSEPH DEL PIZZO'S CV

CURRICULUM VITAE
JOSEPH DEL PIZZO, M.D.
VICE CHAIRMAN, DEPARTMENT OF UROLOGY
E. DARRACOTT VAUGHAN DISTINGUISHED PROFESSOR OF UROLOGY AND SURGERY

A. GENERAL INFORMATION

Office address:	Brady Urologic Associates - New York-Presbyterian Hospital 525 E 68th Street, Starr 918 New York, N.Y. 10021
Office telephone:	(212) 746-5250
Office fax:	(212) 746-0412
Email:	jod2009@med.cornell.edu
Citizenship:	U.S.

B. EDUCATIONAL BACKGROUND

Degree	Institution name, city and state	Dates attended	Year Awarded
M.D.	Albert Einstein College of Medicine New York, NY	1991-1994	1994
B.S. Biology	State University of New York Binghamton, NY	1986-1990	1990

C. PROFESSIONAL POSITIONS AND EMPLOYMENT

Post-doctoral training including residency/fellowship

Title, Institution name, city and state	Dates held
General Surgery Internship University of Maryland School of Medicine Baltimore, Maryland	1994-1995
General Surgery Second Year Resident University of Maryland School of Medicine Baltimore, Maryland	1995-1996
Urologic Surgery Resident University of Maryland School of Medicine Baltimore, Maryland	1996-1999
Urologic Surgery Chief Resident	1999-2000

University of Maryland School of Medicine
Baltimore, Maryland

Fellowship in Minimally Invasive Urologic Surgery 2000-2001
Laparoscopy/Endourology
The New York-Presbyterian Hospital of Cornell University
New York, NY

Academic positions (teaching and research)

Title, Institution name, city and state *Dates held*

Instructor in Urology 2000-01
Director, Laparoscopic Living Kidney Donor program
Weill Cornell Medical College of Cornell University
New York, NY

Assistant Professor of Urology 2001-2007
Director, Laparoscopic and Minimally Invasive Urology
Director, Laparoscopic Living Kidney Donor program
Weill Cornell Medical College of Cornell University
New York, NY

Associate Professor of Urology 2007-2017
Director, Laparoscopic and Minimally Invasive Urology
Weill Cornell Medical College of Cornell University
New York, NY

Associate Professor of Transplantation Surgery
Director, Laparoscopic Living Kidney Donor program 2009-2017
Weill Cornell Medical College of Cornell University
New York, NY

Professor of Urology and Urology in Surgery 2017-present
Weill Cornell Medical College of Cornell University
New York, NY

Hospital positions (e.g., attending physician, if applicable)

Title, Institution name, city and state Dates held
Assistant Attending Urologist 2001-2007
NewYork-Presbyterian Hospital, Cornell
New York, NY

Associate Attending Urologist 2007-2017
NewYork-Presbyterian Hospital, Cornell
New York, NY

Attending Urologist 2017-present
New York-Presbyterian Hospital, Cornell
New York, NY

Vice Chairman, Depart of Urology 2012-present
New York-Presbyterian Hospital, Cornell
New York, NY

Director, Advanced Minimally Invasive Kidney Donor Program
2022-present

D. LICENSURE, BOARD CERTIFICATION, MALPRACTICE

Licensure

State	Number	Date of Issue	Date of last registration
National Board of Examiners	4-006-708-4		
New York	217223	4/20/2000	12/2024
Maryland	D0052527	09/02/1997	09/30/2002
DEA number	DB5591626		

Board Certification

Full Name of Board	Certificate #	Date
The American Board of Urology	13042	2003 - 2025

Malpractice Insurance

Do you have Malpractice insurance? Yes

Name of Provider: MCIC

Premiums paid by:
NewYork-Presbyterian Hospital

E. PROFESSIONAL MEMBERSHIPS (*medical and scientific societies*)

Member	American Urological Association	2001-present

Member	Urologic Society for Transplantation and Renal Surgery	2009-present
Member	American Academy of Clinical Urologists	2001-present
Member	New York Section of Urology	2001-present
Member	Endourology Society	2000-present
Member	Society of Laparoendoscopic Surgeons	2001-present

F. HONORS AND AWARDS

Name of award	Date awarded
John Coleman Outstanding Urology Resident Teaching Award	2006, 2016
Best Doctors in America	2004-2024
New York Super Doctors	2009-2024
Top Doctors, New York Metro Area	2007-2024
Top Urologists in America	2007-2024
Best Urology Video *The New York Experience of Robot-Assisted Pyeloplasty.* 13th International Congress of Laparoendoscopic Surgeons, SLS Annual Meeting New York, New York	2004
Honorable Mention – Best Scientific Paper *Laparoscopic live donor nephrectomy: Donor tolerance and renal allograft outcomes stratified by age.* 13th International Congress of Laparoendoscopic Surgeons, SLS Annual Meeting New York, New York	2004
First place – Best Urology Paper *Minimizing the incidence of vascular complications during right sided laparoscopic live donor nephrectomy* 12th International Congress and Endo Expo, SLS Annual Meeting, Las Vegas, Nevada	2003
U.S. Surgical Laparoscopy Scholar	2000
Pfizer Scholar in Urology	2000
The Society of Laparoendoscopic Surgeons Resident Achievement Award	2000
Travel grant – Resident Essay Contest Loss of cell cycle regulators $p27^{Kip1}$ and cyclin E in transitional cell carcinoma of	1999

the bladder correlates with tumor grade and patient survival
3rd Annual SBUR Meeting, Paris, France.

First place - Resident Essay Contest 1999
Loss of cell cycle regulators $p27^{Kip1}$ and cyclin E in transitional cell carcinoma of the bladder correlates with tumor grade and patient survival
57th Annual Mid-Atlantic Section, AUA, Hilton Head, SC

First place – Resident Essay Contest 1998
Helical CT arteriography for evaluation of living renal donors undergoing laparoscopic nephrectomy
56th Annual Mid-Atlantic Section, AUA, West Palm Beach, Florida

Travel grant – Resident Essay Contest 1998
Laparoscopic donor nephrectomy: The first 200 cases
56th Annual Mid-Atlantic Section, AUA, West Palm Beach, Florida

Third place – Resident Essay Contest 1997
Treatment of radiation induced hemorrhagic cystitis with hyperbaric oxygen
1997 Mid-Atlantic Section, AUA, Hot Springs, Virginia

Outstanding Achievement in Research 1994
Albert Einstein College of Medicine

G. INSTITUTIONAL/HOSPITAL AFFILIATION

Primary Hospital Affiliation:	NewYork Presbyterian Hospital, Cornell
Other Hospital Affiliations:	None
Other Institutional Affiliations:	None

H. EMPLOYMENT STATUS

Name of Current Employer(s): Weill Cornell Medical College of Cornell University

Employment Status

- Full-time salaried by Cornell

I. CURRENT AND PAST INSTITUTIONAL RESPONSIBILITIES AND PERCENT EFFORT

Teaching Dates

Lecturer- Resident Basic Science Conference 7/01-present
Resident Clinic Covering Physician 7/01-present
Medical Student Lecturer: Kidney stone basic science 7/01-present
Clinical Care

Director, Advanced Minimally Invasive Urology/Kidney Donor Program 7/01-present
Responsibility for resident education and skill development
In minimally inasvive laparoscopic renal surgery including
donor nephrectomy for renal transplantation and extirpative
and reconstructive surgery for renal disease

Staffing of resident clinic on weekly basis

Administrative duties	Dates
Operating Room Technology Committee Member	7/03-present
Vice-Chairman, Quality Assurance Committee	2012-present
Physician Organization Leadership Committee	2019-present
Patient Flow Committee	2024

Research 7/01-present

Clinical research and maintenacnce of a large data base for the Brady urology clinical experience in minimally invasive urologic surgery

Current percent effort	%
Teaching	20%
Clinical Care	60%
Administration	15%
Research	5%
Total	100%

J. EXTRAMURAL PROFESSIONAL RESPONSIBILITIES

Editorship 2004-present
*Current Urology Reports***,** Adrenal Diseases Section

Manuscript Peer Review 2004-present
Journal of Urology, Journal of Endourology, British Journal of Urology, Journal of Transplantation

Faculty Instructor 2002-2012
American Urological Association Annual Meeting
Introduction to Laparoscopy Course

Faculty Instructor 2001-2010
American Urological Association Office of Education
Houston, TX
AUA Hand Assisted Laparoscopy Course

Course Director 2009-2014
Brady Urologic Associates/Weill Cornell Medical College
New York, NY
Single Incision Laparoscopy Course

L. BIBLIOGRAPHY

Peer Reviewed Articles

1. **Del Pizzo JJ**, Sigman DB, and Sklar GN: Total transplant ureteral reconstruction: A modification of the Boari Flap. *Techniques in Urology*, 3:3, 168-70, 1997. PMID: 9422450

2. **Del Pizzo JJ**, Jacobs SC and Sklar GN: Ureteroscopic evaluation in renal transplant recipients. *J Endourol*, 12:2, 135-38, 1998. PMID: 9607439

3. **Del Pizzo JJ**, Jacobs SC, Bartlett ST and Sklar GN: The use of bladder in total transplant ureteral reconstruction. *J Urol*, 159:750-54, 1998. PMID: 9474140

4. **Del Pizzo JJ**, Jacobs SC, Bartlett ST and Sklar GN: Urologic complications in bladder drained pancreatic allografts. *Brit J Urol*, 81, 543-47, 1998. PMID: 9598625

5. **Del Pizzo JJ**, Chew BH, Jacobs SC and Sklar GN: Treatment of radiation-induced hemorrhagic cystitis with hyperbaric oxygen: Long term follow-up. *J Urol,* 160 (3):731-33, 1998. PMID: 9720533

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13. **Del Pizzo JJ**, Jacobs SC, Bishoff J, and Jarett TW: Pleural Injury during Laparoscopic Renal Surgery: Early recognition and Management. *J Urol*, 169(1): 41-44, 2003. PMID: 12470468

14. **Del Pizzo JJ.** Trans-abdominal laparoscopic adrenalectomy. Curr Urol Rep, (1):81-86, 2003 PMID: 12537946

15. Munver R**, Del Pizzo JJ**, Sosa RE, Poppas DP: Minimally invasive surgical management of ureteropelvic junction obstruction: Laparoscopic and robot-assisted laparoscopic pyeloplasty. *J Long-Term Effects of Medical Implants*, 13(5):367-384, 2003 PMID: 14649575

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21. Boorjian S, Munver R, Sosa RE, **Del Pizzo JJ**: Right Laparoscopic Live Donor Nephrectomy: A Single Institution Experience. Transplantation, 77(2): 32-36, 2004. PMID:14966422

22. Munver R, Sosa RE, **Del Pizzo JJ**: The advantages of hand-assisted laparoscopy, Curr Urol Rep, 5(2):100-107, 2004. PMID: 15028201

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41. Rosoff JS, Raman JD, Sosa RE, **Del Pizzo JJ**: Laparoscopic radical nephrectomy for renal masses 7 centimeters or larger. *JSLS*, 13(2), 148-53, 2009. PMID: 19660207

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44. Gimenez E, Leeser DB, Wysock JS, Charlton M, Kapur S, **Del Pizzo JJ**. Laparoendoscopic single site live donor nephrectomy: Initial experience. *J Urol*. 184(5):2049-53, 2010. PMID:20850822

45. Rosoff JS, Otto BJ, **Del Pizzo JJ**. The emerging role of robotics in adrenal surgery. Curr Urol Rep 11(1):38-43, 2010.PMID: 20118094

46. Afaneh C, Ramasamy R, Leeser DB, Kapur S, **Del Pizzo JJ**. Is right sided laparoendoscopic single site live donor nephrectomy feasible? *Urology*. 2011 PMID:21397302

47. Leeser DB, Wysock J, Gimenez SE, Kapur S, **Del Pizzo JJ**. Single Port Donor Nephrectomy. J Vis Exp.(49), 2011. PMID: 21445037

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49. Herman MP, **Del Pizzo JJ**. Simultaneous Bilateral Single Port Radical Nephrectomies. JSLS. 15:96-99, 2011. PMID: 21902952

50. Ramasamy R, Afaneh C, Katz M, Chen X, Aull MJ, Leeser DB, Kapur S, **Del Pizzo JJ**. Comparison of complications of laparoscopic versus laparoendoscopic single site donor nephrectomy using the modified Clavien grading system. J Urol. 186(4):1386-90, 2011. PMID:21855950

51. Cha EK, Lee DJ, **Del Pizzo JJ**. Current Status of robotic partial nephrectomy (RPN). BJU Int. 108(6 Pt 2):935-41, 2011. PMID:21917094

52. Afaneh C, Aull MJ, Gimenez E, Wang G, Charlton M, Leeser DB, Kapur S, **Del Pizzo JJ**. Comparison of Laparoendoscopic Single-site Donor Nephrectomy and Conventional Laparoscopic Donor Nephrectomy: Donor and Recipient Outcomes. Urology. Dec;78(6):1332-7, 2011. PMID: 21996107

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63. Seklehner S, Laudano MA, Chughtai B, Jamzadeh A, **Del Pizzo JJ**, Engelhardt PF, Lee RK. Trends in the utilization of percutaneous and open nephrolithotomy in the treatment of renal calculi. **J** Endourol. 27(8):984-8, 2013. PMID: 23590666

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JOSEPH DEL PIZZO'S TESTIMONY HISTORY

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

JOSEPH J. DEL PIZZO, MD'S LIST OF TESTIMONY

Pursuant to Fed. R. Civ. P. 26(a)(2)(B)(v), Plaintiffs provide the following list of testimony: During the previous 4 years, Joseph J. Del Pizzo, MD has testified as an expert at trial or by deposition in the following actions:

1. 6/2/2023: Judy Cook v John Bell, MD et al; Commonwealth of Kentucky, Fayette Circuit Court Division; Case No. 19-CI-00091;

2. 11/10/2023: Aditya v. Cleveland Clinic; 19th Judicial Circuit, St. Luci County, Florida; Case No.: 2022CA000177;

3. 12/20/2024: Wade Williams v Herb Singh, MD et al; District of Travis County, Texas.

JOSEPH DEL PIZZO'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	)	
	)	
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	)	
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

JOSEPH J. DEL PIZZO, MD'S STATEMENT OF COMPENSATION

Pursuant to Fed. R. Civ. P. 26(a)(2)(B)(vi), Plaintiffs provide the following statement of compensation: Joseph J. Del Pizzo, MD has charged $750 per hour for work on the present matter.