Exhibit 384

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Specific Causation Expert Report: David Fancher

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Signed by:
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February 3, 2025

Re: David Fancher
DOB: [redacted]958

I am writing this letter in response to your request to provide a medical expert evaluation of the records of David Fancher with respect to his diagnosis of renal cell carcinoma 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 a large volume of patients with renal masses treated and diagnosed as renal cell carcinoma. 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."

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., $\leq$ 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. Fancher's renal cell carcinoma with consideration of the at least "as likely as not" standard.

IV. Methodology

I relied on peer reviewed scientific literature pertaining to kidney cancer risk associated with exposure to TCE, PCE, VC and benzene including occupational and environmental exposure. In evaluating the causal relationship between exposure to these organic compounds and renal cell carcinoma, several meta-analyses considered to be

of high utility as well as epidemiologic evidence was 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. Fancher'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. Fancher's renal cell carcinoma. As part of this methodology, I considered the potential risk factors that exist for renal cell carcinoma, determined which of those potential risks had any possible relevance to Mr. Fancher and finally made a determination as to whether those risk factors were causally related to Mr. Fancher'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, 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 levels of exposure at Camp Lejeune to these toxins were sufficient to cause kidney cancer. 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 David Fancher's kidney cancer. This exceeds the "at least as likely as not" standard required in this case. Further, it is my opinion David Fancher 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. Medical History

David Fancher was a member of the armed services, USMC from 1976 through 1981. He was stationed at Camp Lejeune from October 1979 through June 1981, including living at the Mainside Barracks supplied by Hadnot Point distribution system.

In December of 1997, aged 39, he was evaluated for the presence of red blood cells in his urine (hematuria). Initial evaluation with intravenous pyelogram was suspicious for a kidney mass given loss of the normal appearing contour of the right kidney. A subsequent CT scan the following day confirmed a large 13x8x7.5-centimeter solid enhancing mass with central necrosis involving the right kidney. Subsequent MRI of the abdomen did not demonstrate retroperitoneal lymphadenopathy or gross involvement of adjacent vascular structures including the inferior vena cava. As a result, he underwent open right radical nephrectomy performed by Dr. Charles Witten on December 12, 1997 at Columbia Medical Center in Sanford, Florida. Surgical pathology was consistent with a 9.5-centimeter clear cell renal cell carcinoma with partial involvement of the renal capsule. His post operative course was complicated by an incisional infection that required local wound care and antibiotics. He has undergone intermittent radiologic surveillance since that time, from 1998-2002 and from 2014-present, and currently has no

evidence of recurrent disease. His overall kidney function has been stable with a serum creatinine ranging from 1.0-1.4mg/dl.

His other surgical history includes inguinal hernia repair in 1993 and cholecystectomy in 2014, as well as prior knee surgery and repair of a flexor tendon injury while in the Marine Corps. He has chronic low back pain/sacroiliitis which has been intermittently treated with injection therapy. He has had routine gastrointestinal screening and has had resection of colon polyps during this surveillance. He is currently on metformin for diabetes mellitus, as well as medical therapy for hypertension and hyperlipidemia.

VI. Factual History

During his deposition, Mr. Fancher testified that he developed a post-operative wound infection that required local wound care with home dressing changes performed primarily by his wife, Camilla Fancher. He also testified that he continues to have chronic incisional numbness and pain, with an asymmetric cosmetic deformity/flank bulge that limits his ability to participate in many of his pre-nephrectomy activities, including golf, softball and fishing. His most recent radiologic imaging is a CT scan of the abdomen and pelvis on 10/11/2024, which revealed no evidence of recurrent disease as well as chronic marked atrophy of the right rectus abdominis muscle, consistent with this asymmetric cosmetic deformity/flank bulge. Recent photographs that I reviewed clearly show this deformity in his right flank.

Mr. Fancher testified that he was stationed at Camp Lejeune from October 1979 through June 1981, other than periods of deployment. During his time at Camp Lejeune, he lived at the Mainside Barracks from October to November 1979, moved to an apartment off base from November 1979 through May 1980, and returned to the Mainside Barracks from November 1980 through June 1981, except for his 30-day deployment in March/April 1981. He testified that he showered primarily at the Mainside Barracks (even when living off base) within an open shower area where individual shower heads were separated by curtains. He showered at least once per day, often more based upon the level of training, and his average shower was thirty minutes in duration. In addition, his three meals per day were on base in the dining hall while living on base, and he ate dinner there approximately three times a week even when living off base. 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 Mainside Barracks.

While living off base, Mr. Fancher stayed in the barracks 1-2 nights a week. He ate breakfast and lunch at the same chow hall every day and dinner in the chow hall approximately 3 times a week. He would still shower on base as well.

These facts provide the basis for the opinion that Mr. Fancher's exposure was substantial. The facts indicate Mr. Fancher was 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. Fancher'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 effects that his renal cell carcinoma diagnosis has had on his entire family, including his children.

VII. 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 50^{th}-75^{th} 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 $<50^{th}$ 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/m³, 27-270 ug/m³ and >270 ug/m³, 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 studies (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 of 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.

VIII. 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; 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.

IX. 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).[2,20,21] Interpretation of the findings in meta-analyses published and reviewed in the scientific literature for 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, 22] 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, including Bove 2014a, Bove 2014b, the ATSDR 2018 mortality study, the 2024 Bove mortality study and the 2024 Bove cancer incidence study.[8,9,10,14,23]

X. 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 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.[1] 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 renal cell carcinoma 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. Fancher'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 contaminant 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

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. Fancher would have met the criteria for medium exposure for each of the chemicals individually and also for TVOC exposure.

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

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. Fancher had a similar exposure.

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.

XI. Specific Causation: TCE, PCE, VC and Benzene Exposure and David Fancher's Renal Cell Carcinoma

There are risk factors linked with an increased risk in the development of renal cell carcinoma. Those include exposure to the toxic chemicals noted above, tobacco use, prolonged hypertension, and excess body weight. An association between occupational risk factors and renal cell carcinoma has been established in several epidemiologic studies.[6] Occupations that have been linked to renal cell carcinoma 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 Mr. Fancher's kidney cancer. Based upon the review of David Fancher'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. Fancher 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.[1]

(2) David Fancher was stationed at the Mainside Barracks from October 16, 1979 through June 12, 1981. This included time where he lived at the Mainside Barracks, and a period of time where he lived in an apartment off the base in Jacksonville, North Carolina. The soldiers and civilian personnel at Camp Lejeune typically experienced multiple routes of exposure. In his deposition testimony, Mr. Fancher stated that he continued to eat and hydrate on the base daily, as well as shower there a minimum of once per day for a minimum of thirty minutes. 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.[23]

(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 1979 through June 1981 was as follows in ug/L-months:

Date	TCE (ug/L)	PCE (ug/L)	Vinyl Chloride (ug/L)	Benzene (ug/L)
10/1/1979	71	3	4	4
11/1/1979	507	23	33	6
12/1/1979	504	23	33	6
1/1/1980	264	12	17	7
2/1/1980	378	17	24	6
3/1/1980	433	20	28	6
4/1/1980	273	12	17	8
5/1/1980	322	15	21	6
12/1/1980	541	26	37	6
1/1/1981	295	14	19	8
2/1/1981	387	18	26	7
3/1/1981	397	19	27	6
4/1/1981	266	12	17	9
5/1/1981	322	18	22	7
6/1/1981	380	18	26	7
Totals	5,340	247	351	99

The median level of these contaminants in the water during this time period was 378 ug/L for TCE, 18 ug/L for PCE, 24ug/L for VC and 6ug/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 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. David Fancher, 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. Fancher was likely exposed to the following amounts of the four primary chemicals in the water at Camp Lejeune: TCE: 5,340 ug/l-M, PCE: 247ug/l-M, VC: 351 ug/l-M and benzene: 99 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] In addition, based upon the duration-based intensity of exposure supported by the Camp Lejeune, Mr. Fancher can be

categorized within the low group that is known to cause kidney cancer (1-5 quarters, HR 1.36).[10] Mr. Fancher was exposed to the water at Camp Lejeune for approximately 337 days. Mr. Fancher'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. Fancher was found to have a large right renal mass subsequently diagnosed as renal cell carcinoma at age 39, which is 18 years after the start of 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 exposure over a 15-year period.

XII. Differential Diagnosis as to Cause

Consideration of risk factors for kidney cancer is performed in the analysis of a likely cause.

1. Unmodifiable risk factors
    a. Age
        i. Sporadic renal cell carcinoma is a disease of older adults. The average age of diagnosis in the United States is 64, though most patients are diagnosed between ages 65 and 74.
    b. Race
        i. In the United States, African Americans, Hispanic Americans and Native Americans have a greater risk of renal cell carcinoma than Caucasian Americans.
    c. Family history/Genetic syndromes
        i. Familial renal cell carcinoma has been defined as 2 or more individuals in a family diagnosed with renal cell carcinoma without evidence of a known hereditary cancer syndrome. A family history of a first-degree relative or second-degree relative with RCC has been found to increase the risk for a renal cell carcinoma.[24]
        ii. To date, four major inherited RCC syndromes have been identified. These include hereditary papillary renal carcinoma (HPRC), von Hippel-Lindau disease (VHL), Birt-Hogg-Dubé syndrome (BHD), and hereditary leiomyomatosis and renal cell cancer (HLRCC). With the exception of HPRC, these syndromes are associated with other tumors in other organs.
2. Modifiable risk factors
    a. Tobacco use
        i. Cigarette smoke contains many carcinogens such as polycyclic aromatic hydrocarbons
    b. Obesity
        i. Significant obesity has been shown in the literature to increase the risk of RCC. While this is a known risk factor for RCC, it is generally considered not to be as great of a risk factor as others, such as exposure to known carcinogens, familial history, etc.
        ii. While the precise pathogenesis remains unelucidated, obesity promotes resistance to insulin-like growth factor which may facilitate unregulated cell proliferation and tumor

growth. It is also thought to release inflammatory cytokines and promote the over-production of DNA damaging free radicals.

c. Poorly controlled hypertension
    i. Significantly elevated and sustained hypertension has been associated with increased risks for RCC. While this is a known risk factor for RCC, it is generally considered not to be associated with as great a risk as, for example, exposures to known carcinogens, familial history, etc.
d. Occupational/environmental exposures
    i. Known occupational chemicals associated with renal cell carcinoma include trichloroethylene, tetrachloroethylene, benzene, vinyl chloride, herbicides and cadmium.

Mr. Fancher had significant exposure to multiple toxins known to be causally associated with kidney cancer: TCE, PCE, VC and Benzene. Mr. Fancher had exposure to these toxins over an almost two-year time period. Developing kidney cancer at age 39 is not common and is indicative of an exposure to the known toxins Mr. Fancher was exposed to at Camp Lejeune.

On the other hand, in the medical records provided as well as deposition testimony, Mr. Fancher did not have evidence of any specific risk factor documented at the time of his diagnosis at age 39, nor did he have known exposure to other environmental toxins including herbicides or pesticides. The medical records do not detail a BMI or weight at the time of kidney cancer diagnosis that met the criteria for obesity and he did not have hypertension at that time. There is no evidence that any other factor unrelated to Camp Lejeune would offset the contribution of his known exposure to the contaminated water at Camp Lejeune.

Given the significantly strong correlation between the water at Camp Lejeune and kidney cancer, including at the levels that existed during the time Mr. Fancher was present at Camp Lejeune, combined with the fact that there is really no other known risk factor that would significantly increase the development of kidney cancer for Mr. Fancher, it is overwhelmingly likely that Mr. Fancher's kidney cancer was caused by the drinking water 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. Fancher'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. Fancher's kidney cancer. I have rejected all of those as well, as shown below.

XIII. 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. Fancher, each of these factors indicates a substantial exposure.

For example, Mr. Fancher was on base for a total of approximately 337 days. This is almost a full year's worth of just time on base. This exposure in terms of total number of days exposed took place over a 20-month time

period. 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. I will not repeat the analysis listed above, which describes this intensity, but will state that these levels have been shown in the literature to be incredibly hazardous and known to cause kidney cancer.

Mr. Fancher 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. Fancher 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.

My opinion that Mr. Fancher had substantial exposure is based upon Mr. Fancher's deposition, the concentrations in the water at the time Mr. Fancher was exposed and corresponding documents from Mr. Fancher'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. Fancher had substantial exposure to the toxins at Camp Lejeune and is consistent with that opinion. The charts detail a reasonable estimated dose of ingestion exposure for Mr. Fancher. Dr. Reynolds' charts are found below:

		Chart 1: 1L	**Chart 2: ATSDR**	**Chart 3: 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/FM exposure assumptions)**
TCE	5,340	92,052	422,266	577,666
PCE	247	4,263	19,535	26,742
VC	351	6,068	27,803	38,065
BZ	99	1,771	8,083	11,095

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. Fancher 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. Fancher 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. Fancher's diagnosis of kidney cancer.

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

XIV. 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 specify several causes the government thinks may be causally related to the kidney cancer Mr. Fancher developed. I have reviewed each of these potential arguments and reject each as detailed below:

1. The Government claims Mr. Fancher had a family history of throat cancer and that may be relevant to his kidney cancer diagnosis. This argument lacks merit because a family history of throat cancer is not associated with an increased risk of renal cell carcinoma. This includes major inherited RCC syndromes mentioned in this report (hereditary papillary renal carcinoma (HPRC), von Hippel-Lindau disease (VHL), Birt-Hogg-Dubé syndrome (BHD) associated with other tumors in other organs.

2. The Government claims that Mr. Fancher may have been exposed to asbestos. Mr. Fancher testified he was not aware of any exposure to asbestos and that his school district cleaned up asbestos in his school before he got there. This is given little value based on the above.

3. The Government claims that the exposure to the chemicals at Camp Lejeune may not have been sufficient to have caused Mr. Fancher'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 that are hazardous to humans generally and are known to cause kidney cancer.

4. The Government claims the length of time between exposure and diagnosis of kidney cancer may indicate an alternative cause of Mr. Fancher's kidney cancer. This argument 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.[8,23] Two of these studies even conducted sensitivity analyses with up to 20-year lags without substantive changes in results.[8,23] Analysis of these study designs using the 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.

XV. 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.[8,9,10,14,23] The Bradford Hill considerations are employed here for a structured analysis to determine whether this particular association with Mr. Fancher is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Fancher'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 David Fancher was exposed to and kidney cancer.[8,9,10,23]

b. Consistency

Consistency refers to studies being done in different populations yielding similar results. Multiple cohort[8,9,10,14] 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.[8,23] This was a consistent finding despite varied methods of determining exposure within these studies. David Fancher, 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.[8,23] Mr. Fancher was diagnosed with renal cell carcinoma at age 39, which is 18 years after the start of his 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. Fancher's exposure to the Camp Lejeune water and kidney cancer.

f. Analogy

David Fancher'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. Fancher did not have evidence of any specific risk factor documented at the time of his diagnosis at age 39, 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. Fancher's kidney cancer is considered through the Bradford Hill analysis, it is my conclusion that exposure is more likely than not a cause of kidney cancer. Given David Fancher'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. Fancher's kidney cancer for purposes of the differential diagnosis and causal relationship.

XVI. Mr. Fancher's Injuries

I will talk about Mr. Fancher's harms as a result of his kidney cancer, including the bulge on the right side of his body. This bulge began following his kidney surgery in 1997. To a reasonable degree of medical certainty, the bulge in his abdomen arose as a direct result of the surgery to remove his kidney. Additionally:

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

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

3. The Plaintiff is expected to live a normal life expectancy.

4. The medical billing relating to Mr. Fancher's kidney cancer diagnosis, the surgery to remove his kidney and the follow up treatment related to his kidney cancer was reasonable and medically necessary.

XVII. 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 his kidney cancer diagnosis.

Sincerely,

Joseph Del Pizzo, MD

CITATIONS

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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

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6. **Del Pizzo JJ**, and Sklar GN: The O'Brien peel-away sheath: An alternative for percutaneous transplant nephroscopy. *J Endourol,* 13(1): 31-33, 1999. PMID: 10102125

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11. Szostak M, **Del Pizzo JJ**, and Sklar GN: The Plug-and-patch: A new technique for repair of corporal perforation during placement of penile prostheses. *J Urol*, 163(4), 1125-7, 2000. PMID: 10737496

12. **Del Pizzo JJ**, Shichman SJ, and Sosa RE.: Laparoscopic Adrenalectomy: The New York-Presbyterian Experience. *J Endourol*, 16(8): 1-7, 2002. PMID: 12470468

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

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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	)	
	)	
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 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.