

Exhibit 385



Specific Causation Expert Report: Allan Wayne Howard

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Signed by:

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February 3, 2025

Re: Allan Wayne Howard
DOB: [REDACTED]/1959

I am writing this letter in response to your request to provide a medical expert evaluation of the records of Alan Wayne Howard 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., < 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. Howard'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 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. Howard'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. Howard'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. Howard and finally made a determination as to whether those risk factors were causally related to Mr. Howard'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 level of exposure at Camp Lejeune to these toxins were 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 Allan Wayne Howard's kidney cancer. This exceeds the "at least as likely as not" standard required in this case. Further, it is my opinion Alan Howard 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

Allan Wayne Howard was a member of the armed services, USMC from 1977 through 1981. He was stationed at Camp Lejeune from September 1977 through February 1979, including living at the Mainside Barracks supplied by Hadnot Point distribution system.

In October 2008, aged 49, he was evaluated for elevated serum liver function tests. He initially underwent abdominal sonogram, which identified a complex mass involving his right kidney. A subsequent CT scan revealed a 4.2-centimeter solid enhancing mass involving the right kidney and multiple bilateral pulmonary parenchymal and pleural-based nodules. MRI of the head and nuclear bone scan were both negative.



Due to the presence of the pulmonary nodules, he was seen in consultation by Thomas Olencki, DO at the Ohio State University (OSU) Wexner Medical Center Division of Medical Oncology. He was referred to Dr. Ahmad Shabsigh and underwent open right radical nephrectomy at Ohio State University Hospital on December 3, 2008. His post operative recovery was unremarkable. Surgical pathology was consistent with a 4.2-centimeter clear cell renal cell carcinoma with focal involvement of the renal capsule Pathologic stage (T1b). Initially planned for adjuvant immunotherapy via a Mediport, his chest CT scans remained stable and radiologic surveillance was recommended. Since that time, he has had no evidence of recurrent or metastatic disease relating to renal cell carcinoma. His overall kidney function has been stable, and his serum creatinine in May 2024 was 1.0mg/dl.

He suffered a pulmonary embolism in 2020 after a pelvic fracture sustained in a bicycle accident and was placed on anti-coagulation (apixaban). He was subsequently diagnosed with B cell non-Hodgkins lymphoma in 2023, aged 64. He was treated at Kettering Cancer Center in Kettering, Ohio, with the drug combination Polatuzaman vedotin, Rituximab, Cyclophosphamide, Doxorubicin, and Prednisolone (Pola-R-CHP).

He has a history of basal cell carcinoma of the skin diagnosed in 2008. He had a tubular adenoma removed during colonoscopy screening in 2017. He has left renal cysts, pulmonary nodules and mild ascending aortic aneurysmal dilatation (4.2 cm in diameter) for which he receives routine radiologic surveillance. He also has a history of hypothyroidism and hyperlipidemia. He has a distant history of short-term tobacco use, with a two pack-year history while in the USMC. Record indicate his mother may have had a history of skin cancer, and his maternal aunt may have had a reported history of colon cancer, although Mr. Howard is not able to confirm this.

After discharge from the USMC, he worked as a police office in Dayton for 26 years, retiring in 2008. At the time of his kidney cancer diagnosis, he was a long-distance runner, completing up to 10 miles daily, and weighed 156 pounds.

VI. Factual History

During his deposition, Mr. Howard testified that he was stationed at Camp Lejeune from September 1977 through February 1979, other than periods of annual leave and a deployment for less than a month in January of 1978. He lived at the Mainside Barracks and testified that he showered primarily at the Mainside Barracks within an open shower area with individual shower heads and no windows. He showered at least once per day, more often on certain days based upon level of physical training. He also testified that in addition to his personal showers, he would clean his machine gun weekly using hot water in the shower for 30-45 minutes. 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 Mainside Barracks. He testified he would have two glasses of water or Kool-Aid with each meal and testified that when he was training, he would drink 6-7 canteens of water a day.

These facts provide the basis for the opinion that Mr. Howard's exposure was substantial. The facts indicate Mr. Howard 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. Howard'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 effect that his renal cell carcinoma diagnosis has had on himself, his family, including his children and friends. He continues to be active and lead a healthy lifestyle with daily biking and exercise but remains concerned for possible recurrence or the diagnosis of another primary cancer. This concern has intensified since his more recent diagnosis of B cell non-Hodgkins lymphoma in 2023.

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 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/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 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 $\geq 50\%$ 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 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 et 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.

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. Howard'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. Howard meets the criteria for the medium exposure categories for each of the individual chemicals and also TVOC exposure in this study.



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. Howard had similar exposure.

Dr. Hatten states in his 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 Allan Wayne Howard's Renal Cell Carcinoma

There are risk factors linked to 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 also 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 were the cause of the Mr. Howard's kidney cancer. Based upon the review of Mr. Howard's medical records, his time stationed at Camp Lejeune, and review of the scientific and epidemiological evidence, it is my opinion that it is as 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. Howard 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) Allan Wayne Howard was stationed at the Mainside Barracks for 449 days from September 4, 1977 through February 8, 1979 (not including time away from the base for annual leave and a deployment to the Caribbean from January 31 through March 7, 1978). This included time where he lived at the Mainside Barracks. The soldiers and civilian personnel at Camp Lejeune typically experienced multiple routes of exposure. In his deposition testimony, Mr. Howard stated that he continued to eat and hydrate on the base daily, as well as shower there a minimum of once per day for five to ten minutes. Scientific



studies have demonstrated all three routes of exposure are significant in contributing to overall cancer risk.

- (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 September 1977 through February 1979 was as follows in ug/L-months:

Date	Total Days	TCE (ug/L)	PCE (ug/L)	Vinyl chloride (ug/L)	Benzene (ug/L)
9/4-9/30/77	27	338	13	18	4
10/1-10/31/77	31	69	2	3	4
11/1-11/18/77, 11/29-11/30/77	20	544	22	30	4
12/1-12/31/77	31	513	21	28	4
1/1-1/31/78	31	250	10	14	4
3/7-3/31/78	25	352	15	20	3
4/1-4/30/78	30	231	9	13	5
5/1-5/30/78	31	278	12	16	4
6/1-6/30/78	30	333	14	19	3
7/1-7/13/78 7/29-7/31/78	16	388	17	23	3
8/1 -8/31/78	31	475	20	28	4
9/1-9/30/78	30	364	16	22	4
10/1-10/31/78	31	74	3	4	4
11/1-11/30/78	30	544	24	33	5
12/1-12/21/78	28	546	24	33	4
1/13-1/31/79	19	268	12	16	6
2/1-2/8/79	8	370	17	23	5
Total	449	5,937	251	343	70

The median level of these contaminants in the water during this time period was 352 ug/L for TCE, 15 ug/L for PCE, 20ug/L for VC and 4 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. Allan Wayne Howard, 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. Howard was likely exposed to the following amounts of the four primary chemicals in the water at Camp Lejeune: TCE: 5937 ug/l-M, PCE: 251ug/l-M, VC: 343 ug/l-M and benzene: 70 ug/l-M. Mr. Howard'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. Howard can be categorized within the low/medium group (1-5 quarters, HR 1.36).¹⁰ Mr. Howard was exposed to the water at Camp Lejeune for approximately 449 days over approximately 17 months. Mr. Howard'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. Howard was found to have a right renal mass subsequently diagnosed as renal cell carcinoma at age 49, 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.^{8,23} Two of these studies even conducted sensitivity analyses with up to 20-year lags without substantive changes in results.^{8,23}

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). Except for 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.

In the medical records provided as well as deposition testimony, Mr. Howard did not have evidence of obesity, hypertension, or relevant family history/genetic syndromes documented at the time of his diagnosis at age 49. He did provide a brief history of tobacco use (two pack-year) while in the USMC but quit thirty years prior to his diagnosis of renal cell carcinoma. 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.²⁵ He does describe responding, as a member of the Dayton police, to a fire after a train derailment, with possibility of exposure to burning phosphorus. This is not known to be significantly carcinogenic in humans or animals, and 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. Howard had exposure to these toxins over a seventeen-month time period 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. Howard was present at Camp Lejeune, combined with a very low dose of tobacco use over a short period of time that stopped thirty years prior to his diagnosis of renal cell carcinoma, it is more likely than not that Mr. Howard'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. Howard'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. Howard'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. Howard, each of these factors indicates a substantial exposure.

For example, Mr. Howard was on base for a total of approximately 449 days. This is well over a year's worth of time on base. This exposure in terms of total number of days took place over a 17-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. Howard 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. Howard 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. Howard's exposure to the water at Camp Lejeune was substantial.

My opinion that Mr. Howard had substantial exposure is based upon Mr. Howard's deposition, the concentrations in the water at the time Mr. Howard was exposed and corresponding documents from Mr. Howard'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. Howard had substantial exposure to the toxins at Camp Lejeune and are consistent with these opinions. The charts detail a reasonable estimated dose of ingestion exposure for Mr. Howard. 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	5,937	153,943	668,552	660,782	1,019,982
PCE	251	6,472	28,107	27,780	42,882
VC	343	8,859	38,473	38,026	58,697
BZ	70	1,831	7,952	7,859	12,132

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. Howard 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. Howard 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. Howard’s diagnosis of kidney cancer.

I use these charts to add weight to the differential diagnosis analyses above and to opine Mr. Howard’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 specific several causes that the government thinks may be causally related to the kidney cancer Mr. Howard developed. I have reviewed each of these potential arguments and reject each as detailed below:

1. The Government claims Mr. Howard had a personal and family history of basal cell carcinoma of the skin and that may be relevant to his kidney cancer diagnosis. This argument lacks merit because there is no causal relationship between basal cell carcinoma of the skin and renal cell carcinoma. Some genetic mutations associated with an increased risk of basal cell carcinoma can be linked to an increased risk of renal cell carcinoma in syndromes such as hereditary leiomyomatosis and renal cell cancer (HLRCC), which does not apply to Mr. Howard and his known medical history.



2. The Government claims Mr. Howard may have been a smoker at one time 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.²⁵ Mr. Howard had a very limited smoking history (two pack-years) and quit approximately thirty years prior to his diagnosis of renal cell carcinoma.
3. The Government claims Mr. Howard may have had other chemical exposures and that may be relevant to his kidney cancer diagnosis. This argument lacks merit because he describes a possible exposure to burning phosphorous during a train derailment in 1986. This is not known to be significantly carcinogenic in humans or animals, and he has no other known exposure to other environmental toxins such as herbicides or pesticides. Further, Mr. Howard stated in his deposition that he did not have any chemical exposure in Beruit, as Defendants have postulated, and that he wasn't deployed in Beruit. There is no actual evidence of any exposure in Beruit, and so therefore that is given little weight. Finally, defendants have stated that motorcycle maintenance may have been a factor in Mr. Howard's kidney cancer. This is not accurate because as a general proposition this would be a very low exposure given the frequency of Mr. Howard's activity with motorcycles. Mr. Howard further testified that he uses protective gear and low toxicity cleaners.
4. The Government claims the exposure to the chemicals at Camp Lejeune may not have been sufficient to have caused Mr. Howard'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.
5. The Government claims that the length of time between exposure and diagnosis of kidney cancer may indicate an alternative cause of Mr. Howard'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. Howard is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Howard'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 Alan Wayne Howard 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^{8,9,10,23} and case control¹⁴ 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. Alan Wayne Howard, 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 to the diagnosis of kidney cancer.^{8,23} Mr. Howard was diagnosed with renal cell carcinoma at age 49, which is 29 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. Howard's exposure to the Camp Lejeune water and kidney cancer.

f. Analogy

Alan Wayne Howard'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. Howard did not have evidence of any specific risk factor documented at the time of his diagnosis at age 49, 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. Howard'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 Alan Wayne Howard'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. Howard's kidney cancer for purposes of the differential diagnosis and causal relationship.

XVI. Mr. Howard's Injuries

I will talk about Mr. Howard's harms as a result of his kidney cancer, including the treatment that was required, the surgeries required and the impacts that his kidney cancer diagnosis had on Mr. Howard. Additionally:

1. The harms and injuries and damages suffered by Mr. Howard that are described in this report are permanent.
2. The treatment and care Mr. Howard 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. Howard'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 Mr. Howard's kidney cancer diagnosis.

Sincerely,

Joseph Del Pizzo, MD

CITATIONS

- ¹ Agency for Toxic Substances and Disease Registry. ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases. 2017:1-150.
- ² International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. 2014;106:1-514.
- ³ Andrew AS, Li M, et al. Kidney Cancer Risk Associated with Historic Groundwater Trichloroethylene Contamination. *Int. J. of Environmental Research & Public Health*. 2022;19:618. <https://doi.org/10.3390/ijerph19020618>.
- ⁴ Moore LE, Boffetta P, et al. Occupational Trichloroethylene Exposure and Renal Carcinoma Risk: Evidence of Genetic Susceptibility by Reductive Metabolism Gene Variants. *Cancer Res*. 2010;70(16):6527-6536.
- ⁵ Purdue MP, Rhee J, et al. Differences in risk factors for molecular subtypes of clear cell renal cell carcinoma. *Int. J. Cancer*. 2021;149(7):1448-1454. doi:10.1002/ijc.33701.
- ⁶ Karami S, Lan Q, et al. Occupational trichloroethylene exposure and kidney cancer risk: a meta-analysis. *Occup. Environ. Med*. 2012;69:858-867. doi:10.1136/oemed-2012-100932.
- ⁷ Kelsh MA, Alexander DD, et al. Occupational trichloroethylene exposure and kidney cancer: a meta-analysis. *Epidemiology*. 2010;21(1):95-102. doi: 10.1097/EDE.0b013e3181c30e92
- ⁸ Bove FJ, Ruckart PZ, et al. Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study. *Environmental Health*. 2014. <http://www.ehjournal.net/content/13/1/10>.
- ⁹ Bove FJ, Greek A, Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study. *Environmental Health Perspectives*. 132(1). <https://doi.org/10.1289/EHP14966>.
- ¹⁰ Bove FJ, Greek A, Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study. *Environmental Health*. 2024(23):61. <https://doi.org/10.1186/s12940-024-01099-7>.
- ¹¹ Environmental Protection Agency. Biden-Harris Administration Announces Latest Actions under Nation's Chemical Safety Law to Protect People from Cancer-Causing Chemicals Trichloroethylene and Perchloroethylene. December 9, 20214. <https://www.epa.gov/newsreleases/biden-harris-administration-announces-latest-actions-under-nations-chemical-safety-law>.
- ¹² International Agency for Research on Cancer. Chemical Agents and Related Occupations. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. 2012;100F:1-599.



- ¹³ Aschengrau A, Ozonoff D, et al. Cancer Risk and Tetrachloroethylene-contaminated Drinking Water in Massachusetts. *Archives of Environmental Health*. 1993;48(5):284-292.
- ¹⁴ Agency for Toxic Substances and Disease Registry. Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune. 2018:1-126.
- ¹⁵ Purdue MP, Stewart PA, et al. Occupational exposure to chlorinated solvents and kidney cancer: a case-control study. *Occup. Environ. Med*. 2017;74(4):268-274. doi:10.1136/oemed-2016-103849.
- ¹⁶ Ruder A, Ward E, et al. Mortality in dry-cleaning workers: An update. *Am. J. Ind. Med*. 2001;39(2):121-132. doi: 10.1002/1097-0274(200102)39:2<121::aid-ajim1000>3.0.co;2-h.
- ¹⁷ Hu J, Mao Y, et al. Renal cell carcinoma and occupational exposure to chemicals in Canada. *Occup. Med*. 2002;52(3):157-164.
- ¹⁸ Greenland S, Salvan A, et al. A case-control study of cancer mortality at a transformer-assembly facility. *Int. Arch Occup. Environ. Health*. 1994;66:49-53.
- ¹⁹ Seyyedsalehi MS, Bonetti M, et al. Occupational benzene exposure and risk of kidney and bladder: a systematic review and meta-analysis. *European Journal of Cancer Prevention*. 2024. doi: 10.1097/CEJ.0000000000000911.
- ²⁰ Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011.
- ²¹ National Toxicology Program. Report on Carcinogens Monograph on Trichloroethylene (RoC Monograph 05). 2015:1-236.
- ²² Scott CS & Jinot J. Trichloroethylene and Cancer: Systematic and Quantitative Review of Epidemiologic Evidence for Identifying Hazards. *International Journal of Environmental Research and Public Health*. 2011;8:4238-4272.
- ²³ Bove FJ, Ruckart PZ, et al. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environmental Health*. 2014. <http://www.ehjournal.net/content/13/1/68>.
- ²⁴ Jakobsson RG, Nasic S, et al. Family History and Risk of Renal Cell Carcinoma: A National Multiregister Case-Control Study. *The J. of Urology*. 2024;211:71-79. <https://doi.org/10.1097/JU.0000000000003765>.
- ²⁵ Hunt J, Van der Hel OL, et al. Renal cell carcinoma in relation to cigarette smoking: Meta-analysis of 24 studies. *Int. J. Cancer*. 2005;114:101-108.

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 2009-2017
Director, Laparoscopic Living Kidney Donor program
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
New York-Presbyterian Hospital, Cornell
New York, NY

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

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

2017-present

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

2012-present

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:
New York-Presbyterian Hospital

E. PROFESSIONAL MEMBERSHIPS (medical and scientific societies)

Member	American Urological Association	2001-present
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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 <i>The New York Experience of Robot-Assisted Pyeloplasty.</i> 13 th International Congress of Laparoendoscopic Surgeons, SLS Annual Meeting New York, New York	2004
Honorable Mention – Best Scientific Paper <i>Laparoscopic live donor nephrectomy: Donor tolerance and renal allograft outcomes stratified by age.</i> 13 th International Congress of Laparoendoscopic Surgeons, SLS Annual Meeting New York, New York	2004
First place – Best Urology Paper <i>Minimizing the incidence of vascular complications during right sided laparoscopic live donor nephrectomy</i> 12 th 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: New York 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 Clinical Care	7/01-present

Director, Advanced Minimally Invasive Urology/Kidney Donor Program Responsibility for resident education and skill development In minimally invasive laparoscopic renal surgery including donor nephrectomy for renal transplantation and extirpative and reconstructive surgery for renal disease	7/01-present
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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
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Clinical research and maintenance 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 <i>Current Urology Reports</i> , Adrenal Diseases Section	2004-present
--	--------------

Manuscript Peer Review <i>Journal of Urology, Journal of Endourology, British Journal of Urology, Journal of Transplantation</i>	2004-present
Faculty Instructor American Urological Association Annual Meeting <i>Introduction to Laparoscopy Course</i>	2002-2012
Faculty Instructor American Urological Association Office of Education Houston, TX <i>AUA Hand Assisted Laparoscopy Course</i>	2001-2010
Course Director Brady Urologic Associates/Weill Cornell Medical College New York, NY <i>Single Incision Laparoscopy Course</i>	2009-2014

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
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
7. Sigman DB, **Del Pizzo JJ**, and Sklar GN: Endoscopic retrograde stenting for transplant hydronephrosis. *J Endourol*, 13(1): 21-25, 1999. PMID: 10102123
8. Sigman DB, Hasnain JU, **Del Pizzo JJ**, and Sklar GN: Real time trans-esophageal echocardiography for intraoperative surveillance of patients with renal cell carcinoma with venal caval extension undergoing radical nephrectomy. *J Urol*, 161(1): 36-38, 1999. PMID:10037362

9. **Del Pizzo JJ**, Sklar GN, Levin B, Wong-You-Cheong JJ, Cho E, Flowers JL, and Jacobs SC: Helical computerized tomography angiography for evaluation of living renal donors undergoing laparoscopic nephrectomy. *J Urol*, 62(1): 31-35, 1999. PMID: 10379733
10. **Del Pizzo JJ**, Borkowski A, Jacobs SC, and Kyprianou N: Loss of cell cycle regulators p27^{Kip1} and cyclin E in transitional cell carcinoma of the bladder correlates with tumor grade and patient survival. *Am J Pathol*, 155(4): 1129-1136, 1999 PMID: 10514396
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
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
16. Munver R, **Del Pizzo JJ**, Sosa RE: The evolution and current applications of hand-assisted laparoscopy. *Contemporary Urology*, 15(10):30-58, 2003.
17. Munver R, **Del Pizzo JJ**, Sosa RE: Adrenal-preserving minimally invasive surgery: The role of laparoscopic partial adrenalectomy, cryosurgery, and radiofrequency ablation of the adrenal gland, *Curr Urol Rep*, 81-8687-92, 2003.
18. Stifelman MD, Handler T, Neider AM, **Del Pizzo JJ**, Sosa RE and Shichman SJ: Hand-assisted Laparoscopy for large renal specimens: A multi-institutional study. *Urology*, 61: 78-82, 2003 PMID: 12559271
19. Scherr DS, Ng C, Munver R, Sosa RE, Vaughan, ED, **Del Pizzo, JJ**: Practice patterns among urologic surgeons treating localized renal cell carcinoma in the laparoscopic age: Technology versus Oncology. *Urology*, 62(6): 1007-12, 2003. PMID: 14665345
20. Munver R, Palese MA, Sosa RE and **Del Pizzo JJ**. Laparoscopic Live Donor Nephrectomy: Donor and Recipient Outcomes Stratified by Age. *JSLs*; 8(3):37-41 2004.
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
23. Munver R, **Del Pizzo JJ**, Sosa RE: Hand-assisted laparoscopic nephroureterectomy for upper tract transitional cell carcinoma. *J Endourol*, 18(4): 351-8, 2004. PMID:15253785
24. Munver R, Sosa RE, **Del Pizzo JJ**: Laparoscopic pyeloplasty: History, evolution and the future. *J Endourol* , 18(8):748-55, 2004. PMID: 15659895
25. Boorjian S, Ng C, Munver R, Palese MA, Sosa RE, Vaughan ED, **Del Pizzo JJ**, Scherr DS. Abnormal selective cytology results predict recurrence of upper-tract transitional-cell carcinoma treated with ureteroscopic laser ablation. *J Endourol*, 18(9): 912-6, 2004. PMID:15659932
26. Munver R, Palese MA, Sosa RE, Richstone L, Boorjian S, and **Del Pizzo JJ**. Renal allograft outcomes following laparoscopic live donor nephrectomy: Analysis and stratifications by donor age. *J Endourol.*;18(1) 20-01, 2004.
27. Palese MA, Stifelman ME, Munver R, Sosa RE, Philipps CK, Dinlec C, and **Del Pizzo JJ**: Robot-Assisted Laparoscopic Dismembered Pyeloplasty: A Combined Experience. *J Endourol*, 19(3):382-386, 2005. PMID: 15865532
28. **Del Pizzo JJ**, Schiff, JD, and Vaughan, ED: Laparoscopic adrenalectomy for pheochromocytoma. *Curr Urol Rep* 6(1):78-85, 2005. PMID: 15610701
29. Palese MA, Munver R, Philipps CK, Dinlec C, Stifelman ME, and **Del Pizzo JJ**: Robot-Assisted Laparoscopic Dismembered Pyeloplasty. *JSLS*, 9(3):252-7, 2005.
30. Schiff JD, Palese MA, Vaughan ED, Sosa RE, Coll DE, **Del Pizzo JJ**: Laparoscopic vs open partial nephrectomy in consecutive patients: the Cornell experience. *BJU Int*. Oct; 96(6):811-4, 2005. PMID: 16153207
31. Boorjian S, Ng C, Munver R, Palese MA, Vaughan ED, Sosa RE, **Del Pizzo JJ**, Scherr DS: Impact of delay to nephroureterectomy for patients undergoing ureteroscopic biopsy and laser tumor ablation of upper tract transitional cell carcinoma. *Urology*, 66(2):283-7, 2005. PMID: 16098357
32. Raman JD, Palese MA, Ng CK, Boorjian SA, Scherr DS, **Del Pizzo JJ**, Sosa RE: Hand-assisted laparoscopic nephroureterectomy for upper urinary tract transitional cell carcinoma. *JSLS*, 10(4):432-8, 2006. PMID:17575752
33. Rosoff J, Raman J, **Del Pizzo JJ**: Feasibility of the laparoscopic approach in management of xanthogranulomatous pyelonephritis. *Urology*,68(4):711-4, 2006. PMID: 17070338
34. Barocas D, Rohan SM, Kao J, Gurevich RD, **Del Pizzo JJ**, Vaughan ED, Akhtar M, Chen, YT, Scherr DS: Diagnosis of renal tumors on needle biopsy specimens by histological and molecular analysis. *J Urol*, 176(5), 1957-62,2006. PMID: 17070218

35. Barocas D, Mathew S, **Del Pizzo JJ**, Vaughan ED, Sosa RE, Fine, RG, Akhtar M, Scherr DS. Renal cell carcinoma sub-typing by histopathology and fluorescence in situ hybridization on a needle-biopsy specimen. *BJU Int.* Feb; 99(2): 290-5, 2007.
36. Volfson IA, Munver R, Esposito M, Dakwar G, **Del Pizzo JJ** and Stock JA: Robotic assisted urologic surgery: safety and feasibility in the pediatric population. *J Endourol*, 21(11): 1315-18, 2007.
37. Brophy RH, Gamradt SC, Barnes RP, Powell JW, **Del Pizzo JJ**, Rodeo SA and Warren RF: Kidney injuries in professional American football: implications for management of an athlete with one functioning kidney. *Am J Sports Med*, 85-90, Nov 2007 PMID: 17986635
38. Mezitis SG, Gellar M, Bocchieri, E, **Del Pizzo, JJ**, and Merlin S: Association of pheochromocytoma and ganglioneuroma: unusual finding in neurofibromatosis type 1. *Endocr Pract*, 13(6): 647-51, 2007. PMID: 17954422
39. Rosoff, JS, Raman JD, **Del Pizzo JJ**: Laparoscopic Adrenalectomy for Large Adrenal Masses. *Curr Urol Rep*, 9(1):73-79, 2008. PMID: 18366978
40. Butt FK, Gritsch HA, Schulam P, Danovitch GM, Wilkinson A, **Del Pizzo JJ**, Kapur S, Serur D, Katznelson S, Busque S, Melcher ML, McGuire S, Charlton M, Hil G, Veale JL. Asynchronous, out-of-sequence, transcontinental chain kidney transplantation: a novel concept. *Am J Transplant*, 9(9), 2180-85, 2009 PMID: 1956335
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
42. Lauer E, **Del Pizzo JJ**, Raman JD: Needleoscopic ablation of small adrenal masses. *Curr Urol Rep*, 10(1), 73-77, 2009. PMID: 19116099
43. Cheng EY, Leeser DB, Kapur S, **Del Pizzo JJ**: Outcomes of laparoscopic donor nephrectomy without intraoperative systemic heparinization. *J Urol* 183(6), 2282-6, 2010. PMID: 20400133
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

48. Cha EK, Ng CK, Jeun B, Dunning A, Reifsnyder JE, DiPietro JR, Mazumdar M, Shih G, Auh YH, **Del Pizzo JJ**, Shariat SF, Scherr DS. Preoperative radiographic parameters predict long-term renal impairment following partial nephrectomy. *World J Urol* 2011. PMID: 21604019
49. Herman MP, **Del Pizzo JJ**. Simultaneous Bilateral Single Port Radical Nephrectomies. *JLSLS*. 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
53. Wang GJ, Afaneh C, Aull M, Charlton M, Ramasamy R, Leeser DB, Kapur S, **Del Pizzo JJ**. Laparoendoscopic single site live donor nephrectomy: single institution report of initial 100 cases. *J Urol*. 186(6):2333-7, 2011 PMID: 22014813
54. Afaneh C, Sheth S, Aull MJ, Leeser DB, **Del Pizzo JJ**. Laparoendoscopic Single Site Nephrectomy in Obese Living Renal Donors. *J Endourol*. 2011 PMID:22050506
55. Afaneh C, Ramasamy R, Aull MJ, Leeser DB, Sosa RE, Kapur S and **Del Pizzo JJ**. The Evolution of Laparoscopic Right Donor Nephrectomy: Progression to Single Site Surgery. *Transplantation technologies and Research*. Volume 1, Issue 2, 2011.
56. 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*. 2011 Oct;186(4):1386-90.
57. Reifsnyder JE, Ramasamy R, Ng CK, Dipietro J, Shin B, Shariat SF, **Del Pizzo JJ**, Scherr DS. Laparoscopic and open partial nephrectomy; complication comparison using the Clavien system. *JLSLS*. 2012 Jan-Mar;16(1):38-44. PMID:22906328
58. Leeser DB, Aull MJ, Afaneh C, Dadhania D, Charlton M, Walker JK, Hartono C, Serur D, **Del Pizzo JJ**, Kapur S. Living donor kidney paired donation transplantation: experience as a founding member center of the National Kidney Registry. *Clin Transplant*. 2012 May-Jun;26(3):E213-22. Epub 2012 Mar 12. PMID:22872872
59. Balachandran VP, Aull MJ, Charlton M, Afaneh C, Serur D, Leeser DB, **Del Pizzo JJ**, Kapur S. Kidneys from older living donors provide excellent intermediate-term outcomes after transplantation. *Transplantation*. 2012 Sep 15;94(5):499-505. PMID: 22892992

60. Aull MJ, Dadhania D, Afaneh C, Leeser DB, Hartono C, Lee JB, Serur D, **Del Pizzo JJ**, Suthanthiran M, Kapur S. Early corticosteroid withdrawal in recipients of renal allografts; a single-center report of ethnically diverse recipients and recipients of marginal deceased-donor kidneys. *Transplantation*. 2012 Oct 27;94(8):837-44. PMID 23001353
61. Cha EK, Ng CK, Jeun B, Dunning A, Reifsnnyder JE, DiPietro JR, Mazumdar M, Shih G, Auh YH, **Del Pizzo JJ**, Shariat SF, Scherr DS. Preoperative radiographic parameters predict long-term renal impairment following partial nephrectomy. *World J Urol*. (4):817-22, 2013. PMID: 21604019
62. Rosoff JS, Fine RG, Velez MC, **Del Pizzo JJ**. Laparoendoscopic single-site radical nephrectomy for large renal masses. *J Endourol*, 27(1):34-9, 2013 PMID:22984849
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
64. Aull MJ, Afaneh C, Charlton M, Serur D, Douglas M, Christos PJ, Kapur S, **Del Pizzo JJ**. A randomized, prospective, parallel group study of laparoscopic versus laparoendoscopic single site donor nephrectomy for kidney donation. *Am J Transplant*. 14(7):1630-7, 2014 PMID: 24934732
65. Seklehner S, Laudano MA, Jamzadeh A, **Del Pizzo JJ**, Chughtai B, Lee RK. Trends and inequalities in the surgical management of ureteric calculi in the USA. *BJU Int*. 2014 Mar;113(3):476-83, 2013 PMID: 24053734.
66. Schiffman M, Moshfegh A, Talenfeld A, **Del Pizzo JJ**. Laparoscopic renal cryoablation. *Semin Intervent Radiol*. 31(1):64-9, 2014. PMID: 24596441
67. Seklehner S, Laudano MA, **Del Pizzo JJ**, Chughtai B, Lee RK. Renal calculi: trends in the utilization of shockwave lithotripsy and ureteroscopy. *Can J Urol*. 22(1):7627-34, 2015. PMID: 25694010.
68. Chughtai B, Scherr D, **Del Pizzo JJ**, Barbieri C, Kaplan SA, Schlegel PN, Sedrakyan A. National Trends and Cost of Minimally Invasive Surgery in Urology. *Urol Prac* 2(2), 49-54, 2015.
69. Chen B, Finnerty BM, Schamberg NJ, Watkins AC, **DelPizzo JJ**, Zarnegar R. Transabdominal robotic repair of a congenital right diaphragmatic hernia containing an intrathoracic kidney: a case report. *J Robot Surg*. 2015 Dec;9(4):357-60, 2015 PMID: 26530841
70. Golumbos DM, Chughtai B, Trinh QD, Mao J, O'Malley P, Scherr DS, **Del Pizzo JJ**, Hu JC, Sedrakyan A. Adoption of Technology and its impact on Nephrectomy Outcomes, a U.S. Population-Based Analysis (2008-2012). *J Endourol*. 31(1) 91-99. 2017, PMID:27809567

71. Veale JL, Capron AM, Nassiri N, Danovitch G, Gritsch HA, Waterman A, **Del Pizzo JJ**, Hu JC, Pycia M, McGuire S, Charlton M, Kapur S. Vouchers for Future Kidney Transplants to Overcome “Chronological Incompatibility” Between Living Donors and Recipients. *Transplantation*. PMID:28333861
72. Golumbos DM, Chughtai B, Trinh QD, Thomas D, Mao J, Te, A, O’Malley P, Scherr DS, **Del Pizzo JJ**, Hu JC, Sedrakyan A. Minimally invasive vs open nephrectomy in the modern era: does approach matter? *World J Urol*, 2017.PMID: 28477204
73. Van de Mijl JC, Al Hussein Al Awamlh B, Khan AI, Posada-Calderon L, Oromendia C, Fainberg, J, Alshak M, Elahjji R, Pierce H, Taylor B, Gudas LJ, Nanus DM, Molina AM, **Del Pizzo JJ**, Scherr DS. (2019) Validation of risk factors for recurrence of renal cell carcinoma” Results from a single institution series. *PLOS ONE* 2019 Dec 9;14(12): e0226285. <https://doi.org/10.1371/journal.pone.0226285> PMID: 31815952
74. McClure T, Sedrakyan A, LaRussa S, Sun T, Mao J, **Del Pizzo JJ**, Hu J. Underutilization of Renal Mass Biopsy: Surveillance using the Medicare Database between 2004 and 2016. *J Vasc Interv Radiol*. 2020 May;31(5):854-857. doi: 10.1016/j.jvir.2019.11.023. Epub 2020 Apr 15. PMID 32305241.
75. Ma LX, Craig KM, Mosquera JM, Robinson BD, Scherr DS, **Del Pizzo JJ**, McClure TD, Khani F: Contemporary Results and Clinical Utility of Renal Mass Biopsies in the Setting of Ablative Therapy: A single center experience. Ms. No. CTC-D-20-00246R1. *Cancer Treatment and Research Communications*, 2020. PMID: 32979705
76. Friedlander DF, Brant A, McClure TD, **Del Pizzo JJ**, Nowels MA, Trinh QD, Sedrakyan A, Chughtai B: Real world comparative effectiveness of shockwave lithotripsy versus ureterorenoscopy for the treatment of urinary stones. *World J Urol*, 2020 Sept 9. PMID: 32909172
77. Sultan S, Finn C, Craig-Schapiro R, Aull MJ, Watkins A, Kapur S, **Del Pizzo JJ**. Simultaneous Living-Donor Kidney Transplant and Laparoscopic Native Nephrectomy: An Approach to Kidney Transplant Candidates with Suspected Renal Cell Carcinoma. *J Endourol*. 2020 Dec 31. doi: 10.1089/end.2020.0841. Epub ahead of print. PMID: 33238756.
78. Wilcox Vanden Berg RN, Calderon LP, LaRussa S, Enobakhare O, Craig K, **Del Pizzo JJ**, McClure TD. Microwave ablation of cT1a renal cell carcinoma: oncologic and functional outcomes at a single center. *Clin Imaging*. 2021 Aug;76:199-204. doi: 10.1016/j.clinimag.2021.04.016. Epub 2021 May 3. PMID: 33964597.
79. Zhu A, Abrahimi P, Scherr DS, **Del Pizzo JJ**. Using the Appendix as a Ureteral Substitute During Pyeloplasty in a Patient with Ureteropelvic Junction Obstruction. *Surgery: Case Reports* Vol 6No 2, March 2022.
80. Zhang TR, Thorogood SL, Miyauchi J, **Del Pizzo, JJ**, Schlegel PS. Acute testicular infarction in the setting of SARS-Cov-2 infection and diabetic vasculopathy. *Urol Case Rep* 2023 March; 47:102342. PMID: 36748071

Books, Book Chapters and Invited Reviews

1. Moldwin, R. and **Del Pizzo, JJ.**: Problems Associated with Outpatient Antibiotic Usage. In: *Antibiotic Therapy in Urology*; Lippincott-Raven Publishers, Philadelphia; 119-135, 1996.
2. **Del Pizzo, JJ**, and Jarow, JP: Management of Male Infertility. In: *Urology for Primary Care Physicians*, W.B. Saunders Publishers, Philadelphia; 335-348, 1999.
3. Vaughan, ED, Blumenfeld, J, **Del Pizzo, JJ**, et al.: Diagnosis and Management of Adrenal Disorders. In: *Campbell's Urology*, Eighth Edition, W.B. Saunders Publishers, Philadelphia, pp3507-3559, 2002.
4. **Del Pizzo, J.J.**: Getting Started in Laparoscopy. In *Essential Urologic Laparoscopy: The Complete Clinical Guide*, Edited by: SY Nakada, Humana Press Inc, New Jersey, 1-8, 2003.
5. Milowsky, MI, **Del Pizzo, J.J.**, Nanus, D.M.: Kidney cancer. In *Atlas of Cancer*, M Markman (Ed), Lippincott Williams and Wilkins, Philadelphia, 443-449, 2003.
6. **Del Pizzo, J.J.**: Pleural Injury During Urologic Laparoscopic Surgery: recognition, management and prevention. In: *Complications of Urologic Laparoscopic Surgery*, Edited by: S Ramakumar and TW Jarrett, Marcel Dekker, Inc, New York, 83-92, 2005.
7. **Del Pizzo, JJ**: Laparoscopic Donor Nephrectomy: Technique. In: *Textbook of Laparoscopic Urology*, Edited by IS Gill, Informa Healthcare, New York, 337-367,2006.
8. **Del Pizzo, JJ**, Palese, MA, Munver, R, Poppas, DP: Robotic-assisted pyeloplasty in Adult and Pediatric Population. In: *Textbook of Laparoscopic Urology*, Edited by IS Gill, Informa Healthcare, New York, 303-312,2006.
9. **Del Pizzo JJ**: Miscellaneous Adult Robotic Surgery. In: *Robotics in Urologic Surgery*, Edited by JA Smith and AK Tewari, Saunders Elsevier, Philadelphia, 141-7, 2008.
10. Afaneh, C, Aull MJ, **Del Pizzo JJ**, Kapur SA: Surgical Advances in Laparoscopic Donor Nephrectomy. In: *Current Concepts in Kidney Transplantation*, Edited by Sandip Kapur, InTech Publishing, 273-85, 2012.

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.