# Exhibit 414

# Specific Causation Expert Report for Patient Jaqueline Tukes

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## 1. Introduction

This report evaluates the cause of the renal cell carcinoma (RCC) in Plaintiff Jaqueline Tukes, and whether it is causally related to her exposure to the water at Camp Lejeune. The analysis integrates findings from epidemiology, toxicology, and mechanistic research, with emphasis on competing risk factors in her medical history and occupational exposures.

## 2. Professional Background and Qualifications

I am a nationally recognized urologic oncologist with extensive expertise in the diagnosis, treatment, and research of genitourinary cancers, including renal cell carcinoma (RCC). I graduated from the University of California, San Francisco (UCSF) School of Medicine, where I developed a strong foundation in patient care and clinical research. Following medical school, I completed my urology residency at the Cleveland Clinic, a program renowned for its leadership in urologic innovation and patient-centered care. I also completed a Society of Urology-accredited 3-year fellowship at the National Cancer Institute (NCI), where I acquired extensive knowledge of renal cell carcinoma and familial RCC syndromes. This experience provided me with a deep understanding of the genetic and environmental factors driving kidney cancer and expertise in managing complex cases involving hereditary cancer syndromes.

Currently, I serve as an Assistant Professor of Urology at Johns Hopkins University School of Medicine and Director of Urologic Oncology at Sibley Memorial Hospital. I have evaluated numerous cases involving potential links to environmental and occupational exposures, leveraging my expertise in interpreting epidemiological data, mechanistic studies, and clinical outcomes to assess causation and inform patient management. I also train urology residents and fellows, sharing my clinical expertise and mentoring the next generation of urologic oncologists.

My clinical focus in RCC includes advanced techniques for nephron-sparing surgery, minimally invasive partial nephrectomy, and the management of patients with complex tumors, including young-onset and familial renal malignancies. I have authored numerous publications and have been invited to speak and lecture on these topics at national and international conferences.

My combined clinical and research background uniquely positions me to assess and elucidate the role of environmental exposures in RCC development, providing a comprehensive perspective on the interplay between genetics, environmental factors, and clinical outcomes.

## 3. Methodology

I utilized the differential diagnosis methodology, a scientifically valid approach widely accepted in the medical and scientific communities, to systematically evaluate all potential factors contributing to the patient's cancer. This methodology ensures a thorough and objective analysis by prioritizing the identification and exclusion of less likely causes based on evidence. It involves a comprehensive review of the patient's medical, family, and exposure histories, focusing on primary and competing risk factors. Temporal relationships, such as latency periods between exposures and disease onset, are analyzed, and mechanistic and pathophysiological evidence—such as genetic and molecular markers—are correlated with the known effects of

suspected carcinogens. Additionally, the patient's risk profile is validated against established epidemiological research to ensure consistency and accuracy. By integrating clinical, exposure, and mechanistic data, this methodology provides a robust framework for assessing causation, grounded in scientific principles and supported by peer-reviewed literature.

**4. Materials Reviewed**

The materials I relied upon are listed on the attached document entitled materials considered list.

**5. Causation Standard**

Under the statute at issue in this case, there are two ways to meet the causation burden:

> "(1) 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."

The ATSDR, in their Assessment of the Evidence analyzing Camp Lejeune in 2017,[1] defined these classifications as follows:

"Sufficient evidence for causation: the evidence is sufficient to conclude that 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 the science and medicine in my field and it informs this causation analysis.

**6. Patient Background**

**Date of Birth**: February 3, 1965

**Medical History**:

- Multifocal clear cell papillary renal cell carcinoma (RCC)
  - Right partial nephrectomy in 2010
  - Left partial nephrectomy in 2018
  - Left partial nephrectomy in 2019
  - Right total nephrectomy in 2022
  - Complete left nephrectomy in June 2023, resulting in anephric status.
- In-center hemodialysis and peritoneal dialysis following anephric status, from June 2023 until kidney transplant in April 2024.
- Kidney transplant (deceased donor transplant) on April 23, 2024.
- Anemia of ESRD, CKD (secondary to nephrectomies), hypertension, osteoarthritis, hyperparathyroidism of renal origin, and sleep apnea.

**Family History**:

- There is a question as to whether Mrs. Tukes' mother and cousin had kidney cancer.
  - Mother died from metastatic disease, but it is important to note that the medical records for Mrs. Tukes do not indicate a biopsy confirming metastatic disease from kidney cancer.
  - The genetic counseling records from UNC Chapel Hill state:
    - "Mother (Christine, d. 66) – diagnosed with an unknown cancer which was metastatic at diagnosis. Ms. Tukes remembers that her mother had a renal mass, but it is unclear if it truly was a renal primary cancer." (01553_TUKES_0000000479).

- Three sisters and one brother with no cancer history.
- Paternal family history includes lung cancer and smoking.

### 7. Dr. Irving Allen Report

I have read the report of Plaintiff's expert Dr. Irving Allen. Dr. Allen's report states that he believes more likely than not that Mrs. Tukes' kidney cancers were not hereditary. He states this based upon the genetic testing that was done at UNC and elsewhere.

While Dr. Allen states that Mrs. Tukes' kidney cancers were not likely in fact caused by a hereditary or familial condition, he does state that Mrs. Tukes was made more susceptible to exposures to carcinogens at lower levels due to two genetic alterations in the PMS2 and SMARCA4 genes. These genes have DNA reparative and tumor suppressor functions. As a result of these two genetic alterations, when Mrs. Tukes was exposed to carcinogens, even at lower levels, she was genetically more susceptible to cancers generally, including kidney cancer, as likely as not.

This is significant in this case because while Mrs. Tukes did not have any other risk factors we know existed in fact and that were likely to significantly increase her risk for kidney cancer, this makes her exposure that much more significant in terms of cancer risk. The levels of the chemicals Mrs. Tukes was exposed to at Camp Lejeune are similar to levels in the literature that show an increased risk of kidney cancer, however, when it is factored into the analyses that she was as likely as not genetically susceptible to cancers at these low levels, it makes the strength of that association even stronger.

### 8. Exposure Assessment and Factual History

**Contaminant Levels at Camp Lejeune (Days Exposed 570)**:

- **Tetrachloroethylene (PCE)**: 82.85 µg/L
- **Trichloroethylene (TCE)**: 3.65 µg/L
- **Vinyl Chloride (VC)**: 13.04 µg/L
- **Benzene (BZ)**: 60 µg/L

Mrs. Tukes was born on [redacted] 1965 and in Jacksonville, North Carolina. Mrs. Tukes has been married for over 40 years. 01553_TUKES_0000000009; Tukes 2024 Dep. 15:13, 17:24. She has three children who all live in Jacksonville, NC. Tukes 2024 Dep. 18:6-11, 135:19-23.

During her time on Camp Lejeune, Mrs. Tukes first lived at the Hostess House located on Hadnot Point in June of 1985. Tukes 2025 Dep. 14:20-15:3;42:23-43:1. Mrs. Tukes then lived for approximately five months (from July 1985 through December 1985) at Sherwood Mobile Home Park, across the street from Camp Lejeune. Tukes 2025 Dep. 46:2-12.

Mrs. Tukes then moved to Tarawa Terrace in December of 1985 with her husband, Willie, and her son, Antonio. Tukes 2025 Dep. 46:11-12; Tukes 2024 Dep. 37:3-9, 40:16, Ex. 5, Housing Occupancy Record. The family lived at Tarawa Terrace until January 1987. They lived in a two-bedroom house for the entirety of their time at Camp Lejeune. Tukes 2024 Dep. 37:10-38:9.

Mrs. Tukes spent most of her days on base. Tukes 2024 Dep. 39:10-15. During the summer, she would fill up a personal pool with hose water for her son to play in outside of their house. Tukes 2024 Dep. 40:3-6, 41:5-17.

Mrs. Tukes drank from the water fountains. Tukes 2024 Dep. 49:14-19. While at the hospital for her pregnancy, she drank the water from the water fountains. Tukes 2024 Dep. 49:20-22; Tukes 2025 Dep. 29:7-9. She would do the same from the water fountains at the Commissary and the Exchange where she would do her shopping. Tukes 2024 Dep. 55:23-57:8. She remembers not liking the taste of the water. Tukes 2024 Dep. 65:24-66:9.

Mrs. Tukes mixed a pitcher of Kool-Aid almost every day and kept a pitcher in the fridge. She drank a lot of the Kool-Aid herself. Tukes 2024 Dep. 52:8-15, 52:24-53:4. She made mixed pitchers of lemonade often during the week. She would share lemonade pitchers with her husband and son. Tukes 2024 Dep. 53:7-18.

Mrs. Tukes would have water with breakfast. She would normally have two or three cups. Tukes 2024 Dep. 51:12-22. She had mixed orange juice from concentrate with water and drank that with her breakfast several times a week as well. Tukes 2024 Dep. 59:1-14. With lunch, she drank at least two glasses of Kool-Aid, tea, or lemonade. Tukes 2024 Dep. 59:23-60:15. She testified that she would often meet her husband for lunch at Hadnot Point and would drink a cup of water from the concession stand. Tukes 2024 Dep. 39:22-25, 50:3-7.

Mrs. Tukes cooked three meals each day. Tukes 2024 Dep. 57:18-24. She also cooked with water every time she made dinner and commonly boiled her vegetables, rice, and meat in water for dinner. Tukes 2024 Dep. 60:16-61:6.

Mrs. Tukes testified that she would shower for up to 15 to 20 minutes a couple of times a day and her showers would be hot. Tukes 2024 Dep. 49:1-8, 54:2-17, 55:1-7. Sometimes she would use a bath.

Mrs. Tukes washed her own dishes after every meal in hot water. Tukes 2024 Dep. 61:13-15, 106:11-23. She did her own laundry at her house which would expose her to the toxic water. Tukes 2024 Dep. 50:9-25. Mrs. Tukes would mop her floors with hot water. Tukes 2024 Dep. 62:14-22.

Mrs. Tukes testified that she would brush her teeth upwards of 6-7 times a day. Tukes 2024 Dep. 107:4-13.

This testimony serves to evidence the substantial nature of Mrs. Tukes' exposure at Camp Lejeune. It evidences that Mrs. Tukes' exposure came from ingestion, inhalation and dermal exposure.

## 9. The Levels of the Toxins in the Water at Camp Lejeune

The levels of PCE in the Camp Lejeune water supply exceeded the EPA's maximum contaminant levels (MCLs). PCE levels reached 8.28 µg/L, exceeding the MCL of 5 µg/L.

Mrs. Tukes' exposure at Tarawa Terrace:

| Exposure Dates | TCE (ug/l-M) | PCE (ug/l-M)(TechFlowMP Model) | PCE (ug/l-M)(MT3DMS Model) | VC (ug/l-M) | BZ (ug/l-M) |
|---|---|---|---|---|---|
| 12/18/1985-12/31/1985 | 0.16 | 3.58 | 8.27 | 0.76 | 0.00 |
| 1/1/1986-1/31/1986 | 0.18 | 3.95 | 8.85 | 0.82 | 0.00 |
| 2/1/1986-2/28/1986 | 0.19 | 4.24 | 9.42 | 0.83 | 0.00 |
| 3/1/1986-3/31/1986 | 0.24 | 5.40 | 12.14 | 1.01 | 0.00 |
| 4/1/1986-4/30/1986 | 0.22 | 4.93 | 10.83 | 0.89 | 0.00 |
| 5/1/1986-5/31/1986 | 0.23 | 5.25 | 11.56 | 0.91 | 0.00 |
| 6/1/1986-6/30/1986 | 0.25 | 5.61 | 12.28 | 0.92 | 0.00 |
| 7/1/1986-7/31/1986 | 0.26 | 5.97 | 13.06 | 0.94 | 0.00 |
| 8/1/1986-8/31/1986 | 0.28 | 6.36 | 13.84 | 0.96 | 0.00 |
| 9/1/1986-9/30/1986 | 0.30 | 6.75 | 14.61 | 0.97 | 0.00 |
| 10/1/1986-10/31/1986 | 0.31 | 7.12 | 15.42 | 0.99 | 0.00 |
| 11/1/1986-11/30/1986 | 0.33 | 7.52 | 16.21 | 1.00 | 0.00 |
| 12/1/1986-12/31/1986 | 0.34 | 7.89 | 17.03 | 1.01 | 0.00 |
| 1/1/1987-1/8/1987 | 0.36 | 8.28 | 17.85 | 1.03 | 0.00 |
| | **3.65** | **82.85** | **181.37** | **13.04** | - |

Mrs. Tukes' exposure at Hadnot Point:

| Exposure Dates | TCE (ug/l-M) | PCE (ug/l-M) | VC (ug/l-M) | BZ (ug/l-M) |
|---|---|---|---|---|
| | | | | |
| 6/18/1985-6/30/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 7/1/1985-7/18/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 7/19/1985-7/31/1985 | 0.00 | 0.00 | 0.00 | 3.00 |

| | | | | |
|---|---|---|---|---|
| 8/1/1985-8/31/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 9/1/1985-9/30/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 10/1/1985-10/31/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 11/1/1985-11/30/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 12/1/1985-12/17/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 12/18/1985-12/31/1985 | 0.00 | 0.00 | 0.00 | 3.00 |
| 1/1/1986-1/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 2/1/1986-2/28/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 3/1/1986-3/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 4/1/1986-4/30/1986 | 0.00 | 0.00 | 0.00 | 4.00 |
| 5/1/1986-5/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 6/1/1986-6/30/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 7/1/1986-7/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 8/1/1986-8/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 9/1/1986-9/301986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 10/1/1986-10/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 11/1/1986-11/30/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 12/1/1986-12/31/1986 | 0.00 | 0.00 | 0.00 | 3.00 |
| 1/1/1987-1/8/1987 | 0.00 | 0.00 | 0.00 | 2.00 |
| | - | - | - | **60.00** |

As will be discussed below, there are scientific studies, including those by the ATSDR, that demonstrate these concentrations are sufficient to cause genotoxic effects and cause renal cell carcinoma. Mrs. Tukes' exposure levels meet or exceed the thresholds identified in epidemiological studies linking these contaminants to RCC risks.

I have also reviewed the summary exposure charts from Plaintiff's expert Kelly Reynolds. Dr. Reynolds provides estimated doses of ingestion for Mrs. Tukes based on the following assumptions for amounts of ingestion: 1L, ATSDR's CTE assumption, ATSDR's RME assumption and Mrs. Tukes' deposition statements.

The total ranges of exposures for Mrs. Tukes, according to these charts, is listed below:

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

## 10. Causation Analysis

I have reviewed the general causation expert reports of Drs. Hatten and Bird. The analysis in those reports supports my opinions in this report and finds that the four chemicals in the water at Camp Lejeune are causally related to kidney cancer. I also researched and read the epidemiology, toxicology and mechanistic evidence that exists relating to the toxins at issue in this case and agree that the toxins, as they existed in combination in the water at Camp Lejeune, are causally related to kidney cancer under a more likely than not standard, which exceeds the "at least as likely as not" standard. A summary of some of the evidence is below and is used for purposes of weighing the potential harmful effects of the exposure Ms. Tukes had to the water at Camp Lejeune.

### a. TCE and Kidney Cancer

Trichloroethylene (TCE) is a widely used industrial solvent and volatile organic compound (VOC) known to contaminate soil and groundwater, as seen at Camp Lejeune. It has been classified as a known human carcinogen by both the International Agency for Research on Cancer (IARC) and the Environmental Protection Agency (EPA), with specific evidence linking TCE exposure to kidney cancer.[2]

#### i. Epidemiological Evidence

IARC concludes there is sufficient evidence in humans for TCE's carcinogenicity, particularly causing kidney cancer.[2] The Agency for Toxic Substances and Disease Registry (ATSDR) also recognizes sufficient evidence of causation for kidney cancer associated with TCE.[1] A 2010 meta-analysis by Kelsh et al. demonstrated a statistically significant relative risk (RR) of 1.42 (95% CI 1.17-1.77) for occupational TCE exposure and kidney cancer.[3] A 2011 EPA manuscript reported an overall RR of 1.27 (95% CI 1.13-1.43), with higher risks for groups exposed to elevated TCE levels (RR 1.58, 95% CI 1.28-1.96).[4]

#### ii. Meta-Analyses and Studies

Karami et al. (2012) reviewed 9 studies and found an elevated RR of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer, with consistent results across cohort and case-control designs.[5] The study noted that misclassification of exposure in earlier research likely underestimated the true risk.[5]

#### iii. Mechanistic Plausibility

TCE is metabolized into nephrotoxic compounds, such as S-(1,2-dichlorovinyl)-L-cysteine (DCVC), which bioactivate in the kidneys, causing DNA damage and mutations. TCE exposure induces oxidative stress, leading to lipid peroxidation and impaired antioxidant activity, which are key drivers of renal carcinogenesis.

#### iv. Conclusion

**In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on all uses of trichloroethylene (TCE) to safeguard public health from the associated risks, including kidney cancer, linked to TCE exposure.[6]**

Collectively, epidemiological data, mechanistic studies, and meta-analyses provide robust evidence of the causal link between TCE exposure and kidney cancer, highlighting its significant public health implications. It is overwhelmingly probable that TCE causes kidney cancer.

In this case Mrs. Tukes' levels of TCE would have acted additively to the other chemicals in the water at Camp Lejeune.

### b. PCE and Kidney Cancer

Perchloroethylene (PCE), also known as tetrachloroethylene, is a volatile organic compound widely used in the dry-cleaning industry and as a degreaser. It is classified as a *probable human carcinogen* by IARC (Group 2A),[2] and the EPA has determined that PCE is "Likely to be Carcinogenic to Humans" by all exposure routes.[7]

### i. Epidemiological Evidence

**Cape Cod Study**: Aschengrau et al. examined individuals exposed to PCE-contaminated drinking water, finding a relative risk (RR) of 1.23 (95% CI 0.40-3.11) for kidney cancer with any exposure.[8] Although underpowered, this study was very similar to the circumstances at Camp Lejeune, including the levels of exposure, and therefore provides very relevant information. Aschengrau also found a relative risk (RR) of 1.36 for low exposures relating to kidney cancer.[8]

**U.S. Kidney Cancer Study**: Purdue et al conducted a case-control study and reported an odds ratio (OR) of 3.1 (95% CI 1.3-7.4) for high cumulative PCE exposure and kidney cancer, indicating a strong association.[9]

**Dry-Cleaning Industry Studies**: Ruder et al. found a standardized mortality ratio (SMR) of 1.41 (95% CI 0.46-3.30) among 1,708 dry-cleaning workers exposed to PCE.[10] Karami et al. found an elevated risk of developing renal cell carcinoma (OR) of 2.0 (95% CI: 0.9-4.4), which increased for longer employment in the dry-cleaning industry to 2.5 (95% CI 0.4-14.4).[11]Callahan et al. identified a dose-response relationship, with the highest exposure group showing a hazard ratio (HR) of 13.2 (95% CI 1.9-90.8) for kidney cancer mortality.[12]

**Camp Lejeune Study**: Bove et al. examined civilian workers at Camp Lejeune and found a standardized mortality ratio (SMR) of 1.30 (95% CI 0.52-2.67) for kidney cancer.[13] In Bove 2014a, with particular respect to PCE, the relative risks of kidney cancer based on low, medium and high exposures were: 1.40 for low exposure, 1.82 for medium exposure and 1.59 for high exposure.[14] Mrs. Tukes would have had exposures similar to the low exposure group, which is associated with a HR of 1.40.

### ii. Mechanistic Evidence

**Metabolic Activation**: PCE is metabolized into trichloroacetic acid (TCA) and other metabolites that can form DNA adducts, leading to mutations and carcinogenesis.

**Oxidative Stress**: PCE exposure induces reactive oxygen species (ROS) and lipid peroxidation, which cause DNA strand breaks and mutations.

**Cytotoxicity and Proliferation**: PCE and its metabolites trigger cytotoxic effects, promoting compensatory cell proliferation, which increases cancer risk.

### iii. Conclusion

**In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on all uses of perchloroethylene (PCE) to protect public health from the associated risks, including its link to various cancers including kidney cancer.**[6]

Mrs. Tukes' levels of PCE are similar to levels in the literature that show a

causal association between PCE and RCC. According to Bove 2014a's cumulative exposure charts, Mrs. Tukes would have been categorized as above the middle of the "low" exposure group for PCE. This corresponded to a HR of 1.40.[14]

Mrs. Tukes has similar levels of PCE exposure shown in the literature to be associated with kidney cancer. For example, in Aschengrau (1993), the relative delivered dose (RDD) up to the 90th percentile was between 27.1 and 44.1 mg.[8] Mrs. Tukes levels of exposure, taken cumulatively, including ingestion, inhalation and dermal, evidence similar levels from this study and indicate an increased risk of kidney cancer similar to those found in the study.

Epidemiological studies, particularly those involving occupational and environmental exposures, along with mechanistic data, support a causal association between PCE and kidney cancer. The findings underscore the carcinogenic potential of PCE. It is more likely than not that PCE causes kidney cancer.

**c. Other Contaminants: Vinyl Chloride and Benzene**

Both compounds are linked to genotoxic effects but are less robustly associated with RCC than TCE. Synergistic effects with other carcinogens (e.g., PCE) amplify the risk.

**i. Epidemiological Evidence**

Benzene has a significant body of literature that shows a causal relationship with kidney cancer. A meta-analysis was performed analyzing 29 studies and found that there was a relative risk (RR) of 1.2 (95% CI 1.03 – 1.39).[15] Additionally, Hu (2002) found a monotonic response relationship between benzene and kidney cancer (RCC) and a RR of 1.8 (95% CI: 1.2-2.6) for kidney cancer.[16] There are other studies as well showing this causal relationship, including the Camp Lejeune studies.[13,14]

Vinyl Chloride has been less studied than the other three chemicals at issue; however, when it has been studied there has been epidemiologic literature showing a causal association between vinyl chloride and kidney cancer. For example, the Hu (2002) study found a monotonic response relationship between vinyl chloride and kidney cancer with an OR of 2.0 (95% CI: 1.2 – 3.3).[16] Further, the Camp Lejeune and Bove studies support this causal relationship.

**ii. Mechanistic Evidence**

Benzene's carcinogenicity in the kidney is driven by its metabolism into reactive intermediates, including hydroquinone and benzoquinones, which generate oxidative stress and DNA damage. These metabolites induce DNA strand breaks, chromosomal aberrations, and mutations, contributing to genomic instability. Benzene exposure also impairs DNA repair mechanisms and forms DNA adducts, further exacerbating mutagenesis. These pathways collectively increase the risk of renal carcinogenesis.

Vinyl chloride is metabolized into reactive intermediates like chloroethylene oxide and chloroacetaldehyde, which form DNA adducts, such as etheno-deoxyadenosine. These adducts disrupt genomic integrity and lead to mutations in key genes regulating DNA repair and cell

cycle control. Additionally, vinyl chloride induces oxidative stress and TP53 pathway dysregulation, both of which are implicated in RCC development. Its toxic effects on renal cells underscore its role in kidney carcinogenesis.

**iii. Conclusion**

There is evidence to support the causal relationship of benzene and vinyl chloride to kidney cancer at least as likely as not and using an equipoise standard.

**d. Conclusion For All Chemicals**

There was a monotonic response relationship between TVOCs at Camp Lejeune and kidney cancer.
Bove 2014a.[14] The HRs were 1.42 (low exposure), 1.44 (medium exposure) and 1.54 (high exposure).[14]

The epidemiological and occupational studies collectively underscore the link between TCE, PCE, benzene, and vinyl chloride exposures and renal cell carcinoma. Findings from these Camp Lejeune studies, occupational analyses, and dose-response models reinforce the carcinogenic roles of these chemicals, particularly in populations with high or prolonged exposures.

The data from the Camp Lejeune studies of Bove and ATSDR provide compelling data that exposures of this kind are causally related to kidney cancer.[14,17]

**11. Patient-Specific Considerations**

**Genetic Predisposition**: Genetic testing in 2019 revealed a variant of unknown significance (VUS). Further testing at UNC Chapel Hill did not show any definitive pathogenic mutations associated with hereditary kidney cancer syndromes. Additional genetic analysis by Dr. Irving Allen identified heterozygous variants in PMS2 and SMARCA4 genes, both classified as likely benign but suggestive of increased susceptibility to carcinogen-induced cancers. History of exposure to contaminated water at Camp Lejeune, which is a known risk factor for kidney cancer.

**Latent Period**: Ms. Tukes was exposed to contaminated water from 1985 to 1987. Her initial diagnosis of kidney cancer occurred in 2010, approximately 23 years later, which aligns with known latency periods for environmentally induced cancers.

**Exposure**: While these levels were lower than some other documented exposures, they still are comparable to levels in the literature that have been associated with RCC, especially when Ms. Tukes' genetic susceptibility to cancer at these lower exposure levels is taken into account.

The general causation reports of Drs. Hatten and Bird detail the levels at which the chemicals at issue are known to be causally associated with kidney cancer. I have read the reports and agree with the findings in those reports as to the levels that are known to cause kidney cancer. For example, some of the levels known to cause kidney cancer, relating to these toxins, are as follows:

- **Cumulative exposure to 27.1-44.1 mg of PCE**[8]
- **Sustained exposure to 0-25.3 ppb of TCE**[18]
- **Exposure to a TCE concentration of 267.4 ppb**[19]
- **Cumulative exposure of 1 - 3,100 µg/L-month of TCE**[14]
- **Cumulative exposure of 1 - 155 µg/L-month of PCE**[14]
- **Cumulative exposure of 1 – 4,600 µg/L-month of exposure to all compounds at Camp Lejeune**[14]
- **Cumulative exposure of 3,100 – 7,700 µg/L-month of TCE**[14]
- **Cumulative exposure of 155 - 380 µg/L-month of PCE**[14]
- **Cumulative exposure of 4,600 – 12,250 µg/L-month of exposure to all compounds at Camp Lejeune**[14]
- **Cumulative exposure greater than 7,700 µg/L-month of TCE**[14]
- **Cumulative exposure greater than 380 µg/L-month of PCE**[14]
- **Cumulative exposure greater than 12,250 µg/L-month of exposure to all compounds at Camp Lejeune**[14]
- **Approximately 18 months of residence on base from 1975 to 1985**[14]
- **Employment on base for 2.5 years**[13]
- **Cumulative exposure to 110 – 11,030 ppb-months of TCE**[17]
- **Cumulative exposure to 36 - 711 ppb-months of PCE**[17]
- **Cumulative exposure greater than 11,030 ppb-months of TCE**[17]
- **Cumulative exposure greater than 711 ppb-months of PCE**[17]

Mrs. Tukes' exposure meets levels that are known to be hazardous to humans and that cause kidney cancer. This data is important in the differential diagnosis analysis conducted below.

**12. Substantial Exposure**

As shown above through the deposition testimony of Mrs. Tukes, her exposure to these chemicals at these concentrations were substantial. Mrs. Tukes was, on a daily basis, frequently and consistently exposed to these chemicals through all three known routes of exposure. Mrs. Tukes was drinking the water at her meals, she was showering and inhaling the toxic chemicals at least daily and often multiple times a day and she was exposed to the chemicals on her skin daily. This exposure is made substantial, in part, given that Mrs. Tukes was genetically more susceptible to environmental exposures to carcinogens at lower levels. All of this combined shows a substantial exposure that is more likely than not causally related to the kidney cancer she developed. I was able to draw my conclusions as to the substantial nature of Mrs. Tukes' exposure from the records reviewed in this case, her deposition testimony and the concentrations in the water at the times Mrs. Tukes was exposed (in combination with the fact she was more susceptible at lower levels).

In addition to this information, the charts from Plaintiff's expert Kelly Reynolds indicate a substantial exposure. For example, Dr. Reynolds calculated that Mrs. Tukes would have ingested 5,875 ppb of PCE assuming the testimony from her deposition.

These numbers are demonstrative of the substantial exposure for Mrs. Tukes and an exposure that is causally related to the development of her kidney cancer. The ingestion numbers are strengthened by the fact that these charts only take into account one exposure route: ingestion. Mrs. Tukes would have been exposed via inhalation and dermal exposure. When factoring in those routes of exposure, Mrs. Tukes' actual exposure dose would be greater.

The charts from Kelly Reynolds provide additional support for my opinions and are consistent with them.

**13. Differential Diagnosis to Determine Etiology and Competing Risk Factors**

**a. Primary Risk Factors**

**Genetic Predisposition**: There is a potential maternal and cousin history of kidney cancer. This familial history has not been accurately confirmed however, based on the medical records. This suggests a possible familial component, even though not confirmed. Genetic testing was performed at UNC Chapel Hill and was all negative with regard to any hereditary genes or conditions. Dr. Allen's report indicates that while no definitive pathogenic mutations were identified for the causation of kidney cancer, the presence of heterozygous variants in PMS2 and SMARCA4 as likely as not lowered the threshold for carcinogenesis when exposed to environmental carcinogens.

**Environmental Exposure**: Documented exposure to Camp Lejeune water contaminated with TCE, PCE, VC and benzene, all associated with RCC development.

**b. Competing Risk Factors**

No significant history of occupational exposure or other known carcinogens apart from Camp Lejeune.

Chronic kidney disease (CKD) was a consequence of multifocal RCC and subsequent nephrectomies rather than a contributing cause.

Mrs. Tukes had a history of hypertension, but it was not likely a cause of her kidney cancer. Hypertension is a less significant risk factor compared to environmental exposure to water with multiple carcinogens, such as her exposure at Camp Lejeune. Further, during the time Mrs. Tukes was diagnosed with her many kidney cancers, her hypertension was at times high and was at times well controlled and in the normal range. Finally, this is consistent with Mrs. Tukes treating oncologist, Dr. Jayaram, who testified that he did not believe Mrs. Tukes' hypertension was related to her kidney cancer. (Jayaram Dep. P. 28).

Mrs. Tukes was slightly overweight with BMIs in the high 20s up to 31 during the times that she developed kidney cancers from 2010 through 2023. Again, her BMI was at times higher than others during this time period, which makes it less likely that Mrs. Tukes' weight was related to her kidney cancer. Further, as a general matter, BMI causation of RCC, especially bilateral and

multifocal tumors, is a much lower risk than exposure to multiple carcinogenic compounds at the same time over the course of 19 months at Camp Lejeune.

**c. Conclusion**

The combination of Ms. Tukes' documented exposure to carcinogenic chemicals at Camp Lejeune, along with her genetic susceptibility to carcinogenic exposures, provides a compelling basis for multifactorial causation of her renal cell carcinoma. While there is the potential for a history of maternal and cousin kidney cancer, this was never confirmed and therefore cannot be used definitively. Any familial connection to Mrs. Tukes' kidney cancer is made significantly less likely as a result of the genetic testing that was performed at UNC Chapel Hill. All testing was negative for a hereditary condition. The presence of heterozygous variants in PMS2 and SMARCA4 indicates a genetic predisposition that likely lowered her threshold for cancer development upon exposure to environmental carcinogens. Given the well-documented link between TCE/PCE exposure and RCC and considering Ms. Tukes' quantified environmental exposure history, it is **more likely than not** that her exposure to Camp Lejeune contaminants was a primary factor in the development of her renal cell carcinoma, outweighing the potential contribution from other risk factors.

**14. Reponses to the Defendants' Supplemental Answers to Interrogatories**

In response to Interrogatories sent by the Plaintiff, Defendant has stated the below may have been contributory to her kidney cancer. I will respond to each in turn below:

**a. A Potential Family History of Kidney Cancer**

As described above, it is unclear if there is in fact a family maternal history of kidney cancer. Mrs. Tukes' mother had a renal mass, but there was no confirmed pathological diagnosis of metastatic kidney cancer seen in any of the medical records for Mrs. Tukes. Additionally, genetic testing conducted at UNC Chapel Hill was negative for any hereditary kidney cancer syndromes. While no genetic test can completely exclude hereditary factors, the likelihood of a hereditary cause for Mrs. Tukes' kidney cancer is very low. This is consistent with the testimony of Mrs. Tukes treating physicians, Dr. Irving Allen and also Mrs. Tukes' genetic counselor, Mary Garbarini.

**b. A Family History of Cancer Other than Kidney Cancer**

A family history of other cancers does not alter the analyses because kidney cancer has distinct etiological and genetic pathways separate from other malignancies. While certain cancers can be linked to shared genetic mutations, no hereditary predisposition was identified in Mrs. Tukes' genetic testing that would suggest a causal relationship between other familial cancers and her kidney cancer.

**c. A Family History of Rhabdomyolysis and Kidney Failure**

A family history of rhabdomyolysis and kidney failure does not alter the analysis because neither condition is an established risk factor for renal cell carcinoma (RCC). Rhabdomyolysis is a condition characterized by the rapid breakdown of skeletal muscle, leading to the release of myoglobin into the bloodstream, which can cause acute kidney injury (AKI) in severe cases. While repeated or severe episodes of rhabdomyolysis can contribute to chronic kidney disease (CKD), it does not have a mechanistic link to cancer development.

Furthermore, familial rhabdomyolysis is typically associated with genetic mutations affecting muscle metabolism, none of which are known to predispose individuals to RCC. Kidney failure resulting from rhabdomyolysis is due to acute tubular necrosis and nephrotoxicity from myoglobin, not oncogenic processes.

Because RCC arises from mutations in renal epithelial cells, there is no known biological mechanism linking rhabdomyolysis or its genetic predisposition to kidney cancer development.

**d. A Family History of High Cholesterol, Hypertension, Kidney Disease and Stroke**

A family history of high cholesterol, hypertension, kidney disease, and stroke does not significantly contribute to the etiology of Mrs. Tukes' RCC. While hypertension is a known risk factor for kidney cancer, it is a relatively weak risk factor compared to prolonged exposure to carcinogens like TCE, PCE, benzene, and vinyl chloride.

**e. An Incident of Suffering from a Hernia**

A hernia does not contribute to the development of kidney cancer because it has no known carcinogenic mechanisms related to renal cell carcinoma. Hernias are structural conditions affecting the abdominal wall and have no association with genetic mutations or toxic exposures linked to RCC.

**f. An Incident of Pancreatitis**

An incident of pancreatitis does not contribute to the causation of RCC. Pancreatitis is an inflammatory condition affecting the pancreas and is not linked to carcinogenesis in the kidney. There is no established scientific evidence that acute or chronic pancreatitis increases the risk of renal cancer.

**g. Tuberculosis Exposure**

First, it is unclear from the records that Mrs. Tukes was actually ever exposed to Tuberculosis through her patients. However, even assuming that as a nurse Mrs. Tukes had patients with Tuberculosis, there is no scientific evidence to suggest that tuberculosis exposure is associated with an increased risk of kidney cancer. Unlike known carcinogens such as TCE, PCE, benzene, and vinyl chloride, tuberculosis does not have a direct carcinogenic mechanism. While some

infections can contribute to cancer through chronic inflammation and immune dysregulation, this has not been reported renal cell carcinoma (RCC). Given the absence of a documented link between tuberculosis and RCC, any potential exposure to tuberculosis would not be a relevant competing risk factor in this case.

**h. Mrs. Tukes Exposure May Not Have Been Significant Enough**

This was addressed above extensively, however, Mrs. Tukes' exposure was at levels that are similar to literature that shows a causal association with kidney cancer. Mrs. Tukes was also as likely as not, according to Dr. Irving Allen, more susceptible to cancers at low levels of environmental carcinogenic exposures. When all of these facts are taking into account, Mrs. Tukes had a substantial exposure and one that is at levels that are known to cause kidney cancer. In this case, given the lack of other significant risk factors, Mrs. Tukes' Camp Lejeune exposure is the most likely cause of her very unique presentation of kidney cancers.

**i. The Latency Period May Indicate An Alternative Cause**

Mrs. Tukes latency period of approximately 23 years fits within the expected time frame for the development of kidney cancer following an exposure such as the one she had at Camp Lejeune. Scientific literature supports that kidney cancer can develop decades after initial exposure to carcinogens such as TCE and PCE. This is consistent with studies examining occupational and environmental exposures to these chemicals, reinforcing that her cancer's latency period does not suggest an alternative cause.

**15. Mrs. Tukes' Damages**

I will discuss Mrs. Tukes' harms suffered as a result of the Camp Lejeune water and Mrs. Tukes' kidney cancer. This includes the medical treatment related to her kidney cancer, the surgeries required to remove her kidneys and the typical medical course for individuals like Mrs. Tukes. Additionally:

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

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

3. The medical billing for Mrs. Tukes' treatment and care related to her kidney cancer is fair and reasonable and this treatment was medically necessary.

**16. Conclusion**

Considering Mrs. Tukes' exposure to TCE, PCE, Benzene and vinyl chloride, the prolonged latency period, and the consistent epidemiological and mechanistic evidence, it is more likely than not that her RCC was caused by environmental contaminants at Camp Lejeune. This is made more likely by the fact Mrs. Tukes was genetically more susceptible to environmental

exposure to carcinogens, such as the Camp Lejeune water, at low levels. This makes the fact that she was exposed to the chemicals more dangerous and put Mrs. Tukes at much higher risk for the development of her kidney cancer. Competing risk factors such as the potential familial background (understanding this has not been confirmed), hypertension for a period of time and being overweight are insufficient to explain the diagnosis.

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# ARMINE SMITH'S CV

CURRICULUM VITAE
The Johns Hopkins University School of Medicine

(Signature) _____ 2/4/2025
(Typed Name) Armine Karapetian Smith, MD

## DEMOGRAPHIC AND PERSONAL INFORMATION

### Current Appointments

University

2013-present Assistant Clinical Professor of Urology, George Washington School of Medicine
2014-present Assistant Professor of Urology, Department of Urology, Johns Hopkins University School of Medicine Hospital
2013-present Urologist, George Washington Hospital
2014-present Urologist, Johns Hopkins Hospital
2014-present Urologist and Director of Johns Hopkins Urologic Oncology, Sibley Memorial Hospital
2024-present Site Fellowship Director for the National Cancer Institute, Johns Hopkins National Capital Region

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### Education and Training

Undergraduate

2001 B.S., Biology, University of Southern California, Los Angeles, CA

Doctoral/graduate

2005 M.D., University of California in San Francisco, San Francisco, CA

Postdoctoral

2011 Resident, Urology, Cleveland Clinic, Cleveland, OH
2014 Fellow, Urologic Oncology, Urologic Oncology Branch, National Cancer Institute, Bethesda, MD

### Professional Experience

2015-2017 Participant, MBA program in Healthcare Management, Johns Hopkins University Carey Business School, Baltimore, MD

## PUBLICATIONS:

Original Research [OR]

1. Van der Veen RC, Dietlin TA, **Karapetian A,** Holland SM, Hofman FM: Extra-cellular superoxide promotes T cell expansion through inactivation of nitric oxide. J Neuroimmunol. 2004 Aug;153(1-2):183-9. *Engagement with discovery and data analysis
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1. **Smith AK,** Hansel DE, Klein EA. Epidermoid cyst of the testicle. Urology. 2009 Sep;74(3):544.
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### Book/Textbook Chapters, Monographs [BC]

1. **Smith AK,** Palmer JS: Pediatric robotic urology (Current clinical urology): Chapter 3/Robotic instrumentation and equipment. Humana Press. Sep 9, 2009.
2. **Smith AK,** Matin SF, Jarrett TW. Wein: Campbell-Walsh Urology, 11th ed. Chapter 58/Urothelial Tumors of the Upper Urinary Tract and Ureter. Elsevier 2013.
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## Other Publications:

### Opinions, Perspectives, Political Commentary, Advocacy, Essays [OP]

1. **Smith A.** Technological innovations driving cancer treatment. MedTech Outlook. Nov 2021.
2. **Smith A.** Quality of Life in the Phase 2/3 Trial of N-803 Plus Bacillus Calmette-Guérin in Bacillus Calmette-Guérin–Unresponsive Nonmuscle-Invasive Bladder Cancer. Urol Pract. 2024 Mar;11(2):375.
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2. https://www.youtube.com/watch?v=EwsLHnF_a6w Jul 2016
3. https://clinicalconnection.hopkinsmedicine.org/videos/treating-bladder-cancer-qanda August 2016
4. https://www.bizjournals.com/washington/news/2016/09/29/some-d-c-doctors-are-tackling-cancer-cures-in-a.html Sept 2016
5. https://greaterwashingtonmd.hopkinsmedicine.org/videos/physician-profile-armine-smith-md April 2018
6. https://archive.storycorps.org/interviews/ddb002360/ May 2018*
7. https://hopkinskimmel.libsyn.com/cancer-matters-with-dr-bill-nelson-bladder-cancer-in-women Nov 2018
8. https://clinicalconnection.hopkinsmedicine.org/videos/surgical-advances-for-prostate-cancer June 2021
9. https://ysmu.am/en/content/ysmu_visiting_professor_medicine_demands_to_be_ready_for_challenges/#sthash.0tR5F6Ej.dpbs August 2021*
10. Sex After Bladder Cancer - HealthyWomen May 2022*
11. Why Sex and Race Matter in Bladder Cancer Treatment - HealthyWomen May 2022*
12. https://www.youtube.com/watch?v=D_HdctamXRI Nov 2022
13. Ask the Expert: Urothelial Bladder Cancer - HealthyWomen Oct 2023*
14. Los sobrevivientes de cáncer de vejiga y los segundos cánceres primarios - HealthyWomen March 2024*
15. Bladder Cancer Survivors and Second Cancers - HealthyWomen March 2024*
16. The Biggest Risk Factor for Bladder Cancer Has Nothing to Do With Pee, Diet, or Genes (verywellhealth.com) June 2024*
17. UroToday: Optimizing Care for Women with Bladder Cancer with Armine Smith & Ashish Kamat https://www.urotoday.com/video-lectures/bladder-cancer/video/4159-advancing-female-bladder-cancer-care-insights-from-aua-s-first-specialized-course-armine-smith.html June 2024*
18. Oncology News Central: Oncologists Concerned Over FDA's Bladder Cancer Guidance, with David McConkey https://www.oncologynewscentral.com/article/oncologists-concerned-over-fdas-bladder-cancer-guidance July 2024*

### Other Media [OM]

1. Blog: https://www.beckershospitalreview.com/ehrs/what-clinicians-patients-think-of-charging-for-mychart-messages.html, November 2022
2. Blog: https://twitter.com/urogenpharma Nov 2022
3. Blog: https://www.beneficiusnow.com/2022/12/20/healthsystems-have-begun-charging-for-patient-portal-interactions/?slreturn=20230002112633, December 2022

4. LinkedIn https://www.linkedin.com/in/armine-smith-md-5b7b7042
5. Twitter @akfsurgeon

## FUNDING

### EXTRAMURAL Funding – Previous (2008)
Identification number RPC ID#2008-1012
Sponsor Cleveland Clinic Research Program Committee Funding
Total direct cost $60,000
Role: PI, 25% FTE

### EXTRAMURAL Funding – Previous (2008)
Identification number FAMRI ID#072099CIA
Sponsor Flight Attendant Medical Research Institute Grant
Total direct cost $250,000
Role: co-PI, 10% FTE
PI: Warren Heston, PhD

### INTRAMURAL Funding – Previous (2015)
Identification number JHGBCI ID#80035923
Sponsor Greenberg Bladder Cancer Institute Grant
Total direct cost $50,000
Role: PI, 10% FTE

### INTRAMURAL Funding – Previous (2018)
Identification number: Evaluation of PSMA-based PET as an imaging biomarker of androgen receptor signaling in high-risk localized and locally advanced prostate cancer
Sponsor The Robert L. Sloan Fund for Cancer Research
Total direct cost $70,000, 2-year renewable
Role: co-PI, 1% FTE
PI: Curtiland Deville, MD

### INTRAMURAL Funding – Previous (2020)
Identification number: Exploration of the Microbiome in Patients with Newly Diagnosed Bladder Cancer
Sponsor The Robert L. Sloan Fund for Cancer Research
Total direct cost $70,000, 2-year renewable
Role: co-PI, 1% FTE
PI: Jean Hoffman-Censits, MD

### INTRAMURAL Funding – Current (2024)
Identification number: Association of microbiome composition and its modulation with response to first line therapy in patients with bladder cancer.
Sponsor The Robert L. Sloan Fund for Cancer Research
Total direct cost $70,000, 2-year renewable
Role: PI, 2% FTE

### INTRAMURAL Funding – Current (2024)
Identification number: Mary and Armeane Choksi Scholar
Sponsor Mary and Armeane Choksi
Total direct cost $500,000 5-year
Role: PI
Description: This endowed scholar award was granted directly to me in recognition of my contributions to urologic oncology research and education, supporting my ongoing efforts to advance innovative treatments and multidisciplinary collaboration at Sibley Memorial Hospital.

### INTRAMURAL Funding – Current (2024)
Identification number: Kovler fund for translational research
Sponsor Kovler family
Total direct cost $200,000

Role: PI and fund manager
Description: Dedicated to advancing translational research at Sibley Memorial Hospital, with a focus on fostering innovative approaches in urologic oncology.

### EXTRAMURAL Funding - Pending

Identification number FAMRI ID# 213133
Sponsor Flight Attendant Medical Research Institute Grant
Total direct cost $300,000
Role: PI, 5% FTE

## CLINICAL ACTIVITIES

### Clinical Focus

My clinical focus is urologic oncology and program building in the National Capital Region (NCR). In July 2014 I assumed my current position at Johns Hopkins University, being the only faculty member of the Brady Urological Institute in the DMV area. Under my regional leadership as a director of urologic oncology, the department was able to build a successful program at the Sibley Memorial Hospital with reputation of stable clinical excellence, which now has a stable referral stream and four additional full-time urologists and plans to add at least two more full-time urologists over the course of the next 2 years. The program's success can be seen from my personal work RVUs, which increased from 44 in the academic year 2014 to 2,578 in 2015 and 9,132 in 2022, reaching 152% of RVU targets. In collaboration with the Sidney Kimmel Comprehensive Cancer Center and Radiation Oncology Department, I have created the bi-weekly regional Multidisciplinary Prostate Cancer Program. Working with the Greenberg Bladder Cancer Institute, I have established a one-of-a-kind Women's Bladder Cancer Program, which provides gender-specific multidisciplinary care and clinical trials for patients, has ongoing educational series drawing patients from all over the US, and monthly support programs. This model for care for women with bladder cancer is now being replicated on the national level via Bladder Cancer Advocacy Network. Personally, I have a regional and nationwide reputation for complex reconstructive surgeries, and I am recognized in the field as an expert in bladder cancer in women.

### Certification

2011-present Advanced Cardiac Life Support license, American Heart Association
2011-present Basic Life Support license, American Heart Association

### Medical, other state/government licensure

2011-present Physician and Surgeon, Maryland Board of Physicians, # D73382
2011-present Controlled Dangerous Substances License, Maryland, #M76529
2011-present Controlled Substance Registration (DEA), #FS2759237
2014-present Physician and Surgeon, District of Columbia Board of Medicine #MD042441
2014-present Controlled Dangerous Substances License, District of Columbia, #CS1400387

### Boards, other specialty certification

2017-present The American Board of Urology Diplomate, No 18052

### Clinical (Service) Responsibilities

2011-2014 Urology attending, National Cancer Institute
2013-current Urology attending, George Washington Hospital
2014-2016 Urology attending, Suburban Hospital
2014-2018 Consultant, National Cancer Institute
2014-current Co-director of Prostate Cancer Multidisciplinary Clinic, Sibley Memorial Hospital
2014-current Urology attending, Sibley Memorial Hospital
2014-current Urology attending, Johns Hopkins Hospital
2018-current Co-director of Women's Bladder Cancer Program, Greenberg Bladder Cancer Institute

### Clinical Productivity

FY 2014 Work RVUs 33
FY 2015 Work RVUs 2578
FY 2016 Work RVUs 4866
FY 2017 Work RVUs 6036
FY 2018 Work RVUs 5285
FY 2019 Work RVUs 5782
FY 2020 Work RVUs 5900

FY 2021 Work RVUs 7701
FY 2022 Work RVUs 9132
FY 2023 Work RVUs 8943
FY 2023 Work RVUs 8861

### Clinical Draw from outside local/regional area

A minority of the patients that I see come from the centralized JH scheduling pool; these referrals are both from the National Capital Region (NCR) and nationwide. About 70% of the patients are direct referrals to me for bladder cancer treatment and nephron-sparing kidney surgery; these referrals come from the local primary care physicians, gynecologists and urologists, Bladder Cancer Advocacy Network, and other patients. Outside of NCR, in the past 5 years I have seen international patients from Middle East, South America and Armenia. From the nationwide pool, I have had patients from Arkansas, California, Delaware, Florida, Indiana Nevada, New Jersey, New Mexico, New York, Ohio, Pennsylvania, and West Virginia.

### Clinical Program Building / Leadership

2014-present Staff Urologist and Director of Urologic Oncology at Sibley Memorial Hospital
Established and developed Johns Hopkins Urology practice in the NCR

### Clinical Demonstration Activities to external audience, on or off campus

2015 Laparoscopic surgery/hands-on animal lab, George Washington School of Medicine
2016 Laparoscopic surgery/hands-on animal lab, George Washington School of Medicine

## EDUCATIONAL ACTIVITIES

### Educational Focus

I am a passionate educator dedicated to advancing urologic surgery and oncology training. As a faculty member at both Johns Hopkins University and George Washington University, I provide mentorship, hands-on instruction, and structured training for urology residents and urologic oncology fellows. In 2024, I became the Site Fellowship Director for the National Cancer Institute, overseeing the entire Johns Hopkins National Capital Region. This role has established a regular rotation for NCI fellows across our sites, further integrating advanced oncologic training throughout the region. Additionally, I have hosted several international residents as observers, elevating the reputation of Johns Hopkins Urology at Sibley to an international level. We also welcome a steady stream of observers from various Johns Hopkins engineering programs, fostering interdisciplinary collaboration that is poised to result in multiple innovative projects. Beyond clinical education, I have co-authored chapters in multiple editions of *Campbell-Walsh-Wein Urology*, focusing on the pathogenesis and surgical management of urologic cancers—contributions that serve as foundational resources for urologic training programs worldwide.

## Teaching

### Clinical instruction

JHMI/Regional

2014-present Supervising attending for GW and JH urology residents
2023-present Supervising attending for urologic oncology branch fellows

Classroom instruction

2019 Content developer and speaker, Tips and tricks for successful cystectomy
George Washington School of Medicine
2023 Content developer and speaker, Partial nephrectomy in the modern era
JHU School of Medicine
2024 Content developer and speaker, Variant histologies in bladder cancer
JHU School of Medicine

### CME instruction

JHMI/Regional

11/2020 Content developer and speaker
Audience: JHCP providers
Advances in the treatment of benign prostatic hyperplasia. CME series
12/2020 Content developer and speaker
Audience: JHCP providers
Asymptomatic Microhematuria. Simplified. CME series

Invited National

10/2018 Member faculty
ASCO Advantage Prostate Cancer course, American Association for Cancer Research

12/2019 Member faculty
ASCO Advantage Renal Cell Carcinoma course, American Association for Cancer Research

1/2020 Member faculty
ASCO Advantage Prostate Cancer course, American Association for Cancer Research

4/2020 Member faculty
ASCO Advantage Renal Cell Carcinoma course, American Association for Cancer Research

9/2020 Member faculty
ASCO Advantage Renal Cell Carcinoma course, American Association for Cancer Research

1/2021 Member faculty
ASCO Advantage Prostate Cancer course, American Association for Cancer Research

10/2021 Member faculty
ASCO Advantage Renal Cell Carcinoma course, American Association for Cancer Research

4/2024 Member faculty
ASCO Advantage Renal Cell Carcinoma course, American Association for Cancer Research

National/International

5/2024 Course director and faculty
Audience: American Urologic Association, 2024 Annual Meeting
Optimizing Care for Women with Bladder Cancer: Female Cystectomy and Preservation of Sexual Function

## Workshops /seminars

JHMI/Regional

2/2021 Content developer and speaker
Teamwork in Women's Bladder Cancer: The Advantage of the Multidisciplinary Approach, Greenberg Bladder Cancer Institute
https://www.hopkinsmedicine.org/greenberg-bladder-cancer-institute/about-bladder-cancer/virtual_grand_rounds.html

3/2021 Content developer and speaker
Newly Diagnosed with Bladder Cancer: Questions I Wish I Had Known to Ask, Greenberg Bladder Cancer Institute

6/2021 Content developer and speaker
Non-Muscle Invasive Bladder Cancer in Women, Greenberg Bladder Cancer Institute

9/2021 Content developer and speaker
Surgery and Bladder Cancer in Women, Greenberg Bladder Cancer Institute

12/2021 Content developer and speaker
Life with and after Bladder Cancer for Women, Greenberg Bladder Cancer Institute

3/2022 Content developer and moderator
Integrative Health and Cancer: Six Considerations, Greenberg Bladder Cancer Institute

6/2022 Content developer and moderator
Interpreting Population Science for Cancer Survivors, Greenberg Bladder Cancer Institute

9/2022 Content developer and moderator
The Complexities of Being Female with Bladder Cancer, Greenberg Bladder Cancer Institute

10/2022 Content developer and moderator
Living in the Now: Using Mindfulness as a Tool to Cope with Cancer, Greenberg Bladder Cancer Institute

12/2022 Content developer and moderator
Now What? Managing Health after Bladder Cancer, Greenberg Bladder Cancer Institute

Invited National/international

3/2017 Content developer and speaker
Women and Bladder Cancer Series, Bladder Cancer Advocacy Network
https://www.facebook.com/watch/244123254014/1879686475652215/

8/2022 Content developer and speaker
The importance of Patient Education When Selecting Treatments for Low-grade UTUC, Urology Times

https://www.urologytimes.com/view/ep-4a-the-importance-of-patient-education-when-selecting-treatments-for-low-grade-utuc

7/2023 Content developer and speaker
Urinary diversions: which one is right for me? Bladder Cancer Advocacy Network
https://bcan.org/selecting-your-best-urinary-diversion-for-women/

## Mentoring

### Pre-doctoral Advisees /Mentees

2014-2017 Raju Chelluri, medical research scholar at National Institutes of Health, currently urologist at National Cancer Insitute
Role: research and career mentorship

2014-2018 Lernik Ohanjanian, postdoctoral research fellow at National Institutes of Health, currently medical director at H3 Biomedicine, MA
Role: career mentorship

2015-2017 Mehrsa Jalalizadeh, postdoctoral research fellow at Johns Hopkins University, formerly MD in Iran, currently PhD student at Universidad Estadual de Campinas, Brazil
Role: research mentorship
Shared publications: RA2

2021-2023 Meghan Mcnamara, MPH, research assistant at Johns Hopkins University
Role: research and career mentorship

### Post-doctoral Advisees /Mentees

2014-2015 Paulina Gorney Wilson, MD, chief resident at George Washington University, currently urologist at Inova Fairfax Hospital, VA
Role: career mentorship

2014-2018 Alice Semerjian, MD, chief resident at George Washington University and urologic oncology fellow at Johns Hopkins University, currently director of urologic oncology at Trinity Health Ann Arbor Hospital, MI
Role: career mentorship

### Educational Program Building / Leadership

2020-present CME question writer, Urology for Doximity

2021-present Organizer, lecturer and moderator, Women's Bladder Cancer Series, Johns Hopkins/Greenberg Bladder Cancer Institute

## RESEARCH ACTIVITIES

### Research Focus

Bladder cancer has been the focus of my research. During my research year in residency at Cleveland Clinic and fellowship at the National Cancer Institute, I was involved in translational research in combination treatments with novel drugs and novel drug delivery systems in the cell and animal model of bladder cancer. Building on the foundation acquired during my fellowship, I have continued collaborative discoveries in the basic science and clinical research in bladder cancer. I serve as a reviewer for multiple urologic journals, scientific committee reviewer for Bladder Cancer Advocacy Network and Women in Urologic Oncology Society. During my tenure with Johns Hopkins University, I have served as a regional PI for multiple clinical trials, and since 2021 I have launched four primary IRB-approved clinical trials at Sibley Memorial Hospital, three of which are multi-center trials. Additionally, working with the leadership of the National Capital Region, Brady Urological Institute and Greenberg Bladder Cancer Center, I am currently developing a regional specimen processing center and translational laboratory at Sibley Memorial Center.

### Research Program Building / Leadership

2014-2019 Member, GUMDROP

- Participated in multi-institutional initiative to integrate research across the DMV area

2015-present Special volunteer, National Institutes of Health, Bethesda, MD

- Collaborated with the Urologic Oncology Branch to promote multi-site research projects on bladder cancer

## SYSTEM INNOVATION AND QUALITY IMPROVEMENT ACTIVITIES

### System Innovation and Quality Improvement efforts within JHMI:

2017 Sibley Hospital Robot Committee: establish cross-discipline requirements for new and ongoing users, set benchmarks to assess quality for users, monitor costs and monitor appropriate utilization of robotic equipment.

- Developed robotic credentialing metrics for surgeons practicing at Sibley Hospital

2021 Johns Hopkins Health System Perioperative Playbook Committee: create standardize definitions, key performance indicators, and calculations for perioperative activities at a system level for items like first case starts, turnover time, ERAS, periprocedural optimization, block utilization, block management, inpatient bed management throughput and patient safety and quality issues. Development of the following policies to date:

- JHM Perioperative Clinical and Administrative Policy Development Process standards
- Turn Over Time (TOT) policy
- Periprocedural/Perioperative Code Status and Limitations of Life Sustaining Treatment Policy
- Perioperative/Procedural Fire Prevention and Management Policy
- External Subcutaneous Devices for Management of Diabetes Mellitus (Includes Insulin Pumps and Continuous Glucose Monitors)
- Block Utilization & Management

2022 Sibley Hospital Clinical Resource Management Committee: quality improvement of collective use of hospital resources.

- Improved length of stay and provider use of patient complexity indicators

2023 Sibley Hospital Operating Room Committee: quality improvement of operating room

## ORGANIZATIONAL ACTIVITIES

### Journal peer review activities

ORCID 0000-0002-3846-0235

2012 BMC Urology
2016 American Family Physician
2018 World Journal of Urology
2018 Urologic Oncology
2018 Urology

### Invited Advisory Committees, Review Groups/Study Sections

2019 Scientific committee member for John Quale Travel Fellowship Program, Bladder Cancer Advocacy Network
2020 Scientific committee member for John Quale Travel Fellowship Program, Bladder Cancer Advocacy Network
2021 Scientific committee member for Young Investigator Awards, Bladder Cancer Advocacy Network
2020 Scientific committee member for John Quale Travel Fellowship Program, Bladder Cancer Advocacy Network
2022 Scientific committee member for Young Investigator Awards, Bladder Cancer Advocacy Network
2022 Scientific committee member for Women in Urologic Oncology
2023 Scientific committee member for Young Investigator Awards, Bladder Cancer Advocacy Network
2023 Scientific committee member for John Quale Travel Fellowship Program, Bladder Cancer Advocacy Network
2024 Scientific committee member for Young Investigator Awards, Bladder Cancer Advocacy Network
2024 Steering committee member for Bladder Cancer Advocacy Network Think Tank
2024 Steering committee member for Mid-Atlantic AUA sectional meeting

### Professional Societies

2011-present Member, American Urologic Association (AUA)
2014-present Member, American Association for Cancer Research (AACR)
2014-present Member, Society of Urologic Oncology (SUO)
2019-present Founding member of Women in Urologic Oncology (WUO), a section of SUO, the objective of which is to advance the recruitment, retention, and promotion of women in Urologic Oncology, with the goal of fostering diversity, equity, and inclusion for the SUO overall.
2023-present Member, Mid-Atlantic AUA health policy committee

### Conference Organizer

5/2022 AUA-GBCI bladder cancer symposium

### Consultantships

2015 Asegn

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|---|---|
| 2015-present | Photocure |
| 2022-present | Urogen |
| 2023-present | CG Oncology |

## RECOGNITION

### Awards, Honors

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|---|---|
| 2020 | Northern Virginia Magazine Top Doctor, peer-nominated award based on internal evaluations, patient reviews, and overall quality of care. |
| 2021 | Northern Virginia Magazine Top Doctor, peer-nominated award based on internal evaluations, patient reviews, and overall quality of care. |
| 2021 | Washingtonian Top Doctor, peer-nominated award for excellence in patient care. |
| 2023 | Northern Virginia Magazine Top Doctor, peer-nominated award based on internal evaluations, patient reviews, and overall quality of care. |
| 2023 | Washingtonian Top Doctor, peer-nominated award for excellence in patient care. |
| 2023 | Women's Achiever Award, presented to women who exemplify excellence and exhibit Johns Hopkins Medicine core values by making outstanding contributions to the field of healthcare and/or surrounding communities. |
| 2023 | Outstanding Clinical Instruction Award, for recognition of teaching excellence to urology residents at George Washington University |
| 2024 | Northern Virginia Magazine Top Doctor, peer-nominated award based on internal evaluations, patient reviews, and overall quality of care. |
| 2024 | JHM Physician of the year nominee, for recognition of high standards in practicing medicine, excellent clinical skills, leadership, and collaborative and innovative spirit. |
| 2024 | Washingtonian Top Doctor, peer-nominated award for excellence in patient care. |
| 2024 | *Mary and Armeane Choksi Scholar*, awarded in recognition of contributions to urologic care and research |
| 2025 | Northern Virginia Magazine Top Doctor, peer-nominated award based on internal evaluations, patient reviews, and overall quality of care. |
| 2025 | Washingtonian Top Doctor, peer-nominated award for excellence in patient care. |

## Invited Talks

### National

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|---|---|
| 2017 | Variant histology in bladder cancer. Urotrack, Philadelphia PA |
| 2021 | Gender considerations in muscle invasive bladder cancer: Considering QOL with definitive therapies. Society of Urologic Oncology, Orlando FL |
| 2022 | Gender considerations in muscle invasive bladder cancer. AUA Bladder Health Alliance, Linthicum MD |

### International

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|---|---|
| 2015 | Radical prostatectomy. Keynote Address. Annual Congress of Armenian Association of Urology, Yerevan Armenia |
| 2021 | Muscle-invasive bladder cancer: the current state of organ preservation. West African Surgical Training Initiative, Web-based |
| 2024 | Management of renal cell carcinoma. American Urological Association, Bogota, Colombia |

## Visiting Professorships

| | |
|---|---|
| 2018 | Yerevan State Medical University, Armenia |
| 2021 | Yerevan State Medical University, Armenia |
| 2024 | Yerevan State Medical University, Armenia |

## OTHER PROFESSIONAL ACCOMPLISHMENTS

### Posters

1. Brian H Irwin, Andre Berger, Ricardo Brandina, David Canes, **Armine K Smith**, Sebastien Crouzet, Georges-Pascal Haber, Kazumi Kamoi, Robert J Stein, Mihir M Desai. Experience with complex percutaneous resections for upper tract urothelial carcinoma. 2009 American Urologic Association Annual Meeting, Chicago, IL
2. Brian R Lane, **Armine K Smith**, Benjamin T Larson, Michael C Gong, Inderbir S Gill, Steven C Campbell, Andrew J Stephenson. Development of chronic kidney disease after nephroureterectomy for upper tract urothelial carcinoma and implications for the administration of cisplatin-based chemotherapy. 2009 American Urologic Association Annual Meeting, Chicago, IL
3. Eddie S y Chan, William A. Larchian, **Armine K. Smith**, John B. Klein, Anil A. Thomas, Warren D. Heston, Amit R. Patel. Targeted contrast ultrasound detection and quantification of vascular endothelial growth factor receptors in bladder cancer. 2009 American Urologic Association Annual Meeting, Chicago, IL
4. Amit R Patel, William A. Larchian, **Armine K Smith**, John B. Klein, Anil A. Thomas, Eddie S y Chan. Quantification of sunitinib's antitumor effects in a localized orthotopic bladder cancer model. 2009 American Urologic Association Annual Meeting, Chicago, IL
5. Eddie S.Y. Chan, Amit R. Patel, **Armine K. Smith**, John B. Klein, Anil A. Thomas, Warren D. Heston, William A. Larchian. Optimizing Orthotopic Bladder Tumor Implantation in a Syngeneic Mouse Model. 2009 American Urologic Association Annual Meeting, Chicago, IL
6. Mary Samplaski, **Armine Smith**, William Larchian, Vinod Labhasetwar, Warren Heston. Successful intravesical therapy for bladder cancer utilizing paclitaxel containing nanoparticles. 2010 American Urologic Association Annual Meeting, San Francisco, CA
7. Nitin Yerram, Dmitry Volkin, Faisal Ahmed, Jeffery Nix, An Hoang, Gopal Gupta, **Armine Smith**, W. Marston Linehan, Adam Metwalli, Peter A. Pinto. Long term outcomes of simultaneous bilateral partial adrenalectomy for pheochromocytomas. 2012 American Urologic Association Annual Meeting, Atlanta, GA
8. Mehrsa Jalalizadeh, MD; Leonardo O. Reis, MD, PhD; Hiroki Ide, MD; Hiroshi Miyamoto, MD, PhD; **Armine K. Smith, MD.** Dysregulation of erβ pathway as a mechanism of bcg resistance in urothelial bladder cancer. 2015 Society of Urologic Oncology Annual Meeting, Washington, DC
9. Raju Chelluri, Piyush K. Agarwal, Leonard M. Neckers, **Armine K. Smith**. Synergistic effect of targeted combination therapy in bladder cancer model using hsp90 inhibitors. 2015 American Urologic Association Annual Meeting, New Orleans, LA
10. Mehrsa Jalalizadeh, Leonardo O. Reis, John L. Silberstein, Hiroshi Miyamoto, **Armine K. Smith**. Tumor necrosis factor-related apoptosis-inducing ligand (trail) potentiates the effect of bacillus calmette-guérin (bcg) in urothelial carcinoma model. 2016 American Urologic Association Annual Meeting, San Diego, CA

### Oral/Podium Presentations

1. **Armine K Smith**, Kenneth W Angermeier. Second stage urethroplasty augmented by oral mucosa: an alternative to revision of the first stage. 2008 American Urologic Association Annual Meeting, Orlando, FL
2. **Armine K Smith**, Benjamin T Larson, Andre Berger, Brian R Lane, Donna E Hansel, Andrew J Stephenson, J Stephen Jones. Is there a role for cytology in the diagnosis of upper tract urothelial cancer? 2009 American Urologic Association Annual Meeting, Chicago, IL
3. **Armine K Smith**, Brian R Lane, Benjamin T Larson, Andre Berger, Donna E Hansel, Michael C Gong, Steven C Campbell, Inderbir S Gill, Andrew J Stephenson. Does the choice of technique for management of the bladder cuff affect oncologic outcomes of nephroureterectomy for upper tract urothelial cancer? 2009 American Urologic Association Annual Meeting, Chicago, IL
4. **Armine K Smith**, William A Larchian, Amit R Patel, Shihua Jin, Eddie S y Chan, Anil A Thomas, John B Klein, Warren D Heston, Vinod Labhasetwar. Nanotechnology-mediated delivery of chemotherapy in the treatment of urothelial carcinoma. 2009 American Urologic Association Annual Meeting, Chicago, IL
5. **Armine Smith**, Bethany Kerr, Eric Klein, Warren Heston, Tatiana Byzova. Role of circulating neoplastic progenitor cells in detection and staging of prostate cancer. 2010 American Urologic Association Annual Meeting, San Francisco, CA
6. **Armine K. Smith**, Martha Ninos, James Peterson, Rabindra Gautam, Maria Merino, Berton Zbar, Laura Schmidt, Gennady Bratslavsky, Inger Rosner, An Hoang, Adam Metwalli, Peter A. Pinto, Ramaprasad Srinivasan, W. Marston Linehan. Hereditary papillary renal cell carcinoma: a 20-year experience in management of a unique hereditary cancer syndrome. 2012 American Urologic Association Annual Meeting, Atlanta, GA

## Philanthropic Activities

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|---|---|
| 2014-present | Sibley Memorial Hospital Foundation |
| 2014-present | The James Buchanan Brady Urological Institute Foundation |

# ARMINE SMITH'S STATEMENT OF COMPENSATION

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

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| IN RE: | ) | |
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| 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**

**ARMINE KARAPETIAN SMITH, MD'S STATEMENT OF COMPENSATION**

Pursuant to Fed. R. Civ. P. 26(a)(2)(B)(vi), Plaintiffs provide the following statement of compensation: In the present action, Armine Karapetian Smith, MD charges $650 per hour for review, $750 per hour for deposition testimony, and $8000 per day for trial. Dr. Smith required a $3,250 retainer.

# ARMINE SMITH'S TESTIMONY HISTORY

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

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| IN RE: | ) | |
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| CAMP LEJEUNE WATER LITIGATION | ) | |
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| 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**

**ARMINE KARAPETIAN SMITH, MD'S LIST OF TESTIMONY**

Pursuant to Fed. R. Civ. P. 26(a)(2)(B)(v), Plaintiffs provide the following list of testimony: To the best of her recollection, Armine Karapetian Smith, MD has testified as an expert at trial or by deposition in the following actions during the previous 4 years:

1. Quaranta v. Smolev et al., Maryland;
2. Estate of Mehmet Aras vs. Northwest United Urology, LLC, Illinois, docket number 17 L 010800;
3. Tyron Tann v. Jefferson Health System, Pennsylvania; and
4. Edward Lancaster v. LAMMICO Insurance Company et al, Louisiana, 24th JDC, docket number 809-045 Div L; and

5. Estate of Steven Agnew v. Primary Care Solutions, New Mexico, 1st JDC, docket number D-101-CV-2022-00285.