Exhibit 411

Specific Causation Expert Report for Allan Howard

Armine K Smith, MD

Director of Urologic Oncology
Assistant Professor of Urology
Sibley Memorial Hospital
Johns Hopkins University School of Medicine
5215 Loughboro Road Northwest
Suite 150
Washington DC, 20016

DocuSigned by:
Armine K. Smith, MD
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Armine K. Smith, MD

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1. Introduction

This report evaluates the cause of the renal cell carcinoma (RCC) in Plaintiff Allan Howard, and whether it is causally related to his exposure to the water at Camp Lejeune. The analysis integrates findings from epidemiology, toxicology, and mechanistic research, with emphasis on competing risk factors in his 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 reviewed and relied upon in forming my opinions in this matter are found on the attached materials considered list.

5. Causation Standard

Under the statute at issue in this case, 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."

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 as I understand it and it informs this causation analysis.

6. Patient Background

Date of Birth: [redacted] 1959

Medical History:

- **Renal Cell Carcinoma (RCC)**: Diagnosed in 2008 at age 49 (T1bN0M0, Fuhrman Grade 2).
- **Non-Hodgkin Lymphoma (NHL)**: Diagnosed in 2023, treated with RCHOP therapy.
- Basal and squamous cell carcinoma, hypothyroidism, and hyperlipidemia.

Family History: Pancreatic cancer in father; skin cancer in mother.

Social History: Smoked one pack per day for two years (ceased at young age); retired police officer with documented exposure to contaminated drinking water at Camp Lejeune from 1977-1979.

7. Exposure Assessment

Cumulative Contaminant Levels at Camp Lejeune (Days on Base 449):

- Tetrachloroethylene (PCE): 251 µg/L
- Trichloroethylene (TCE): 5,937 µg/L
- Vinyl Chloride (VC): 343 µg/L
- Benzene (BZ): 70 µg/L

8. Factual History

Mr. Howard enlisted in the Marine Corps in 1977, and he went to Camp Lejeune directly after completing boot camp at Parris Island. Howard Dep. 12:10-13:6. He joined for duty at Camp Lejeune on September 4, 1977, and served there for the remainer of his time in the Marines, until May 30, 1981. 00490_HOWARD_VBA_0000000794. Mr. Howard was transferred from Mainside to Camp Geiger in February of 1979.

From September 1977 to around February 1979, Mr. Howard resided in the Mainside Barracks in an H-style building which he believes was on K Street. Howard Dep. 17:6-8. He ate all his meals

at the mess hall across the street from his barracks and drank two glasses of water or Kool-Aid with each meal. Howard Dep. 28:16-25; 30:9-31:7. He was assigned as a machine gunner (0331) in Charlie Company First Battalion, Second Marine Division, which operated primarily out of Hadnot Point. Howard Dep. 13:16-20.

Mr. Howard spent approximately 10 days per month in the field, where they drank from water buffaloes and canteens. Howard Dep. 17:13-24. He estimates he drank up to 6 to 7 canteens per day for field training. Howard Dep. 32:4-34:14.

Mr. Howard showered at least once a day and twice a day if he had physical training; each shower would be 5-10 minutes. Howard Dep. 24:3-6; 27:18-23. About once a week, he would clean his weapon in the shower for 30-45 minutes. Howard Dep. 25:24-26:25. Mr. Howard did his own laundry using the machines in his barracks about once a week. Howard Dep. 44:11-45:15. He also trained in indoor swimming pools once a year within Hadnot Point for a couple of days. Howard Dep. 45:19-47:8.

When Mr. Howard was working and living at Hadnot Point, he had leave periods as well. 00490_HOWARD_NARA_0000000112. He was also deployed in 1978 on the USS Trenton. Howard Dep. 15:12-19. Excluding deployments and leave, Mr. Howard's spent approximately 449 days living at Camp Lejeune.

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

The levels of TCE, PCE, vinyl chloride, and benzene in the Camp Lejeune water supply far exceeded the EPA's maximum contaminant levels (MCLs). TCE levels, for example, were as high as 546 µg/L for Mr. Howard, which is over 100 times the current MCL of 5 µg/L. PCE levels reached up to 24 µg/L for Mr. Howard, exceeding the MCL of 5 µg/L by almost 5 times. Vinyl chloride levels peaked at 33 µg/L for Mr. Howard, which is over 15 times higher than the MCL of 2 µg/L. Benzene levels peaked at 6 µg/L for Mr. Howard, which is higher than the MCL of 5 µg/L.

The specific concentrations of the water Mr. Howard was exposed to are found in the below chart:

Exposure Dates	Total Days	Exposure Location (Work and Residential)	TCE (ug/l-M)	PCE (ug/l-M)	VC (ug/l-M)	BZ (ug/l-M)
9/4/1977-9/30/1977	27	Hadnot Point	338	13	18	4
10/1/1977-10/31/1977	31	Hadnot Point	69	2	3	4
11/1/1977-11/18/1977, 11/29/1977-11/30/1977	20	Hadnot Point	544	22	30	4
12/1/1977-12/31/1977	31	Hadnot Point	513	21	28	4
1/1/1978-1/31/1978	31	Hadnot Point	250	10	14	4

3/7/1978-3/31/1978	25	Hadnot Point	352	15	20	3
4/1/1978-4/30/1978	30	Hadnot Point	231	9	13	5
5/1/1978-5/31/1978	31	Hadnot Point	278	12	16	4
6/1/1978-6/30/1978	30	Hadnot Point	333	14	19	3
7/1/1978-7/13/1978, 7/29/1978-7/31/1978	16	Hadnot Point	388	17	23	3
8/1/1978-8/31/1978	31	Hadnot Point	475	20	28	4
9/1/1978-9/30/1978	30	Hadnot Point	364	16	22	4
10/1/1978-10/31/1978	31	Hadnot Point	74	3	4	4
11/1/1978-11/30/1978	30	Hadnot Point	544	24	33	5
12/1/1978-12/28/1978	28	Hadnot Point	546	24	33	4
1/13/1979-1/31/1979	19	Hadnot Point	268	12	16	6
2/1/1979-2/8/1979	8	Hadnot Point	370	17	23	5
	449		5,937	251	343	70

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. Mr. Howard's 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 Mr. Howard based on the following assumptions for amounts of ingestion: 1L per day, ATSDR's assumptions of ingestion, Mr. Howard's deposition statements and average ingestion from field training manuals put out by branches of the military.

The total ranges of exposures for Mr. Howard, according to these charts, is listed 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

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 in this case. A summary of some of the evidence is below and is used for purposes of weighing the potential harmful effects of the exposure Mr. Howard 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 cohort 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 Evidence

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]

Mr. Howard's levels of TCE are significantly higher than the levels in the literature that show a causal association between TCE and RCC. According to Bove 2014a's cumulative exposure charts, Mr. Howard would have been categorized as being in the "medium" exposure group for TCE.[7] This was associated with a 1.21 HR.[7] Mr. Howard meets and exceeds other levels in the literature causally associating TCE with kidney cancer.

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.

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.[8]

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.[9] 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.[9]

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.[10]

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.[11] 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).[12] 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.[13]

ii. 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.[14] 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.[7] Mr. Howard would meet the medium exposure group threshold with a RR of 1.82.

iii. 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.

iv. Conclusion

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

Mr. Howard's levels of PCE are significantly higher than the levels in the literature that show a causal association between PCE and RCC. According to Bove 2014a's cumulative exposure charts,

Mr. Howard would have been categorized as being in the "Medium" exposure group for PCE.[7] This corresponded to a HR of 1.82.[7] Mr. Howard meets additional levels of PCE exposure shown in the literature to be associated with kidney cancer.

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., TCE) 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 in men exposed to benzene.[16] There are other studies as well showing this causal relationship, including the Camp Lejeune studies.[7,14]

Vinyl Chloride has been less studied then 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 in men with an OR of 2.0 (95% CI: 1.2 – 3.3).[16] Further, Bove et al. 2014a supports this causal relationship.[7]

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

Mr. Howard's levels of Benzene and VC are higher than the levels in the literature that show a causal association between those chemicals and RCC. According to Bove 2014a's cumulative exposure charts, Mr. Howard would have been categorized as being in the "medium" exposure group for both Benzene and VC.[7] These correspond to HR of 1.38 (Benzene) and 1.61 (VC).[7]

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 for TVOCs at Camp Lejeune and kidney cancer. Bove 2014a.[7] The HRs for this analyses were 1.42 (low exposure), 1.44 (medium exposure) and 1.54 (high exposure).[7]

The epidemiological and occupational studies collectively underscore the link between TCE, PCE, benzene, and vinyl chloride exposures and renal cell carcinoma. Findings from Camp Lejeune studies, occupational analyses, and dose-response models reinforce the carcinogenic roles of these chemicals, particularly in populations with high or prolonged exposures. Mr. Howard's exposure meets the medium exposure groups from each of the four chemicals in the Bove 2014a study.[7] These categories correspond to significant increased risks of kidney cancer, as described above.

Mr. Howard would meet and exceed other levels in the literature relating to the toxins at issue and their association with kidney cancer.

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.[7,17]

11. Patient-Specific Considerations

Age and Tumor Size: Mr. Howard's diagnosis at age 49 and the size of his tumor (T1b) are unusual circumstances for sporadic RCC. RCC typically occurs in older individuals. These factors suggest a significant environmental contribution, particularly exposure to carcinogens like TCE.

Latent Period: The 29-year latency between exposure (1977-1979) and diagnosis (2008) is consistent with TCE's long latency period for cancer development.

Exposure: Mr. Howard's TCE exposure (5,937 µg/L) far exceeds safe thresholds and aligns with levels implicated in RCC cases. The reports of Drs. Hatten and Bird detail the levels at which the chemicals at issue have been 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**[9]
- **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[7]**
- **Cumulative exposure of 1 - 155 µg/L-month of PCE[7]**
- **Cumulative exposure of 1 – 4,600 µg/L-month of exposure to all compounds at Camp Lejeune[7]**
- **Cumulative exposure of 3,100 – 7,700 µg/L-month of TCE[7]**
- **Cumulative exposure of 155 - 380 µg/L-month of PCE[7]**
- **Cumulative exposure of 4,600 – 12,250 µg/L-month of exposure to all compounds at Camp Lejeune[7]**
- **Cumulative exposure greater than 7,700 µg/L-month of TCE[7]**
- **Cumulative exposure greater than 380 µg/L-month of PCE[7]**
- **Cumulative exposure greater than 12,250 µg/L-month of exposure to all compounds at Camp Lejeune[7]**
- **Approximately 18 months of residence on base from 1975 to 1985[7]**
- **Employment on base for 2.5 years[14]**
- **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]**

Mr. Howard's exposure meets and exceeds many of the 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

Further, as shown above through the deposition testimony of Mr. Howard, his exposure to these chemicals at these concentrations was substantial. Mr. Howard was, on a daily basis, frequently and consistently exposed to these chemicals through all three known routes of exposure. Mr. Howard was drinking the water at each meal and when he was training, he was showering and inhaling the toxic chemicals at least daily and often multiple times a day (other than when he was in the field training) and he was exposed to the chemicals on his skin daily. This shows a substantial exposure that is more likely than not causally related to the kidney cancer he developed. I was able to draw my conclusions as to the substantial nature of his exposure from the records reviewed in this case, his deposition testimony and the concentrations in the water at the times Mr. Howard was exposed.

Additionally, the charts from Plaintiff's expert Kelly Reynolds indicate a substantial exposure. For example, Dr. Reynolds calculated that Mr. Howard would have ingested 668,552 ppb of TCE assuming the average ingestion listed in the ATSDR studies. Dr.. Reynolds calculated Mr. Howard's ingestion to be 660,782 ppb of TCE assuming the facts from Mr. Howard's deposition testimony. Dr. Reynold's charts details that if the average ingestion from field manuals published by the United States military were used as the assumption, Mr. Howard's ingestion of TCE would reach 1,019,982 ppb of TCE.

These numbers are demonstrative of the substantial exposure for Mr. Howard and an exposure that is clearly causally related to the development of his kidney cancer. These numbers are strengthened by the fact that these charts only take into account one exposure route: ingestion. Mr. Howard would have been exposed via inhalation and dermal exposure. When factoring in those routes of exposure, Mr. Howard's actual exposure 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. Tobacco Use

Smoking is a recognized risk factor for RCC, increasing relative risk by approximately 1.5 times. However, Mr. Howard's limited smoking history of 2 pack-years (two years, ceased over three decades before diagnosis) suggests minimal contribution to his RCC risk compared to TCE exposure. **2 pack-years** represents a very low cumulative exposure, especially when compared to thresholds associated with significant cancer risk. Theis et al. (2008) indicate that individuals with 20 or more pack-years of smoking have an increased risk of RCC.[20]

Additionally, research indicates that smoking's carcinogenic impact diminishes significantly with long-term cessation, making its role in Mr. Howard's case negligible compared to high-level environmental exposures.[21,22]

b. Family History

While his father's pancreatic cancer may indicate genetic susceptibility to pancreatic cancer, no hereditary kidney cancer syndromes (e.g., VHL disease) are documented in Mr. Howard's family history. Genetic predisposition plays a minimal role in sporadic RCC cases compared to environmental and lifestyle factors.

c. Occupational Exposures

Mr. Howard's post-military occupation as a police officer did not involve significant exposure to nephrotoxic chemicals. Unlike industrial or solvent-heavy professions, his occupational history does not contribute to RCC risk, isolating Camp Lejeune contaminants as the primary causative factor. As stated above, Mr. Howard's exposures to the toxins at Camp Lejeune were substantial and occurred over a prolonged period of time. There simply is no other risk factor for Mr. Howard that is as likely as this exposure in terms of a likelihood of being causally related to his kidney cancer.

There are no other known risk factors for kidney cancer that are applicable to Mr. Howard. The only other risk factors for RCC are age, race, obesity, hypertension and other environmental exposures. Mr. Howard did not have any of these risk factors to discuss.

Finally, I have considered the Defendant's contentions as stated in their supplemental answers to interrogatories as to other potential causes of Mr. Howard's kidney cancer in the below section.

d. Conclusion

Mr. Howard's RCC is more likely than not caused by the water at Camp Lejeune, which was contaminated with TCE, PCE, benzene and vinyl chloride. This is supported by robust epidemiological and mechanistic evidence

14. Reponses to the Defendants Supplemental Answers to Interrogatories

In response to Interrogatories sent by the Plaintiff, Defendant has stated the following may have been contributory to Mr. Howard's kidney cancer, to the exclusion of Mr. Howard's exposure to the water at Camp Lejeune:

a. Plaintiff's Smoking History

As described above, this is not likely related to the development of Mr. Howard's kidney cancer because his smoking history was minimal. He smoked only one pack per day for two years and ceased at an early age. This amounts to 2 pack-years. This was more than three decades before his RCC diagnosis. Two pack-years represent a very low cumulative exposure, and epidemiological studies demonstrate that smoking's carcinogenic impact diminishes significantly with long-term cessation.[21,22] Given the prolonged latency and the far more substantial exposure to TCE, PCE, benzene, and vinyl chloride at Camp Lejeune, smoking's role in his cancer development is negligible.

b. Potential Chemical Exposures in Beirut, After a Train Derailment and During Maintenance of a Motorcycle

Beirut Exposure: According to Mr. Howard's deposition, he stated that he never lived in the Middle East and was not deployed to Beirut. Therefore, any claims regarding chemical exposures from Beirut are unfounded and irrelevant.

Train Derailment Exposure: Mr. Howard testified that his exposure during the 1986 train derailment in Miamisburg, Ohio, was brief and incidental. While the event involved a fire caused by phosphorus in a railroad car, Mr. Howard and other police personnel wore basic paper masks while managing the scene. This limited exposure was short-term and insufficient to cause long-term health effects like kidney cancer. This is especially true compared to his prolonged and intense exposure to contaminants at Camp Lejeune.

Motorcycle Maintenance: Mr. Howard stated that he uses protective gear, including gloves, an N-95 mask, and eye protection, during motorcycle maintenance. The chemicals he uses, such as citrus-based cleaners and dish soap, are low in toxicity and not known to be carcinogenic. He does not use petroleum-based or industrial solvents for maintenance. These controlled and infrequent exposures are unlikely to be significant contributors to the development of his kidney cancer.

As a result, this is not likely related to the development of Mr. Howard's kidney cancer because the duration and intensity of these exposures do not meet the threshold for significant risk. In

contrast, the exposure to Camp Lejeune's water was prolonged and at levels known to cause genotoxic effects directly implicated in RCC development.

c. Plaintiff May Have Had a Previous Basal Cell Carcinoma

This is not likely related to Mr. Howard's kidney cancer because basal cell carcinoma is a localized skin cancer that does not share common etiological pathways with RCC. Basal cell carcinoma is associated with UV radiation exposure rather than the nephrotoxic effects of TCE, PCE, benzene, and vinyl chloride. It does not increase the risk of renal malignancies.

d. Plaintiff's Mother's Skin Cancer and Maternal Aunt's Colon Cancer

This is not likely related to Mr. Howard's kidney cancer because there is no evidence of hereditary kidney cancer syndromes (e.g., von Hippel-Lindau disease) in his family history. His mother's and aunt's cancers are unrelated to renal cell carcinoma and do not indicate a familial predisposition to kidney cancer.

e. Plaintiff's Exposure Levels and Latency

Defendant states "Plaintiff's exposure to water at Camp Lejeune may not have been significant enough to cause the alleged illness or injury, and the length of time (latency period) between any exposure to the water at Camp Lejeune and the onset of the Plaintiff's illness or injury may indicate an alternative cause or that the cause of the Plaintiff's illness or injury is idiopathic."

This is not likely the reality for Mr. Howard because his exposure to Camp Lejeune's contaminated water far exceeded the levels in the literature and science that causally relate TCE, PCE, benzene, and vinyl chloride to kidney cancer. TCE levels at Camp Lejeune, for example, exceeded many of the epidemiology studies and other evidence showing the levels at which these toxins are known to cause kidney cancer. Additionally, there is little question that Mr. Howard's exposure should be considered substantial and more likely than not associated with significant increased risks for kidney cancer.

Finally, the latency period of 29 years between exposure and diagnosis aligns with the latency observed in TCE-related kidney cancer cases.

This analysis of Defendant's contentions is incorporated into the differential diagnosis above as well.

15. Mr. Howard's Damages

I will discuss Mr. Howard's harms suffered as a result of the Camp Lejeune water and Mr. Howard's kidney cancer. This includes the medical treatment related to his kidney cancer, the surgery required to remove his kidney and the typical medical course for individuals like 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 for Mr. Howard's treatment and care related to his kidney cancer is fair and reasonable and this treatment was medically necessary.

16. Conclusion

Considering Mr. Howard's substantial exposure to TCE, PCE, benzene and vinyl chloride, prolonged latency period, and consistent epidemiological and mechanistic evidence, it is more likely than not that his RCC was caused by environmental contaminants at Camp Lejeune. The unusual circumstances of his young age at diagnosis and large tumor size without known genetic predisposition to renal carcinoma further support the significance of toxic exposures in his cancer etiology. Competing risk factors such as minimal smoking history and familial cancer background 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

Personal Data

Business Address 5215 Loughboro Rd, NW, Washington, DC 20016
Tel 202-660-5567
Fax (optional) 202-660-7083
E-mail asmit165@jh.edu

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
2. Greene KL, Elkin EP, **Karapetian A,** Duchane J, Carroll PR, Kane CJ: Prostate biopsy tumor extent but not location predicts recurrence after radical prostatectomy: results from CaPSURE. J Urol. 2006 Jan;175(1):125-9. *Engagement with discovery and data analysis
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4. Lane BR, ***Smith AK,** Larson BT, Gong MC, Campbell SC, Raghavan D, Dreicer R, Hansel DE, Stephenson AJ. Chronic kidney disease after nephroureterectomy for upper tract urothelial carcinoma and implications for the administration of perioperative chemotherapy. Cancer. 2010 Jun 15;116(12):2967-73. (*Joint first co-authorship)
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15. Baraban EG, Vlachou E, Patel S, Kates M, Johnson B, **Smith A,** Shenderov E, Sharma S, Denmeade SR, Brame A, Han M, De Marzo AM, Matoso A, Hoffman-Censits J. Nectin-4 Expression in Prostatic Adenocarcinoma: An Immunohistochemical Study. Prostate. 2025 Jan 2. * Project design and writing
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Review Articles [RA]

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2. Jalalizadeh M, **Smith AK,** Miyamoto H. Intravesical Bacillus Calmette-Guérin Therapy for Bladder Cancer: Molecular Mechanisms of Action. Clinical Immunology, Endocrine & Metabolic Drugs. 2016 Mar 31. ISSN: 2212-7089. *Project design and writing
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Case Reports [CR]

1. **Smith AK,** Hansel DE, Klein EA. Epidermoid cyst of the testicle. Urology. 2009 Sep;74(3):544.
2. Smart A, Wynne M, Baraban E, Ged Y, **Smith A.** Metastasis to the Bladder: A Rare Site of Recurrence of Renal Cell Carcinoma. Case Rep Urol. 2022 Jun 17;2022:4339270.
3. Agrawal P, Baraban E, de la Calle C, Patel S, Kamat AM, **Smith A.** Primary mesonephric adenocarcinoma of the bladder: A case report. Urol Case Rep. 2022 Apr 26;43:102096.
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5. Azari S, Wolfman D, **Smith A.** Hand, foot, and mouth disease presenting with a testicular mass in an adult. Can J Urol. 2024 Apr;31(2):11854-11857. PMID: 38642464.

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.
3. Guo CC, **Smith AK,** Pavlovich CP. The Kidney: A Comprehensive Guide to Pathologic Diagnosis and Management. Chapter 6/Familial Forms of Renal Cell Carcinoma and Associated Syndromes. Springer 2015.
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5. **Smith AK,** Matin SF, Jarrett TW. Wein: Campbell-Walsh Urology, 12th ed. Chapter. 99/Surgical Management of Upper Urinary Tract Urothelial Tumors. Elsevier 2020.
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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.
3. McConkey DJ, Barb JJ, **Smith AK,** Sears CL. Microbiome-based Therapeutics: Cutting-edge Innovation or Perpetual Promise? Eur Urol Focus. 2024 Dec 7:S2405-4569(24)00248-7.

Media Releases or *Invited Interviews [MR] 1

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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.beneficiamo.com/2022/12/20/health-systems-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
Asthma in adults. 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

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

RECOGNITION

Awards, Honors

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

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

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

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

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

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

IN RE:	)	
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	)	
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.