Exhibit 357

Specific Causation Expert Report for Jimmy Laramore
John P. Sfakianos, M.D.

Urologic Oncologist
Professor of Urology
Icahn School of Medicine at Mount Sinai, New York, NY
1425 Madison Avenue, 6th Floor, Room L6-58
New York, NY 10029

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John Sfakianos, MD

I. Background

I graduated from the State University of New York at Buffalo in 2007. I completed my residency in urology in 2012 and a fellowship in urologic oncology in 2014. I am board certified in urology. Currently, I am a professor in urology and the co-chair of the Investigator Initiated Clinical Trials Review Committee at the Icahn School of Medicine at Mount Sinai. As part of my practice, I regularly diagnose and treat patients with bladder cancer. Approximately 70% of my clinical practice is focused on bladder cancer. My practice includes clinical care, surgical care, performing over 150 trans-ureteral resection of bladder tumors and over 50 radical cystectomies every year, and serving as a principal investigator in several clinical trials focused on identifying novel therapeutics for bladder cancer patients. I also have multiple federal funded grants to study the role of BCG in treating patients with non-muscle invasive bladder cancer. Bladder cancer is a disease of environmental exposure thus I often have consultation with my patients regarding their suspected exposures to various chemicals leading to their cancer diagnosis. Approximately 50% of the patients who develop bladder cancer is due to their exposure to cigarettes and the other half to a variety of other carcinogens.[1]

My research has focused on mouse modeling of bladder cancer, which is a carcinogen induced model. N-Butyl-N-(4-hydroxybtyl) nitrosamine (OH-BBN) is an alkylating agent, which is placed in drinking water of mice leading to the production of invasive tumors within 20 weeks. OH-BBN is a carcinogen, which is derived from N-nitroso a compound found in cigarette smoke. For additional discussion of my qualifications, please refer to my curriculum vitae, which is included with this report. All my opinions in this case are to a reasonable degree of medical certainty. My opinions are based upon his review of the materials listed in my forthcoming materials considered list, which include pertinent medical records and scientific papers. All my opinions are based on my education, training and experience and stated to a reasonable degree of scientific and medical certainty. I reserve the right to supplement my opinions if additional information becomes available that may be relevant to my opinions.

II. Mandate

I was asked to provide my opinion on the causation of Jimmy Laramore's diagnosis of bladder cancer. In order to provide this opinion, I reviewed and relied on the following documents and materials:

Medical records for Mr. Laramore, including from:

- The Urology Institute at Renaissance;
- Johns Hopkins Reference Laboratories;
- Advanced Urology Institute; and
- Urology Associates of Southern Mississippi;

[1] Freedman et al, Association between smoking and risk of bladder cancer among men and women JAMA 2012 Sep 13.

Litigation records for Mr. Laramore, including:

- The short form complaint filed in this case;
- The deposition transcript of Jimmy Laramore;
- The deposition transcript of Dr. David Alonzo;
- The deposition transcript of Dr. Warren Hitt;
- The deposition transcript of Dr. Albert Ruiz;
- The deposition transcript of Dr. David Spencer;
- Defendant United States of America's Supplemental Response to Plaintiffs' Leadership Group's First Set of Interrogatories to Defendant United States of America Concerning Track 1 Plaintiff Jimmy C. Laramore
- The general causation reports of Dr. Benjamin Hatten, Dr. Stephen Bird (including his supplemental report), Dr. Kathleen Gilbert, Dr. Laura Plunkett, and Dr. Stephen Culp;
- The exposure report of Kelly Reynolds, MSPH, PhD
- The exposure report of Dr. Stephen Bird
- The exposure report of Dr. Benjamin Hatten

Additional records, including:

- Scientific and medical literature referenced herein;
- Other documents listed on my forthcoming materials considered list.

It is my understanding that the statute at issue in this case states that there are two ways to prove causation:

> (1) Standards – To meet the burden of proof described in paragraph (2) 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 a causal relationship exists; or
> (B) sufficient to conclude a causal relationship is at least as likely as not.

This standard was considered in my approach to determining whether Mr. Laramore's exposure to the water at Camp Lejeune was at least as likely as not the cause of his bladder cancer. Reasonable medical professionals in my field apply the same or similar standards.

III. Methodology

In order to come to a determination that a chemical exposure causes bladder cancer, I first look to determine if there is enough evidence to establish a causal relationship between the subject chemical(s) and bladder cancer. As a urologic oncologist focused on bladder cancer both clinically and in the laboratory, I am well-versed in the suspected causes of bladder cancer in humans and therefore rely on my knowledge, training and experience. In addition, I have relied on accepted, reputable sources such as the ATSDR, IARC Monographs, and the EPA. Finally, I also relied on the general causation reports issued in this case relating to bladder cancer, and the

additional reports related to exposure. Once I come to a determination that there is enough evidence to establish a causal relationship, I proceed to perform a differential etiology on the individual to determine if his or her particular exposure more likely than not caused the development of his bladder cancer.

"In a differential etiology, an expert first determines other known causes of the disease in question and then attempts to ascertain whether those competing causes can be "ruled out" as a cause of plaintiff 's disease…" Reference Manual on Scientific Evidence, Third edition, p. 618. I regularly employ a differential etiology in my practice as part of my clinical work to help improve the treatment options and outcomes of my patients.

During an encounter for bladder cancer, a physician will often try to establish a differential etiology for the development of the patient's disease. (An important distinction, this is a differential etiology, not a diagnosis. The diagnosis is bladder cancer, the cause is the etiology.) This is a thorough, but not exhaustive line of questioning, because the cause of the disease is of less importance than the therapy plan of the disease for the treating physician. The most immediate goal of the visit for a treating physician is to develop the appropriate treatment plan for the patient. However, in a disease like bladder cancer, where the etiology is almost always from an exposure, it is worthwhile to identify the exposure. This will make the patient aware and permit them to avoid the exposure going forward if it is possible. It may also afford an opportunity to warn others, particularly family and coworkers, with similar exposures. Gathering this data also reveals patterns that may be important to the health of society as a whole. In fact, this revelation of patterns leads to the science of epidemiology discussed above.

It is not uncommon for bladder cancer to develop in a patient with more than one risk factor for the development of bladder cancer. Behavior based, environmental, and occupational exposures are well-established risk factors for the development of bladder cancer, and the American Cancer Society recognizes that multiple exposures – such as smoking and workplace exposures – "can act together to cause bladder cancer."[2] These risk factors can act in concert with one another and have additive effects. In Mr. Laramore's case, as I discuss later in this report, he has at least two risk factors for the development of bladder cancer.

It is important to note that the quality of information matters when performing a differential etiology. The more uncertainty surrounding a particular risk factor, the more speculation is introduced into the differential etiology. If insufficient information exists concerning a risk factor or it requires a number of assumptions, then its value in considering it as a risk factor is substantially diminished or depleted altogether. Speculative or conjectural information is of little value in a differential etiology.

I am being compensated at a rate of $550 per hour.

[2] https://www.cancer.org/content/dam/CRC/PDF/Public/8558.00.pdf

IV. Mr. Laramore's factual background

a. Family history

Jimmy C. Laramore was born on February 26, 1959, and is from Fort Mitchell, Alabama. Laramore Dep. 32:4-7. He has been married to his wife, Patricia Laramore, for over 10 years, and they currently live in Biloxi, Mississippi. *Id*. 20:1-9. Mr. Laramore does not have a significant family history of cancer. Of his direct family members, Mr. Laramore is not aware of any family history of cancer besides his mother, who was diagnosed with breast cancer.[3]

b. Occupational and environmental exposures

Mr. Laramore enlisted in the Marine Corps after high school in 1977.[4] After some initial training, Mr. Laramore was sent to his first duty station in El Toro, California toward the end of 1977 or early 1978.[5] At El Toro, Mr. Laramore spent time fixing weapons but does not appear to have been involved in cleaning them.[6] Then, Mr. Laramore was sent to Camp Fuji in Japan and subsequently to Camp Pendleton; in both locations Mr. Laramore was a small arms repairman.[7] After completing three years of service with the Marine Corps, Mr. Laramore worked at Fieldcrest Mill as a slasher helper – a job that involved loading and unloading a machine with yarn but no actual maintenance of the machine itself.[8] Subsequently, Mr. Laramore entered the civil service as a small arms repairman, which involved similar repair work as his time in the Marines Corps but also included cleaning weapons.[9] It is unclear what was used to clean the weapons, though Mr. Laramore notes that it was a "solvent" and that he used long gloves.[10]

Mr. Laramore returned to the Marine Corps for a second three-year commitment in December 1983.[11] The Marines sent him to Camp Lejeune, where he served as a small arms repairman but did not clean weapons.[12] In late 1984, Mr. Laramore was sent to Iwakuni, Japan until late 1985, and then to Barstow, California; in each location he served as a small arms repairman.[13]

After his honorable discharge, Mr. Laramore took on a number of different jobs. Mr. Laramore worked in housing maintenance for four years before getting his CGL

[3] Laramore Dep. 30:21-31:19
[4] Laramore Dep. 33:15-34:7
[5] Laramore Dep. 39:20-40:1
[6] Laramore Dep. 40:2-19
[7] Laramore Dep. 41:23-42:13; 44:7-25
[8] Laramore Dep. 46:3-47:7
[9] Laramore Dep. 47:13-48:23
[10] Laramore Dep. 48:21-49:9
[11] Laramore Dep. 52:23-53:4
[12] Laramore Dep. 54:1-54:7
[13] Laramore Dep. 82:18-83:10; 83:11-25

license and becoming a truck driver for approximately 10 years.[14] Mr. Laramore reports delivering various kinds of freight, including one instance of 12% hydrogen peroxide.[15] Mr. Laramore also reported instances of delivering gas within this ten year period, where his job duties would include filling up the taker with gasoline or diesel. Mr. Laramore denies encountering fumes as the fumes would come out far away from where he would be physically located.[16] Mr. Laramore apparently took a leave of his driving job to work as a salesman for Orkin Pest Control from November 1995 to February 1997.[17] Finally, Mr. Laramore performed various forms of housing maintenance jobs for approximately 7 years until 2007 when went to Iraq and worked as a truck driver as a government contractor. In 2009 he was forced to retire on disability.[18]

c. Medical history

On March 13, 2018, Mr. Laramore was found to have microscopic blood (hematuria) on urine analysis performed by his primary care physician.[19] A CT scan was completed and no etiology for hematuria was identified.[20] After continued identification of microhematuria on urine analysis, Mr. Laramore was referred to a urologist, Dr. David Alonzo, who on 3/30/2020 performed a TURBT.[21] Dr. Alonzo resected two tumors in overlapping sites.[22] The initial pathology was a papillary urothelial carcinoma, invasive, high grade, with the tumor invading the lamina propria and detrusor muscle.[23] Dr. Alonzo explained that he took three biopsies of one of the tumors, with one of the three biopsies indicating that the tumor was muscle-invasive.[24] Dr. Alonzo requested a second opinion on the pathology, which was provided by Dr. Jonathan Epstein of Johns Hopkins Medical Laboratories. Dr. Epstein confirmed invasive high-grade papillary urothelial carcinoma with associated in-situ urothelial carcinoma.[25] Dr. Epstein further reported "[t]here is invasive carcinoma focally involved in thin muscle bundles. It is difficult to distinguish whether these muscle bundles represent muscularis propria (detrusor muscle) that has been partially destroyed by the invasive cancer or muscularis mucosae (lamina propria)" and as a result he stated "[a]dditional tissue sampling is recommended."[26] Dr. Alonzo recommended a repeat TURBT, which was performed by another provider, Dr. Warren Hitt, due to Mr. Laramore's reported extreme circumstances.[27]

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[14] Laramore Dep. 88:9-15; 85:21-25
[15] Laramore Dep. 85:1-7
[16] Laramore Dep. 90:14-92:4
[17] 00594_LARAMORE_WE_0000000004
[18] Laramore Dep. 92:15-93:6
[19] 00594_LARAMORE_VHA_0000004138
[20] 00594_LARAMORE_VHA_0000004131-2
[21] 00594_LARAMORE_VHA_0000001766
[22] 0594_LARAMORE_AUI_0000000003-4
[23] 0594_LARAMORE_AUI_0000000007
[24] Alonzo Dep. 49:10-15
[25] 0594_LARAMORE_AUI_0000000005
[26] *Id*.
[27] 0594_LARAMORE_DHRH_0000000064-66

On an initial consult Dr. Hitt described two potential treatment options – radical cystectomy and radiation therapy / chemotherapy; Mr. Laramore and Dr. Hitt agreed to follow the recommendation of Dr. Alonzo and perform an additional TURBT.[28] Dr. Hitt identified the sites of the previous resection and took additional biopsies.[29] The pathology did not reveal any tumor growth.[30] Dr. Hitt and Mr. Laramore discussed treatment options and to treat his disease as stage I or stage II and decided to proceed as stage I. Dr. Hitt then recommended a course of BCG to prevent recurrence.[31] Mr. Laramore underwent six instillations of BCG treatment and returned to Dr. Hitt for a cystoscopy with a subsequent PET/CT scan.[32] The cytology results identified dysplastic cells and the PET/CT showed results consistent with a viable tumor, both of which indicated the need for an additional TURBT.[33]

Mr. Laramore moved back to Texas from Florida and re-established care with Dr. Alonzo, who recommended performing a surveillance cystoscopy.[34] The cytology report revealed no malignant cells[35] but evidence of one small erythematous lesion not suspicious with NBI light.[36] Dr. Alonzo discussed either performing an immediate TURBT or performing a repeat cystoscopy in three months;[37] Mr. Laramore opted to have a repeat cystoscopy.[38] From then on, Mr. Laramore had regular cystoscopies in approximately 3 month intervals with no evidence of recurrence from June 8, 2021 through September 20, 2022. Mr. Laramore then moved away from Texas and established care with Dr. David Spencer.[39] Mr. Laramore had a cystoscopy on March 23, 2023 with no evidence of recurrence. On October 26, 2023, Mr. Laramore had another surveillance cystoscopy, which revealed evidence of a “papillary-appearing tumor” estimated to be 1 cm that was biopsied and sent for pathology.[40] On December 14, 2023, Mr. Laramore’s bladder was re-biopsied for staging purposes, and a CT scan was ordered.[41] The biopsies were benign and the CT scan found asymmetrical mural thickening of the urinary bladder wall.[42] Dr. Spencer recommended a second induction of BCG treatments which took place over the next several weeks.[43] On May 2, 2024, Mr. Laramore’s first cystoscopy following BCG found no evidence of recurrence.[44] Mr. Laramore returned to Texas and reestablished care with Dr. Alonzo, who scheduled a

[28] 0594_LARAMORE_AUI_0000000019; Hitt Dep. 36:25-37:4.
[29] 0594_LARAMORE_AUI_0000000034
[30] 0594_LARAMORE_AUI_0000000035
[31] 0594_LARAMORE_AUI_0000000045
[32] 0594_LARAMORE_AUI_0000000051-62
[33] 0594_LARAMORE_AUI_0000000057; 0594_LARAMORE_AUI_0000000063-66
[34] 0594_LARAMORE_VHA_0000001774-1777
[35] 0594_LARAMORE_VHA_0000002303
[36] 0594_LARAMORE_VHA_0000001778
[37] 0594_LARAMORE_VHA_0000001782
[38] 0594_LARAMORE_VHA_0000001789
[39] 0594_LARAMORE_VHA_0000002301
[40] 0594_LARAMORE_UA_0000000091-92
[41] 0594_LARAMORE_UA_0000000083-84; 75
[42] 0594_LARAMORE_UA_0000000075; Spencer Dep 46:11-13
[43] 0594_LARAMORE_UA_0000000006
[44] 0594_LARAMORE_UA_0000000169

cystoscopy.[45] The cystoscopy occurred on September 9, 2024, revealed two papillary lesions but was negative for malignant cells.[46]

It appears from the trajectory of Mr. Laramore's most recent medical records that his condition has the potential to worsen. The identification of papillary lesions has the potential to signal the reemergence of bladder cancer, though his most recent medical records would shed light on whether his bladder cancer has recurred.[47] Upon my review, Mr. Laramore's treatment for his bladder cancer was medically necessary and continues to be so due to the risk of recurrence, and the expenses associated with his bladder cancer treatment is reasonable. Given Mr. Laramore's multiple bladder cancer recurrences after the best standard of care therapy available, it is my opinion that he will need continued treatment and possibly require surgical intervention to remove his bladder with the development of a urinary diversion.

V. Exposure assessment

Concerning Mr. Laramore's exposure to volatile organic compounds at Marine Base Camp Lejeune, I have reviewed the report and exposure modeling compiled by Plaintiff's expert, Kelly Reynolds, MSPH, PhD. Dr. Reynolds' modeled levels of exposure support my opinion that Mr. Laramore had substantial exposure to toxins on Camp Lejeune. Her cumulative exposure charts set out reasonable estimates, based on objectively derived data, and the best available evidence to predict exposure levels. And the levels compiled by Dr. Reynolds demonstrate that Mr. Laramore had substantial exposure to TCE, PCE, benzene, and vinyl chloride.

Dr. Reynolds calculated Mr. Laramore's total days of exposure to the contaminated water on base at 374 days of exposure. And on a monthly basis Dr. Reynolds calculated the total exposure, or cumulative consumption, of the VOCs, and totaled these up for a cumulative exposure for each of the toxins.

Using the ATSDR's estimated consumption of water, and utilizing Dr. Reynolds' calculated days on Camp Lejeune, Mr. Laramore was exposed to the below concentrations, which are well beyond exposures demonstrated to be causative of bladder cancer.

[45] 0594_LARAMORE_0000006624
[46] 0594_LARAMORE_0000006616; 18
[47] I understand that additional medical records have been requested, and upon receipt I will supplement this report.

		Chart 1: 1L	Chart 2: ATSDR marine in training (4.334 L consumption per day)	Chart 3: Deposition informed ingestion activities	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	5,889	172,692	748,447	612,853	1,144,208
PCE	280	8,200	35,539	29,100	54,331
VC	509	14,926	64,689	52,970	98,895
BZ	105	3,095	13,414	10,984	20,507

In concluding that Mr. Laramore had substantial exposure to the chemicals at Camp Lejeune, it is important to note that there are three recognized routes of exposure to these toxins: ingestion, inhalation, and dermal. Dr. Reynolds' model only addresses ingestion. Focusing on only one avenue of exposure can result in an underestimation of the true risk. Specifically, "for typical activities of drinking and showering, each exposure route contributes similar internal doses, and the total internal dose for a 10-minute shower or a 30-bath is greater than that from ingesting over 2L of water."[48] Accounting for exposures via inhalation and dermal routes would dramatically increase the exposure numbers in the below chart compiled by Dr. Reynolds.

I have also reviewed the reports of Drs. Benjamin Hatten and Michael Bird. Dr. Bird's recent report sets out various study sources that have identified levels of exposure that demonstrate toxic levels of TCE, PCE, vinyl chloride and benzene. Viewing Dr. Reynolds' exposure numbers against these demonstrated toxic levels clearly establishes that Mr. Laramore's exposure was significant and substantial. Mr. Laramore exceeds each of the demonstrated levels set out here.

[48] Weisel, C. and Wan-Kuen, J., Ingestion, Inhalation, and Dermal Exposure to Chloroform and Trichloroethylene from Tap Water, Environmental Health Perspectives, Vol. 104, Number 1, 48-51, Jan. 1996.

The opinions of Dr. Reynolds, Dr. Hatten, and Dr. Bird confirm that Ms. Dyer's exposure to the chemicals at Camp Lejeune has been documented in other literature to have a positive association with the diagnosis of bladder cancer.

VI. General causation

Before advancing to the application of a differential etiology for Mr. Laramore, it is important to first recognize whether there is enough evidence to establish whether the chemicals in the water at Camp Lejeune are capable of causing bladder cancer as a general matter.

Numerous regulatory and scientific bodies have recognized that these four chemicals are toxic and capable of causing cancer. IARC recognizes TCE, vinyl chloride, and benzene as having sufficient evidence for carcinogenicity in humans, and that that PCE is probably carcinogenic to humans. IARC noted that the bladder "may be [a] target tissue[] for tetrachlorethylene-induced carcinogenesis in humans…"[49] EPA concluded that "TCE is carcinogenic to humans by all routes of exposure," that is, by ingestion, inhalation, and dermal exposure.[50] Further, EPA concluded that PCE is "likely to be carcinogenic in humans by all routes of exposure" by EPA.[51] Similarly, the National Toxicology Program has recognized TCE as "a known human carcinogen"[52] and PCE as "reasonably anticipated to be a human carcinogen."[53] ATSDR's 2017 Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases found sufficient evidence exists for PCE causing bladder cancer, stating that "the epidemiological studies provide sufficient evidence for causation and are consistent with the mechanistic information that certain genetic polymorphism may enhance the production of genotoxic PCE metabolites in the bladder via the GSH conjugate pathway." While ATSDR did not find sufficient evidence for TCE and bladder cancer, later studies have strengthened the association as noted by Dr. Hatten. As reported by Dr. Hatten and Dr. Plunkett, epidemiological studies have identified elevated bladder cancer diagnoses associated with benzene and vinyl chloride.[54]

As reported by Dr. Hatten, Dr. Plunkett, Dr. Gilbert, and Dr. Bird, both TCE and PCE share similar metabolic pathways: toxic metabolites are eventually excreted from

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[49] International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;106:1-514

[50] Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011

[51] Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012

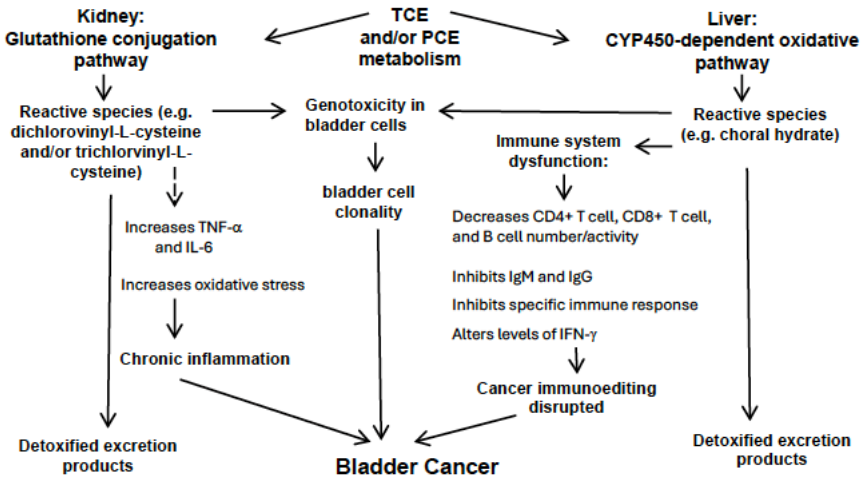
[52] National Toxicology Program (NTP). 2015. Report on Carcinogens monograph on trichloroethylene. Research Triangle Park, NC: National Toxicology Program. RoC Monograph 05

[53] NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service

[54] Hatten pp. 26-29; 31-32; Plunkett ¶ 47

the kidneys into urine where it sits in the bladder until voided.[55] Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well.[56] This means that the toxic metabolites can spend hours in contact with urothelial cells inside the bladder. Below is a figure from Dr. Gilbert explaining the metabolic pathways and outcome for TCE and PCE-induced bladder cancer

Figure 1. Model for TCE and/or PCE-induced bladder cancer



Dr. Gilbert reports that inhalation and dermal exposure from TCE-contaminated water at least doubles ingestion consumption figures (and with similar evidence for PCE).[57] Dr. Gilbert further explains that a mixture of TCE, PCE, and benzene can produce additive effects that can cause bladder cancer in that both TCE and PCE share a similar metabolic pathway and all three chemicals promote chronic inflammation and immunosuppression.[58] Regarding chronic inflammation in particular, Dr. Gilbert concludes that it "is an important driver of bladder cancer and provides support for tumor progression, metastasis, and anti-cancer resistance." In addition, TCE and PCE's can reduce the impact of the body's natural immune response to bladder cancer, which is important given that the most common intravesical treatment used to fight bladder cancer – BCG – essentially activates an adaptive immune response.[59]

Over time, the scientific consensus has progressed to greater certainty, and action, regarding the toxicity of the chemicals at Camp Lejeune. In December 2024 EPA finalized a rule banning on TCE and most commercial uses of PCE under the Toxic Substances Control Act, describing TCE as "extremely toxic" and PCE as "cancer-causing". As noted

[55] Plunkett ¶¶ 33, 43; Hatten p. 39; Bird pp. 17-18
[56] Plunkett ¶¶ 52, 56, 59
[57] Gilbert p. 30
[58] Gilbert p. 32-3
[59] Gilbert Rep. at p. 19-20

by Dr. Bird in his supplemental report, "the EPA determined that any *lesser* restrictions on the use of TCE or PCE would fail to adequately protect public health."[60] Dr. Bird further explained that EPA's safety measures were based on the wastewater concentrations, not consumption, meaning that the risk for those at Camp Lejeune (whose ingested concentrations alone are than the concentrations identified in the EPA rule) is even greater.

Accordingly, there is a sufficient basis to conclude that the chemicals in the water at Camp Lejeune are capable of causing bladder cancer.

VII. Differential etiology

The next step in this analysis is to perform a differential etiology on Mr. Laramore to determine if his exposure to the water at Camp Lejeune is at least as likely as not to cause his bladder cancer.

First, it is helpful to a general background on bladder cancer itself. Bladder cancer arises from the cells lining the urinary system including the bladder, ureter, renal pelvis and prostatic urethra, most commonly in the transitional epithelium (also known as urothelium). Bladder cancer is a disease of carcinogen exposure. As our body encounters toxins it must have mechanisms of which to remove them from the body. Once such mechanism is to filter them in our kidneys. In doing so the carcinogen/toxin is placed into urine for us to then void out of our system. The major issue with this process is that our bladder is a storage organ leading to the urine and carcinogen being exposed to the urothelial (lining of the bladder) in many cases for hours. The carcinogen contact/exposure to the urothelial then leads to the cellular damage, which eventually can lead to cancer formation. Bladder cancer can present as non-invasive bladder cancer (NMIBC) or muscle invasive bladder cancer (MIBC) approximately 70% and 25%, respectively or de novo metastatic cancer in 5%. The primary risk factors for bladder cancer include smoking, exposure to environmental and occupational carcinogens, age, gender, and certain medical conditions. While bladder cancer is treatable when detected early, late-stage diagnosis significantly reduces survival rates.

Bladder cancer can arise from the presence of a single risk factor or a combination of risk factors. Bladder cancer risk factors often interact in a way that magnifies an individual's overall risk, a phenomenon known as synergistic or cumulative risk. For instance, smoking is the most prominent risk factor for bladder cancer, with smokers being three to four times more likely to develop bladder cancer compared to non-smokers. However, when combined with environmental carcinogen exposure, such as that from industrial chemicals like benzene, trichloroethylene (TCE), or perchloroethylene (PCE), commonly found in workplaces or contaminated water supplies, the risk can be significantly higher. Studies show that individuals exposed to both smoking and toxic chemicals, such as those at Camp Lejeune, experience a greater risk of bladder cancer than the sum of the risks posed by each factor alone The interaction between these factors may increase the concentration of carcinogens in the

[60] Bird Suppl. p. 1

bladder, elevate the frequency of genetic mutations, or impair the body's ability to repair cellular damage, thus accelerating the cancerous process.[61] Additionally, genetic predispositions, such as mutations related to Lynch syndrome, can further exacerbate this risk, making it crucial for individuals with multiple risk factors to be monitored closely for early signs of bladder cancer.[62] In this way, the combination of lifestyle choices, environmental exposures, and genetic factors can work together to create a much higher risk for developing bladder cancer.

Interestingly, bladder cancer typically exhibits a significant lag time between exposure to carcinogenic risk factors and the onset of diagnosis, often taking decades for the disease to develop. This delay is primarily due to the slow accumulation of genetic mutations and cellular damage that occurs over time, as carcinogens gradually affect the bladder lining. Chronic exposure to risk factors such as smoking, occupational chemicals, or infections can lead to repeated DNA damage, inflammation, and the gradual formation of abnormal cells that eventually become cancerous. The bladder being a storage organ is exposed to these carcinogens for hours at a time as the bladder fills until voiding ensues. The cumulative effect of these exposures, combined with the bladder's ability to withstand continuous irritation, means that the latency period for bladder cancer is typically long, with diagnoses often occurring 20 to 40 years after initial exposure.[63] That is not to say that bladder cancer cannot arise before 20 years from exposure or after 40 years from exposure.

In this section I explore the known risk factors for bladder cancer and provide references to support the analysis. Next, I consider whether the known risk factors for bladder cancer are applicable to Mr. Laramore. Finally, I consider whether Mr. Laramore's exposure to the water at Camp Lejeune was at least as likely as not the cause of Mr. Laramore's bladder cancer.

[61] Rink et al., Smoking and Bladder Cancer: A Systematic Review of Risk and Outcomes Eur Urol Foc. 2015 Aug;1(1):17-27

[62] Phelan et al, Inherited forms of bladder cancer: a review of Lynch syndrome and other inherited conditions future Oncol 2018 Feb;14(3):277-290.doi: 10.2217/fon-2017-0346.Epub 2018 Jan 18)

[63] Rink et al., Smoking and Bladder Cancer: A Systematic Review of Risk and Outcomes Eur Urol Foc. 2015 Aug;1(1):17-27

Before discussing the risk factors for bladder cancer, it is important to address whether bladder cancer is idiopathic. Idiopathic means that a disease has no known cause and is essentially a diagnosis of exclusion that occurs after other known risk factors are ruled out. I understand and use this term in my medical practice when an individual presents with a condition for which I cannot identify a known cause. While we cannot have complete certainty on the precise cause of many cancers, bladder cancer has well-defined risk factors that explain the vast majority of diagnoses. By and large, blader cancer is a cancer of toxic exposures. We understand that approximately 50% of bladder cancer are due to cigarette smoking, the remainder of the cases once considered to be sporadic are now understood to be secondary to other environmental or occupational exposures leading to the belief that only a small fraction of cases are due to genetic factors alone.[64]

Whenever I perform a differential etiology in my practice, I first consider the relevant risk factors for that disease. If (and only if) I cannot reasonably identify a potential risk factor to cause an individual's disease do I conclude that the cause is idiopathic. As I discuss further in this report, Mr. Laramore was exposed to chemicals at Camp Lejeune that have a known association with the very disease of which he was diagnosed. While I still consider other alternative causes for Mr. Laramore's bladder cancer diagnosis, I do not consider his diagnosis to be idiopathic.

1. Smoking

Smoking is a significant and well-established risk factor for bladder cancer. According to the American Cancer Society, smoking contributes to approximately 50% of all bladder cancer cases in the United States. Smokers are three to four times more likely to develop bladder cancer than non-smokers, with the risk increasing with the intensity and duration of smoking.[65] Cigarette smoke contains a range of carcinogens, including polycyclic aromatic hydrocarbons (PAHs) and aromatic amines, which can cause genetic mutations in the bladder cells. As described above, it is generally understood that the carcinogens from tobacco are absorbed into the bloodstream after being filtered by the kidneys, where they are excreted into the bladder. The initial studies linking cigarette smoking and bladder cancer go back to as early as the 1950's. It is well understood that aromatic amines and their derivatives are the leading carcinogen from cigarette smoking leading to the genetic damage in the bladder causing bladder cancer. The concentrated carcinogens in the urine, which can sit for up to hours at a time, can cause mutations in the DNA of bladder cells, leading to cancer.[66]

[64] Kyle J. Kiriluk M.D., Sandip M. Prasad M.D., M.Phil., Amit R. Patel M.D., Gary D. Steinberg M.D., Norm D. Smith M.D. Bladder cancer risk from occupational and environmental exposures Urologic Oncology: Seminars and Original Investigations, Volume 30, Issue 2, March–April 2012, Pages 199-211)
[65] Freedman 2012
[66] Weiss et al, History of the Relationship between Smoking and Bladder Cancer: A Public Health Perspective Urology. 2022 Aug 14;171:6–10

Despite the recognized connections between smoking and bladder cancer, caution must be exercised. Simply because an individual is a smoker does not mean that smoking is the cause of their bladder cancer diagnosis. Unlike some diseases, bladder cancer is not a signature disease that is solely associated with smoking. Significantly, most smokers do not develop bladder cancer. Freedman analyzed men and women in the United States who smoked and developed bladder cancer with a total population of 467,528 (281,394 men and 186,134 women).[67] Of the study participants, 233,521 (161,435 males and 72,086 females) were former smokers and 67,853 (35,907 males, 31,946 females) were current smokers) for a total of 301,374 current or former smokers. However, the study population had a total of 4,523 (3,896 males, 627 females) total bladder cancer diagnoses. This identifies that incidence of bladder cancer in both smoker and non-smokers to be very low. In Freedman's accompanying meta-analysis of bladder cancer patients the authors identified that, never-smokers represented18% to 44% of the bladder cancer patients, while current smokers diagnosed with bladder cancer ranged from 8% to 63%. In a similar population-based study performed in Australia it identified that by the age of 80 years old an estimated 48.3% of current smokers will develop any cancer compared to 41.1% of never smokers. This study identifies tobacco use does have an increasing risk of overall cancer development, but also importantly the data shows that of the cause for most patients developing cancer by the age of 80 is not related to tobacco use. When specifically analyzing the risk of developing bladder cancer the authors identified a risk of 16.5 vs 36.3 per 100,000 for never vs former smokers, respectively and a risk of 16.5 vs 46.3 per 100,000 for never vs current smokers, respectively. This data shows that the risk of bladder cancer development is influenced by tobacco exposure, but also identifies that tobacco is not the only risk factor leading to bladder cancer.[68]

Mr. Laramore was a smoker with an approximate 30 pack year history prior to his diagnosis. As such, smoking is a potential cause of his bladder cancer and will be weighed in the next section.

2. Environmental and Occupational Exposure to Carcinogens

Environmental exposure to carcinogens is another key risk factor for bladder cancer. These carcinogens include volatile organic compounds (VOCs), aromatic amines, and certain industrial chemicals, which can be found in workplaces and contaminated water supplies.

Occupational exposure is a significant risk factor for bladder cancer, especially for individuals working in industries that involve carcinogenic chemicals. Workers in the manufacturing of dyes, rubber products, and textiles are at an increased risk due to their exposure to aromatic amines, a class of carcinogens strongly linked to bladder cancer. Additionally, individuals working with solvents such as benzene, trichloroethylene (TCE), and perchloroethylene (PCE) are also at heightened risk, as these chemicals

[67] Freedman 2012
[68] Weber et al, Cancer incidence and cancer death in relation to tobacco smoking in a population-based Australian cohort study Int. J. Cancer. 2021;149:1076–1088.

can be absorbed into the body and concentrate in the bladder. Long-term exposure to these substances, particularly in poorly ventilated environments, can lead to chronic bladder irritation and genetic mutations that increase the likelihood of cancer. Furthermore, workers in the metal industry, hairdressers, and those exposed to certain pesticides and industrial waste also face increased risks due to prolonged contact with carcinogenic agent.

Water contamination, particularly in military or industrial settings, also presents a significant risk. For example (and as discussed above), the contamination of drinking water at Camp Lejeune in North Carolina with toxic substances such as trichloroethylene (TCE), perchloroethylene (PCE), and benzene have been associated with an increased incidence of bladder cancer among those who lived or worked there (Morris et al., 2017). TCE, a degreasing agent, and PCE, a solvent used in dry cleaning, are both classified as human carcinogens or suspected human carcinogens by the International Agency for Research on Cancer (IARC). EPA has also banned TCE and most commercial uses of PCE because they are cancer-causing agents and used Camp Lejeune as a key example for why banning these chemicals was necessary.

Mr. Laramore had several potential occupational exposures and a definite water contamination exposure. Of his occupational exposures, the only jobs where Mr. Laramore would have been exposed to a known bladder cancer-causing chemical would be his time as a civil servant and his time as a truck driver. Mr. Laramore's work as a slasher helper and his time working maintenance do not appear to have any appreciable involvement with chemicals associated with bladder cancer. While high exposure to pesticides and herbicides have been linked to bladder cancer[69], Mr. Laramore's role as a salesman for Orkin and his duration does not suggest he had any significant amount of exposure. Mr. Laramore's other military jobs as a small arms repairman, outside of his work as a civil servant and at Camp Lejeune, do not appear to have any involvement with solvents or other cancer-causing chemicals.

The nature of Mr. Laramore's exposure to solvents as a civil servant and to diesel fumes as a truck driver are hard to put into concrete terms. As a civil servant Mr. Laramore used gloves when handling solvents[70], but what is unclear is the ventilation of where he would have been in contact with the solvents. An open air location or a room with a high ventilation would naturally reduce the availability of fumes to be inhaled by an individual. In a similar manner, the circumstances of Mr. Laramore's diesel and gas exposure is ill-defined. While he did haul diesel and gas, he reports standing away from the location where the fumes would be concentrated. And Mr. Laramore hauled all types of freight in his career as a truck driver. Unlike in Camp Lejeune, where Mr. Laramore would have had exposure to toxic chemicals by all routes of exposure (ingestion,

[69] Liang et al, Pesticide exposure and risk of bladder cancer: A meta-analysis, Oncotarget. 2016 Oct 11;7(41)

[70] While the exact solvent(s) that Mr. Laramore used are not known, the time period involved and the similarity of uses as at Camp Lejeune leads me to suspect it was TCE.

inhalation, and dermal), his occupational exposures appear to have only be via inhalation.

While there are significant gaps in understanding the nature of Mr. Laramore's occupational exposure, I do consider it to be potential cause in his bladder cancer diagnosis.

3. Age and Gender

Bladder cancer is predominantly a disease of older adults with the average of diagnosis in the US being 73. This is older than the average age of overall cancer development of 65-70 years of age.[71] While the risk of developing bladder cancer increases with age, it is more related to accumulated exposure to carcinogens over time than the age itself. Therefore, age is more of a proxy of cumulative risk from other factors, rather than a standalone cause. These findings highlight the fact that development of this disease requires decades post-exposure to carcinogens until development.[72] Age is not considered a direct risk factor for bladder cancer because it is primarily associated with environmental factors, such as smoking and exposure to certain chemicals like those found in the workplace). While the risk of developing bladder cancer increases with age, it is more related to accumulated exposure to carcinogens over time rather than age itself. Therefore, age acts as a proxy for cumulative risk from other factors, rather than a standalone cause[62].

Additionally, bladder cancer is more likely to be diagnosed in men than women with a three to one ratio, respectively. Much of the discrepancy can be attributed to the different rates of tobacco use as we see the prevalence of bladder cancer in women much higher in countries with higher rates of tobacco use in females.[73] Other reasons can include the occupational chemical exposures for men working in various factory settings.

I consider both of these risk factors to be fairly weak and I do not consider this a risk factor for Mr. Laramore's case given his exposure over time to both tobacco and environmental factors from Camp Lejeune specifically TCE.

4. Family History and Genetic Predisposition

A family history of bladder cancer or other cancers of the urinary tract can increase an individual's risk. Genetic mutations in genes such as TP53, which plays a critical role in tumor suppression, have been identified in some individuals with bladder cancer. Although the genetic contribution to bladder cancer risk is not as prominent as other factors, individuals with a family history may be at an increased risk of developing the disease. Lynch syndrome, a hereditary condition caused by mutations in mismatch

[71] Siegel et al, Cancer statistics, 2019. *CA Cancer J. Clin.* 2019, *69*, 7–34.
[72] Saginala et al, Epidemiology of Bladder Cancer *Med. Sci.* 2020, *8*(1)
[73] Bray et al, Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J. Clin.* 2018, *68*, 394–424)

repair genes, significantly increases the risk of several cancers, including bladder cancer. Individuals with Lynch syndrome are approximately three to four times more likely to develop bladder cancer compared to the general population.[74]

I do not consider family history or genetic predisposition to be a risk factor for Mr. Laramore. Mr. Laramore has not been diagnosed with Lynch syndrome and has no family history of bladder cancer. Although Mr. Laramore has family history of other types of cancer, these other cancers do not put Mr. Laramore at greater risk of getting bladder cancer.

5. Chronic Bladder Infections and Other Medical Conditions

Chronic bladder infections and certain medical conditions can significantly increase the risk of developing bladder cancer, particularly through prolonged inflammation and cellular damage in the bladder lining. Recurrent urinary tract infections (UTIs) or chronic cystitis can lead to long-term irritation and inflammation of the bladder, creating an environment conducive to cancerous cell changes. Additionally, conditions like bladder stones and the use of medical devices such as urinary catheters can further irritate the bladder and contribute to cancer risk by causing repeated trauma to the bladder lining.[75] Bladder cancer associated with chronic inflammation is more associated with squamous cell carcinoma rather than urothelial carcinoma. Another important risk factor is the use of certain medications, most notably the chemotherapy drug cyclophosphamide, which has been linked to an increased incidence of bladder cancer due to its carcinogenic effects on the bladder cells. These conditions, particularly when combined with other risk factors like smoking or chemical exposure, can further heighten an individual's likelihood of developing bladder cancer.

I do not consider chronic bladder infections or other medical conditions to be a risk factor for Mr. Laramore. Prior to his diagnosis, he did not have a chronic bladder infection, was not prescribed any chemotherapy drug, and did not have any medical condition that put him at greater risk for getting bladder cancer.

VIII. Opinions

a. Opinion 1: exposure to the water at Camp Lejeune is at least as likely as not a cause of Mr. Laramore’s bladder cancer.

Upon understanding the known risk factors for bladder cancer development and review of Mr. Laramore’s medical records, it is my opinion that Mr. Laramore’s exposure to the water at Camp Lejeune is at least as likely as not to have caused his bladder cancer.

[74] Phelan 2018
[75] Yacouba et al, Urinary microbiota and bladder cancer: A systematic review and a focus on uropathogens Seminars in Cancer Biology Volume 86, Part 3, November 2022, Pages 875-884

As stated above, Mr. Laramore was exposed to known bladder cancer-causing chemicals at Camp Lejeune. These chemicals are genotoxic, have epidemiological support for causing bladder cancer, and possess a sufficiently clear mechanism of action to lead to bladder cancer. In addition, as described in Dr. Gilbert's report, these chemicals are immunotoxic and lead to the development of bladder cancer through suppressing the body's adaptive immune response, stimulating pro-imflmatory cytokines, and alter the epigenetics of peripheral lymphocytes. Mr. Laramore had sufficient opportunity to be exposed to the chemicals at Camp Lejeune, as Mr. Laramore was on base for a total of approximately 388 days where he worked, lived, bathed, and drank water from the Hadnot Point Water Treatment Plant and thus exposed from all three potential routes of exposure (ingestion, inhalation, dermal). During Mr. Laramore's time of exposure, 1983-1984, the Hadnot Point Water Treatment Plant experienced some of its highest concentrations of chemical contamination. Mr. Laramore's exposure was substantial and continuous; there was no escape from contaminated water for Mr. Laramore. As set out in the report of Dr. Reynolds and the specific causation reports of Dr. Hatten and Dr. Bird, Mr. Laramore was substantially exposed to the chemicals at Camp Lejeune at levels recognized in the scientific literature to lead to bladder cancer.

The timing of when Mr. Laramore was diagnosed with bladder cancer is noteworthy. Mr. Laramore was diagnosed with bladder cancer at the age of 61, although he had gross hematuria two years prior to his diagnosis which likely signals in hindsight that an earlier diagnosis date is possible. The median age of diagnosis with bladder cancer is 73, meaning that Mr. Laramore's diagnosis was 12 years earlier than median. This indicates that the nature of Mr. Laramore's exposure – which significantly includes the water at Camp Lejeune – was such that it caused a more rapid diagnosis. While it is impossible to perfectly connect age of diagnosis to a specific source, Mr. Laramore's unique exposure (in the sense that it is not shared by a wider population) at Camp Lejeune strongly suggests a correlation.

In addition to Mr. Laramore's age, the latency period between Mr. Laramore's exposure at Camp Lejeune and his eventual diagnosis – approximately 35 years – is in line with what I would expect from a toxic exposure. As discussed above, bladder cancer can have a longer latency extending to 40 years and beyond. In my opinion his invasive cancer at an earlier age is due to the cumulative exposure of two very highly carcinogenic toxins, TCE and PCE which he was exposed to at a young age in which the bladder in more vulnerable for damage along with his chronic tobacco exposure.

To a reasonable degree of medical certainty, Mr. Laramore's exposure to the water at Camp Lejeune was substantial.

b. Opinion 2: other risk factors for bladder cancer are not more likely than not to have caused Mr. Laramore's bladder cancer

Besides exposure to the water at Camp Lejeune, there are two other potential bladder cancer risk factors for Mr. Laramore: smoking and occupational exposure. As a

preliminary matter, it would be incorrect to think that risk factors, as a matter of principle, act as independent from one another and/or in competition with one another. That means that the presence of other risk factors does not negate other potential causes; instead, each risk factor must be evaluated independently and assessed for its potential contribution to causing bladder cancer. If a potential risk factor cannot be meaningfully unbundled and differentiated from other risk factors, then there is lower confidence that one risk factor can predominate over another. In a similar way, a risk factor that relies on speculative inferences cannot be considered on the same level as risk factors that are on stronger footing.

There is no dispute that smoking is a recognized risk factor for the development of bladder cancer, but Mr. Laramore's smoking history cannot be unbundled from his exposure to the contaminated water at Camp Lejeune. Both smoking and exposure to chemicals in the water at Camp Lejeune have well-documented and strong associations with bladder cancer. While smoking increases the risk of developing bladder cancer by 50%, that does not mean that 50% of smokers end up developing bladder cancer. As discussed earlier, the vast majority of smokers do not develop bladder cancer.

Further complicating a neat dividing line between smoking and other risk factors, a number of the epidemiological articles that discuss the chemicals at Camp Lejeune also controlled for smoking. Vlaanderen et al (2014) was a meta-analysis that considered the effect of PCE-exposed workers and bladder cancer that mostly included studies that controlled for smoking. Zhao et al (2005) "observed only weak associations between smoking status…and exposure to TCE…" and ultimately concluded that "the estimated exposure effects reported in this paper were not appreciably confounded by smoking." Hadkhale explained the study's process for addressing the recognized connection between bladder cancer and smoking: "If the risk of lung cancer in a given occupation is elevated, and there are no other work-related exposures than smoking, then the risk of bladder cancer should also be elevated due to smoking…The RRs for bladder cancer clearly differ from this pattern…Though smoking is a well-established risk factor for bladder cancer, occupational differences in bladder cancer risk do not appear to be solely due to smoking." Likewise, Sciannameo (2019) adjusted for smoking as well. Finally, ATSDR controlled for smoking in its studies as well. What this means is that there is a clear trend showing that the development of bladder cancer cannot be explained away by smoking alone, and that exposures to the chemicals at Camp Lejeune provide an additional, significant risk factor.

Tobacco smoke and the chemicals at Camp Lejeune share a similar endpoint in that all are genotoxic. As explained by Dr. Plunkett, the chemicals at Camp Lejeune are metabolized into highly reactive metabolites that are genotoxic in cells and tissues,[76] meaning that they are capable of changing or damaging DNA. Having these reactive, toxic metabolites sit in the bladder for long periods of time provides the right environment for them to interact with the urothelial cells that line the bladder and lead to bladder cancer.

[76] Plunkett ¶ 85

As noted by Dr. Gilbert and Dr. Plunkett, the mixture of the chemicals at Camp Lejeune cannot be ignored as they have compounding effects. Many different carcinogens have been identified, which can lead to cancer development. Carcinogens can cause cancer by inducing genetic mutations, which disrupt normal cell processes such as growth and division. These mutations often impair the body's ability to repair damaged DNA or regulate cell death, leading to uncontrolled cell proliferation and, ultimately, tumor formation. Carcinogens can act through various mechanisms, such as directly damaging DNA, triggering inflammation, or disrupting cellular signaling pathways.[77] When an individual is exposed to more than one carcinogen, the risk of cancer development is often amplified due to the additive or synergistic effects of the exposures. Each carcinogen may induce different types of genetic damage or interfere with distinct cellular processes, increasing the overall likelihood of further mutations and promoting more aggressive tumor growth. Studies have shown that combined exposures to multiple carcinogens, such as tobacco smoke and certain environmental pollutants, significantly raise the incidence of cancers like lung and bladder cancer.[78] This cumulative effect highlights the importance of limiting exposure to multiple carcinogenic agents to reduce cancer risk.[79]

The understanding of how carcinogens lead to cancer development and that different carcinogens can synergistically lead to cellular damage causing initiation of tumor development underscores the importance that we cannot differentiate which carcinogen leads to the initial hit.[80] More importantly any exposure to any carcinogen is damaging and that each individual carcinogen exposure can lead to cancer development, but together more damage can occur with increasing risk of cancer development. That is especially true when an individual is exposed to numerous carcinogens known to lead to bladder cancer over an extended period of time.

Mr. Laramore had two documented occupational exposures – as a civil servant using cleaning solvents, and as a truck driver. These exposures bookend Mr. Laramore's exposure to the chemicals in the water at Camp Lejeune, with his solvent exposure coming first and his exposure to diesel fumes as a truck driver coming later. Mr. Laramore's solvent exposure is particularly challenging to quantify, as several factors that impact any inhalation of the solvent (which would be the primary method of exposure, given that he wore gloves during his job). It would be highly speculative to estimate his quantity, duration, and intensity of exposure.

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[77] Barnes et al, Carcinogens and DNA damage Biochem Soc Trans. 2018 Oct 4;46(5):1213–1224).

[78] Cani et al, How Does Environmental and Occupational Exposure Contribute to Carcinogenesis in Genitourinary and Lung Cancers? Cancers (Basel) 2023 May 19;15(10):2836.)

[79] The Cell: A Molecular Approach. 2nd edition.Cooper GM. Sunderland (MA): Sinauer Associates; 2000.

[80] Halmes et al, Reevaluating Cancer Risk Estimates for Short-Term Exposure Scenarios Toxicological Sciences, Volume 58, Issue 1, November 2000, Pages 32–42)

IARC classified diesel exhaust as probably carcinogenic to humans, primarily for lung cancer but noted a positive association with bladder cancer.[81] Nevertheless, Mr. Laramore's exposure to diesel fumes as a truck driver share many of these challenges as well, a fact that was recognized by IARC in its epidemiological review: "[t]he major limitation of the studies reviewed was the small number with well characterized exposure to diesel or gasoline exhaust."[82] As IARC noted, there are individual factors that impact a particular exposure, including whether the driver was a long haul driver or local (local drivers had higher exposures), when driver performed his job (IARC described a "two- to threefold decline in the levels of exposure to [elemental carbons] between the 1980s and 2001-05."), and the age of the vehicle (older vehicles have higher seepage).[83] In addition, differences in fuel (even amongst diesel fuel) can lead higher or lower exhaust emissions.[84] IARC ultimately concluded that diesel exhaust components are genotoxic and cause DNA mutations, can produce reactive oxygen species that promote oxidative stress, and can cause inflammation.[85] As with Mr. Laramore's solvent exposure, any exposure to diesel fumes would have occurred via inhalation.

The question, then, is whether either of Mr. Laramore's other exposures – smoking, other solvent exposure, and diesel fumes – are more likely to have caused Mr. Laramore's bladder cancer than his exposure to the chemicals at Camp Lejeune. Each of the chemicals associated with these exposures are documented to cause DNA damage. As Dr. Gilbert explains in her report, TCE in particular can negatively affect the immune system's innate ability to surveil for mutated cells and destroy them, thus permitting cancerous cells to grow. Simply put, Mr. Laramore was exposed to a number of chemicals that have known associations with bladder cancer but none of which are more likely to have caused his diagnosis than the others. The longer duration of Mr. Laramore's occupational exposure has to be counterweighed by the single route of exposure and discounted by the numerous factors (the answer to which we cannot completely know) that affect his exposure. Mr. Laramore's smoking history, while significant, is not an overwhelming factor given how epidemiological studies control for smoking and still see increase in bladder cancer diagnoses. Given the amount of exposures Mr. Laramore experienced and their known associations with bladder cancer, it is impossible to discern which "hit" was first, more meaningful than the others, or the deciding factor in causing his bladder cancer. Thus, because I cannot find any risk factor that is more likely than not to have caused his bladder cancer, I conclude that each of his risk factors are at least as likely as not to be the cause, including his exposure to the chemicals at Camp Lejeune.

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[81] International Agency for Research on Cancer. Diesel and Gasoline Engine Exhausts and Some Nitroarenes. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;105
[82] IARC 2014, p. 199
[83] IARC 2014, p 81
[84] Clark et al., Factors affecting heavy-duty diesel vehicle emissions, J Air & Waste Manage. Assoc. 52:84-94 2002.
[85] IARC 2014 p. 422

Appendix 1

CURRICULUM VITAE

John P. Sfakianos, MD

Email:john.sfakianos@mountsinai.org
Tel (Office): (212) 241-4812
[redacted]
Mailing Address (Office): 1425 Madison Avenue, 6th Floor, Room L6-58, New York, NY 10029
Mailing Address (Home): [redacted]

APPOINTMENTS/EMPLOYMENT

Icahn School of Medicine at Mount Sinai, New York, NY	Professor Urology (10/2024-present)
Icahn School of Medicine at Mount Sinai, New York, NY.	Associate Professor Urology (04/2022–10/2024)
Department of Urology Mount Sinai, New York NY	Medical Director (07/2015-06/2018)
Icahn School of Medicine at Mount Sinai, New York, NY	Assistant Professor Urology (07/2014–03/2022)
Memorial Sloan Kettering Cancer Center, New York, NY	Clinical Instructor Surgery/Urology (07/2012-06/2014)

Gaps in Employment
Not applicable

EDUCATION

Leadership Emerging in Academic Departments (LEAD)	Student (2023-present)
Memorial Sloan Kettering Cancer Center (Urologic Oncology)	Fellowship (06/2014)
SUNY Downstate Medical Center (Urology)	Residency (06/2012)
SUNY Downstate Medical Center (Surgery)	Internship (6/2008)
SUNY Buffalo School of Medicine and Biosciences	Doctoral of Medicine (09/2007)
Hunter College CUNY (Biochemistry)	Bachelors of Science (06/2002)

Certification

ISMSS Mindfulness Course	(2023)
American Board of Urology	(2016)

Licensure
New York Medical: 265049
DEA: FS3492674
NPI: 1295904084

HONORS/AWARDS

Bladder Cancer Advocacy Network (BCAN) 2023 Translational Clinical Trial Award	(2022)
Bladder Cancer Advocacy Network (BCAN) 2022 Innovation Award	(2022)
Tisch Cancer Institute Development Funds Award	(2022)
Faculty Council Award Junior Faculty, Icahn School of Medicine	(2021)
Faculty inductee Alpha Omega Alpha	(2020)
Teacher of the Year, Icahn School of Medicine Department of Urology	(2018, 2019)
Sharing and Caring Physician Recognition Award	(2017)

Patents
N/A

Other entrepreneurial opportunities

N/A

OTHER PROFESSIONAL ROLES

Journal Editor
Bladder Cancer- Editorial Board 1/2020-present Urologic Oncology-Consulting editor 1/2020-present
Journal Assignments
Reviewer, European Urology
Reviewer, European Urology Oncology
Reviewer, Journal of Urology
Reviewer, Nature Communication
Reviewer, Seminars in Urologic Oncology

Research Profile

My translational research focuses on human Natural Killer (NK) cells and CD8 T cells and their functional roles in bladder cancer. I collaborate extensively with Drs. Amir Horowitz, Nina Bhardwaj, and Matthew Galsky and the human immune monitoring center (HIMC) to profile human NK cells, T cells and innate lymphoid cells in human bladder cancer. We have established an effective pipeline for profiling NK cells and ILCs as well as for studying their crosstalk with other immune cells (myeloid cells, T cells, neutrophils, and B cells), stroma, and tumors. The Main focus of my research is focused on BCG unresponsive non-muscle invasive bladder cancer. The focus is to understand the underlying immune resistance mechanism and to identify novel therapeutic options. To achieve this goal, we use an array of cutting-edge technologies, including mass cytometry (CyTOF), imaging mass cytometry (IMC), Olink proteomics, and single-cell and Spatial RNA sequencing along with genomic data on HLA class I genes to profile NK and CD8 T cells with ultra-resolution. Our work has identified a novel therapeutic target in the NKG2A:HLA-E pathway.

Clinical Profile

My clinical focus has been on identifying novel methods in helping patients with bladder cancer. My translational research has focused on non-muscle invasive bladder cancer in which our work has led to the funding of a novel Phase 2 clinical trial for BCG unresponsive tumors. Furthermore, my clinical research focus has been centered on patients undergoing radical cystectomy. Our group has been focused on improving the Enhanced Recovery After Surgery (ERAS) pathway for patients undergoing radical cystectomy. Our clinical work has improved outcomes on many aspects of surgery. We have published several patients on improving infection rates by using an antibiogram specific antibiotic regimen and intracorporeal robotic surgery, safety of using oral anticoagulation for VTE prophylaxis and safely performing surgery without drains. Most importantly using regional blocks and intracorporeal robotic surgery we can safely perform surgery without using opioids (Non-opioid protocol (NOP)).

MENTORING PROFILE

I have mentored multiple medical students, residents, and a postdoctoral fellow. Additionally, through collaborations with Drs. Amir Horowitz (Immunology, ISMMS) we have emphasized a co-mentorship dual-training approach focused on both clinical and basic science/translational research. One notable example to highlight focused on three medical students: Drs. Daniel Ranti (ISMMS), Y. Alice Wang

(ISMMS), and Christine Bieber (St. Georges University) whose time with me ranged from one to 2.5 years. These three trainees partnered together to develop an understanding of resistance to M. bovis BCG treatment of non-muscle-invasive bladder cancer (NMIBC), which has resulted in federal NIH/NCI R21 and R01 awards and a funded Phase 2 clinical trial in BCGunresponsive NMIBC. We have submitted a co-first authorship manuscript for peer review, and they have each co-authored numerous clinical and translational research and review articles. All three of them have now matched for top Urology residency programs (Columbia, Johns Hopkins, SUNY Albany). Finally, Daniel Ranti was awarded Mount Sinai's Dr. Harold Lamport Biomedical Research Prize as well as the PORTAL thesis award in clinical research upon his graduation

Diversity and Inclusion Impact

Working with medical students I have been volunteering my time as a Urology faculty member covering the East Harlem Health Outreach Partnership (EHHOP) Urology clinic. The focus of the clinic is to help a diverse underserved patient population at the same time work with medical students to help them understand the field of Urology and help those interested in developing into Urologist.

Overall Impact

Over the last 30 years BCG has been and still is the only FDA approved treatment for non-muscle invasive bladder cancer. There has been little understanding of its mechanism of action in cancer therapy and furthermore little understanding in the biology of resistance to this therapy. My work over the last 10 years has been vital in understanding the immune microenvironment in BCG unresponsive bladder cancer. We have identified a critical role of NK cells and their interactions with other immune cells in the tumor microenvironment as pivotal for resistance. With this knowledge I have been successful in securing numerous funding sources for continued work in this area including a DOD team translational award (co-PI), an R01 (Co-PI) and R21 (PI) and several private grants including the BCAN innovation award (PI). Most importantly our findings have led to an investigator initiated clinical trial targeting NK and T cells in BCG unresponsive bladder cancer. This clinical trial has been funded by BCAN and will initiate enrollment of patient in 2024.

Clinically I have successfully modified an Enhanced Recovery after Surgery (ERAS) protocol for patients undergoing radical cystectomy to allow for the elimination of narcotics after surgery and decreased the risk of infections. I have implemented several protocols that are published in peer reviewed journals using nerve blocks prior to surgery along with anti-inflammatory medications leading to ~90% of patients post radical cystectomy not needing opioids. Furthermore, we developed a Mount Sinai Hospital specific antibiogram for patients undergoing radical cystectomy and modified our antibiotic prophylaxis regimen leading to the reduction of post-operative infections by almost 50%. The changes have allowed patients to be discharged sooner with lower readmissions. The successful implementation of this modified ERAS program for patients undergoing radical cystectomy has been well accepted by collogues nationally and internationally leading to invitations for presentations at several conferences and guideline implementation.

Grants, Contracts and Foundation Support

Past Grants

List Funding Source, Project Title and Number	Role in Project	Dates	Direct Costs	Supplemental info
DOD (PD/PI: Sfakianos.)	**PI;** to characterize the dysfunctional NK cell	7/15/2019 - 7/14/2023	$1,584,753	N/A

Project #: W81XWH1910269 **Project title**: Dissection of suppressive axes underlying Natural Killer cell dysfunction in human bladder cancer	phenotype in the TME and blood of individuals with bladder cancer, identify mechanisms underlying NK cell dysfunction in nonmuscle-invasive and muscle-invasive tumors and to design rational, preclinical interventions to enhance NK function and reverse NK dysfunction. **% effort:** 1.2 CM			
NINR/NIH (PD/PI: Mohamed.) **Project #:** 5R21NR016518 **Project title:** Novel Approach to Enhance Ostomy Care in Patients with Bladder and Colorectal Cancer	**Co-PI;** Study results will guide further refinement of the ostomy-education program for a larger experimental trial. **% effort:** 0.36 CM	05/01/2017 – 04/30/2019	$126,777	N/A
NINR/NIH (PD/PI: Mohamed.) **Project #:** 5 R21 **Project title**: The personal patient profile decision support for patients with bladder cancer	**Co-I;** The personal patient profile decision support for patients with bladder cancer **% effort:** 0.24 CM	07/01/2020-06/30/2022	$225,000	N/A

Current Grants

List Funding Source, Project Title and Number	Role in Project	Dates	Direct Costs	Supplemental info
DISRUPT (SU2C) (PD/PI: Horowitz.) **Project #:** N/A **Project Title:** Understanding the immunogenetic drivers of health disparities in Afro-Caribbean men with prostate cancer	**Co-I;** to define a cohort of Black Afro-Caribbean and EuropeanCaucasian prostate cancer patients receiving care across distinct areas of New York City and perform WES and RNA-seq on germline and tumor tissue to generate HLA-I genotypes and quantify abundance of RNAtranscripts associated with NKcell phenotypes and antitumor functions. **% effort: 0.1 CM**	02/1/2023 - 01/31/2027	$50,000	N/A
NIH/NCI (PD/PI: Horowitz, Sfakianos.) **Project #:** 1 R01 CA269954-01 **Project title:** HLA-E and NKG2A define a novel	**MPI;** to (1) analyze intratumoral NK cells and T cells and their interactions with tumors in response to BCG therapy, (2) longitudinally assess blood and tumor phenotypes in response	02/1/2023 - 01/31/2027	$3,359,953	N/A

immune checkpoint axis in non-muscle-invasive bladder cancer.	to BCG therapy, and (3) determine the effects of combination PD-L1 and NKG2A blockade on anti-tumor immunity **% effort:** 4.2 CM			
NIH/NCI (PD/PI: Sfakianos.) **Project #:** 1 R21 CA274148 **Project title:** Identifying novel resistance mechanisms in non-muscle-invasive bladder cancer treated with Bacillus Calmette-Guerin (BCG)	**PI;** using two distinct cohorts of tumors, both BCG alone and BCG and PD1-combination resistant tumors, to investigate the spatial and immunologic heterogeneity underlying resistance to these standard of care therapies. **% effort:** 0.852 CM	8/1/2022 - 7/31/2024	$150,000	N/A
NIH/NCI (PD/PI: Bhardwaj, N.) **Project #:** CA249175 **Project title**: Dissecting Myeloid-Cell Mediated Resistance to Immune Checkpoint Blockade in Bladder Cancer	**Co-I;** to (1) dissect gene modules associated with response resistance to CPI using high resolution maps of the cellular and molecular landscape of muscle-invasive bladder cancer (MIBC), (2) determine the role of stromal module genes in controlling the immunophenotyped and function of monocyte-macrophages, and (3) refine and validate a monocyte-macrophage-related gene signature as a biomarker of CPI resistance in clinical trial cohorts. **% effort:** 1.0 CM	4/20/2020 - 3/31/2025	$3,502,929	N/A
Bladder Cancer Advocacy Network Clinical Trials Award (PD/PI: Horowitz, Sfakianos, Galsky.) **Project #:** N/A **Project title**: Targeting HLA-E/NKG2A for overcoming BCG resistance in non-muscle-invasive bladder cancer	**MPI**; we will conduct a Phase 2 clinical trial of combination PD-L1/NKG2A blockade (durvalumab/monalizumab) in patients with BCG-unresponsive NMIBC. We will determine whether measuring the proteins HLA-E and NKG2A in pretreatment tumors might be used in the future to identify patients who are most likely to respond to this novel treatment regimen. **% effort:** 1.2 CM	5/1/2023 - 4/30/2026	$2,237,527.89	N/A

NIH/NCI (PD/PI: Mullholand.) **Project #:** R21 CA286309 **Project title:** Targeting the Androgen Receptor to Sensitize Bladder Cancer to Immune Check Point Blockade	**Co-I;** The goal of this project it so better understand the role of the Androgen Receptor in bladder cancer and to assess if inhibition of immune point blockade can be enhanced with co-blockade. **%effor**t: 0.36 CM	1/1/2024-13/31/2025	$434,542	N/A
Bladder Cancer Advocacy Network Clinical Trials Award (PD/PI: Sfakianos) **Project #:** N/A **Project title:** High Resolution Molecular Imaging to Elucidate the Contextual Mechanisms of BCG Resistance in Non-muscle Invasive Recurrent Bladder Cancer	**PI**; This project seeks to understand novel immune mechanisms of NMIBC through the careful dissection of tumor, stromal and immune cells and secondarily from the combinatorial interactions between them. **% effort:** 0.9 CM	08/1/2022-07/31/2024	$300,000	N/A
DOD (PD/PI: Mullholand.) **Project #:** HT94252310186 **Project title:** Determining the contribution of lineage intermediate tumor cells during progression to t-SCNC	**Co-I;** The goal of this project is to understand the impact of AR signaling and targeting agents to preclude the progression of prostate cancer lineage intermediate tumor cells to small cell neuroendocrine cells. **% effort: 1.2 CM**	08/1/2023-07/31/2026	$1,521,002	N/A

Pending Grants

List Funding Source, Project Title and Number	**Role in Project**	**Dates**	**Direct Costs**	**Supplemental info**

Clinical Trial Participation

Project	**Role in Project**	**Dates**	**Award**	**Other Info**
Urogen Pharma A Phase 3, Single-Arm, Multicenter Study to Evaluate the Efficacy and Safety of UGN-102 as Primary Chemoablative Therapy in Patients with Low Grade (LG) Non-Muscle Invasive Bladder	PI	12/18/2022-12/17/2024	$101, 370	Phase III

Cancer (NMIBC) at Intermediate Risk (IR) of Recurrence				
Jiangsu Yalong Meditech Co A Phase I/II Open-Label Multicenter Study to Evaluate the Safety and Efficacy of Oral APL-1202 in Combination with Tislelizumab Compared to Tislelizumab Alone as Neoadjuvant Therapy in Patients with Muscle Invasive Bladder Cancer	PI	5/15/2022-08/10/2024	$67, 314	Phase II/III
Irrimax Corporation Chlorhexidine antiseptic irrigation of the bowel segment during radical cystectomy and urinary diversion	PI	05/1/2023-04/30/2025	$99, 525	Investigator initiated trial
Janssen Research & Development A Phase 2, Open-Label, Multi-Center, Randomized Study of TAR-200 in Combination with Cetrelimab and Cetrelimab Alone in Participants with Muscle-Invasive Urothelial Carcinoma of the Bladder who are Scheduled for Radical Cystectomy and are Ineligible for or Refusing Platinum-based Neoadjuvant Chemotherapy	PI	07/15/2023-07/14/2025	$80, 353	Phase II

Trainees

Name	**Level of Trainee**	**Role & dates of training**	**Training Venue**	**Trainee's current status/employment**
Iyinyeoluwa Okulate	Research Associate	Mentor; 1/2023 - Present	Icahn School of Medicine at Mount Sinai	**Research Associate**, Icahn School of Medicine at Mount Sinai
Jordan Rich	Research Associate	Mentor; 7/2022-08/2023	Icahn School of Medicine at Mount Sinai	**Urology resident**, New York University
Daniel Ranti	Medical student	Mentor; 5/2020 – 1/2023	Icahn School of Medicine at Mount Sinai	**Urology resident**, Columbia University School of Medicine
Y. Alice Wang, PhD	Medical student	Mentor; 5/2020 – 6/2022	Icahn School of Medicine at Mount Sinai	**Urology resident**, Johns Hopkins University School of Medicine

Christine Bieber	Medical student	Mentor; 5/2021 – 6/2022	Icahn School of Medicine at Mount Sinai	**Urology resident,** SUNY Albany School of Medicine
Bérengère Salomé, PhD	Postdoctoral fellow	Mentor; 1/2018 – 8/2022	Icahn School of Medicine at Mount Sinai	**Principal Research Scientist,** Genentech (gRED), Cancer Immunology
Andrew Charap	Medical student	Mentor; 1/2017 – 6/2019	Icahn School of Medicine at Mount Sinai	**ENT resident**, Washington University School of Medicine
Jorge Daza, MD	Medical doctor (Colombia)	Mentor; 6/2017 – 6/2020	Icahn School of Medicine at Mount Sinai	**Urology faculty**, private practice Buffalo NY
Joy Park	Research Associate	Mentor; 6/2017 – 12/2018	Icahn School of Medicine at Mount Sinai	**Graduate student,** SUNY Upstate
Harry Anastos MD	Research Associate	Mentor; 9/2016 – 6/2018	Icahn School of Medicine at Mount Sinai	**Urology Fellow, endourology** Columbia University School of Medicine
Andrew Katims MD	Urology Resident	Mentor; 9/2016- 6/2021	Icahn School of Medicine at Mount Sinai	**Urology Oncology Fellow,** Memorial Sloan Kettering Cancer Center
Zynep Gul MD	Urology Resident	Mentor, 7/2014- 6/2018	Icahn School of Medicine at Mount Sinai	**Assistant Professor,** University of Washington at St. Louis
Kyrollis Atalla MD	Urology Resident	Mentor, 7/2014- 6/2018	Icahn School of Medicine at Mount Sinai	**Assistant Professor**, Icahn School of Medicine at Mount Sinai

Teaching Activities

Teaching Activity/Topic	**Level**	**Role**	**Level of Learners**	**Number of hours week/month/yr**	**Evaluation Summary**	**Years Taught**
2nd Year Urology course	Medical School Course	Lecturer	Medical Students (~100)	2 hours per year		2018-present
Systems Biomedicine course	Masters students	Lecturer	Masters Students (~20)	2 hours per year		2016-present
Urology Residency Education Rounds	Urology Residents	Organizer/Proctor	Urology Residents (24)	10 hour per week		2015-present

ADMINISTRATIVE LEADERSHIP APPOINTMENTS

Intramural Service

East Harlem Health Outreach Partnership (EHHOP) (faculty), Mount Sinah Hospital (01/2024-present)
Clinical Competency Committee (Chair), Department of Urology ISMMS (7/2022-present)

Investigator Initiated Clinical Trials Review Committee (Co-Chair), Tisch Cancer Institute ISMMS (05/2021 – present)
Clinical Research Steering Committee (Member), Tisch Cancer Institute ISMMS (1/2018 – 2024)
Protocol Review and Monitoring Committee (PRMC) (Voting Member), Tisch Cancer Institute (0/12018-12/2022)
Tumor Board, Department of Urology, ISMSS (Coordinator) (07/2014-present)
Disease Focus Group, Urology, ISMSS (07/2014-present)

Extramural Service
NCI/NIH Reviewer, Therapeutic Immune Regulation (TIR) study section (2023-Present)
Urology Care Foundation research grand review panel (2023)
NIH/CSR Reviewer, Kidney, Urology and Digestive Disease study section (2022)
CUASF Bladder Cancer Canada study section (2020 – Present)
Swiss National Science Foundation grant reviewer (2022-Present)

PUBLICATIONS

Peer Reviewed Original Contributions (H-Index = 34, Date last checked: 06/24/2024)

1. Rich JM, Geduldig J, Cumarasamy S, Ranti D, Mehrazin R, Wiklund P, Sfakianos JP, Attalla K. Eliminating the routine use of postoperative drain placement in patients undergoing robotic-assisted radical cystectomy with intracorporeal urinary diversion. Urol Oncol. 2023 Oct 19.

2. Rich JM, Garden EB, Arroyave JS, Elkun Y, Ranti D, Pfail JL, Klahr R, Omidele OO, Adams-Sommer V, Patel G, Schaefer SH, Brown C, Badani K, Lavallee E, Mehrazin R, Attalla K, Waingankar N, Wiklund P, Sfakianos JP. Infections After Adoption of Antibiogram-directed Prophylaxis and Intracorporeal Urinary Diversion for Robotassisted Radical Cystectomy. Eur Urol Focus. 2023 Oct 12.

3. Galsky MD, Daneshmand S, Izadmehr S, Gonzalez-Kozlova E, Chan KG, Lewis S, Achkar BE, Dorff TB, Cetnar JP, Neil BO, D'Souza A, Mamtani R, Kyriakopoulos C, Jun T, Gogerly-Moragoda M, Brody R, Xie H, Nie K, Kelly G, Horwitz A, Kinoshita Y, Ellis E, Nose Y, Ioannou G, Cabal R, Haines GK, Wang L, Mouw KW, Samstein RM, Mehrazin R, Bhardwaj N, Yu M, Zhao Q, Kim-Schulze S, Sebra R, Zhu J, Gnjatic S, Sfakianos J, Pal SK. Gemcitabine and cisplatin plus nivolumab as organ-sparing treatment for muscle-invasive bladder cancer: a phase 2 trial. Nat Med. 2023 Oct 2.

4. Yuk FJ, Carr MT, Schupper AJ, Lin J, Tadros R, Wiklund P, Sfakianos J, Steinberger J. Da Vinci Meets Globus Excelsius GPS: A Totally Robotic Minimally Invasive Anterior and Posterior Lumbar Fusion. World Neurosurg. 2023 Sep 13;180:29-35.

5. Rich J, Tillu N, Grauer R, Busby D, Auer R, Breda A, Buse S, D'Hondt F, Falagario UG, Hosseini A, Mehrazin R, Minervini A, Mottrie A, Sfakianos J, Palou Redorta J, Wijburg C, Wiklund NP, John H. Robotic-Assisted Repair of Ureteroenteric Strictures after Cystectomy with Urinary Diversion: Technique Description and Outcomes from the ERUS Scientific Working Group. J Endourol. 2023 Sep 11.

6. Wong JL, Smith P, Angulo-Lozano J, Ranti D, Bochner BH, Sfakianos JP, Horowitz A, Ravetch JV, Knorr DA. IL-15 synergizes with CD40 agonist antibodies to induce durable immunity against bladder cancer. Proc Natl Acad Sci U S A.2023 Aug 29;120(35).

7. Rich JM, Elkun Y, Geduldig J, Lavallee E, Mehrazin R, Attalla K, Wiklund P, Sfakianos JP. Outcomes from a prospectively implemented protocol using apixaban after robot-assisted radical cystectomy. BJU Int. 2023 Oct;132(4).

8. Daza J, Grauer R, Chen S, Lavallèe E, Razdan S, Dey L, Steineck G, Renström-Koskela L, Li Q, Hussein AA, Mehrazin R, Waingankar N, Guru K, Wiklund P, Sfakianos JP. Development of a predictive model for recurrencefree survival in pTa low-grade bladder cancer. Urol Oncol. 2023 May;41(5).

9. Grossmann NC, Soria F, Juvet T, Potretzke AM, Djaladat H, Ghoreifi A, Kikuchi E, Mari A, Khene ZE, Fujita K, Raman JD, Breda A, Fontana M, Sfakianos JP, Pfail JL, Laukhtina E, Rajwa P, Pallauf M, Poyet C, Cacciamani GE, van Doeveren T, Boormans JL, Antonelli A, Jamil M, Abdollah F, Ploussard G, Heidenreich A, Storz E, Daneshmand S, Boorjian SA, Rouprêt M, Rink M, Shariat SF, Pradere B. Comparing Oncological and Perioperative Outcomes of Open versus Laparoscopic versus Robotic Radical Nephroureterectomy for the Treatment of Upper Tract Urothelial Carcinoma: A Multicenter, Multinational, Propensity Score-Matched Analysis. Cancers (Basel). 2023 Feb 23;15(5):1409.

10. Daza J, Salomé B, Okhawere K, Bane O, Meilika KN, Korn TG, Qi J, Xe H, Patel M, Brody R, Kim-Schulze S, Sfakianos JP, Lewis S, Rich JM, Zuluaga L, Badani KK, Horowitz A. Urine supernatant reveals a signature that predicts survival in clear-cell renal cell carcinoma. BJU Int. 2023 Jul;132(1):75-83.

11. Wong JL, Smith P, Angulo-Lozano J, Ranti D, Bochner BH, Sfakianos JP, Horowitz A, Ravetch JV, Knorr DA. IL-15 synergizes with CD40 agonist antibodies to induce durable immunity against bladder cancer. bioRxiv [Preprint]. 2023 Feb 1:2023.01.30.526266. doi: 10.1101/2023.01.30.526266. Update in: Proc Natl Acad Sci U S A. 2023 Aug 29;120(35).

12. Rosenzweig SJ, Ranti D, Mehrazin R, Sfakianos JP, Wiklund PN, Waingankar N. Characteristics Contributing to Survival Differences Between Black and White Patients Following Cystectomy. Urol Oncol. 2023 Apr;41(4).

13. Antonelli L, Sebro K, Lahmar A, Black PC, Ghodoussipour S, Hamilton-Reeves JM, Shah J, Bente Thoft J, Lerner SP, Llorente C, Lucca I, Preston MA, Psutka SP, Sfakianos JP, Vahr Lauridsen S, Williams SB, Catto J, Djaladat H, Kassouf W, Loftus K, Daneshmand S, Fankhauser CD; Enhanced Recovery After Surgery ERAS Cystectomy Committee. Association Between Antibiotic Prophylaxis Before Cystectomy or Stent Removal and Infection Complications: A Systematic Review. Eur Urol Focus. 2023 Jul;9(4):631-636.

14. Pallauf M, D'Andrea D, König F, Laukhtina E, Yanagisawa T, Rouprêt M, Daneshmand S, Djaladat H, Ghoreifi A, Soria F, Fujita K, Boorjian SA, Potretzke AM, Mari A, Roumiguié M, Antonelli A, Bianchi A, Khene ZE, Sfakianos JP, Jamil M, Boormans JL, Raman JD, Grossmann NC, Breda A, Heidenreich A, Del Giudice F, Singla N, Shariat SF, Pradere B. Diagnostic Accuracy of Clinical Lymph Node Staging for Upper Tract Urothelial Cancer Patients: A Multicenter, Retrospective, Observational Study. J Urol. 2023 Mar;209(3):515-524.

15. Katayama S, Pradere B, Grossman NC, Potretzke AM, Boorjian SA, Ghoreifi A, Daneshmand S, Djaladat H, Sfakianos JP, Mari A, Khene ZE, D'Andrea D, Hayakawa N, Breda A, Fontana M, Fujita K, Antonelli A, van Doeveren T, Steinbach C, Mori K, Laukhtina E, Rouprêt M, Margulis V, Karakiewicz PI, Araki M, Compérat E, Nasu Y, Shariat SF. Biological and prognostic implications of biopsy upgrading for high-grade upper tract urothelial carcinoma at nephroureterectomy. Int J Urol. 2023 Jan;30(1):63-69.

16. Mihalopoulos M, Yaghoubian A, Razdan S, Khusid JA, Mehrazin R, Badani KK, Sfakianos JP, Atallah WM, Tewari AK, Wiklund P, Gupta M, Kyprianou N. Understanding the link between kidney stones and cancers of the upper urinary tract and bladder. Am J Clin Exp Urol. 2022 Oct 15;10(5):277-298. 1

17. Bieber C, Katims A, Ranti D, Sfakianos JP, Amend G. Isolated corpora cavernosa germ cell tumor metastasis requiring complex excision and reconstruction. Urol Case Rep. 2022 Sep 23;45.

18. Reike MJ, Ingersoll MA, Müller DC, Zuiverloon TCM, Strandgaard T, Kamat AM, Williams SB, Seiler R, Todenhöfer T, Dyrskjøt L, Nawroth R, Goebell P, Schmitz-Dräger B, Sfakianos JP, Meeks J, Horowitz A, Black PC. Biology of BCG response in non-muscle invasive bladder cancer - 2021 IBCN Updates Part III. Urol Oncol. 2023 May;41(5):211-218.

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24. Ranti D, Bieber C, Wang YS, Sfakianos JP, Horowitz A. Natural killer cells: unlocking new treatments for bladder cancer. Trends Cancer. 2022 Aug;8(8):698-710

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worse renal function decline after robotic assisted radical cystectomy: Implications for postoperative management. Urol Oncol. 2022 Jun;40(6):275.e11-275.e18. doi: 10.1016/j.urolonc.2022.02.011. Epub 2022 Apr 23. PMID: 35473916.

26. König F, Grossmann NC, Soria F, D'Andrea D, Juvet T, Potretzke A, Djaladat H, Ghoreifi A, Kikuchi E, Hayakawa N, Mari A, Khene ZE, Fujita K, Raman JD, Breda A, Fontana M, Sfakianos JP, Pfail JL, Laukhtina E, Rajwa P, Pallauf M, Cacciamani GE, van Doeveren T, Boormans JL, Antonelli A, Jamil M, Abdollah F, Budzyn J, Ploussard G, Heidenreich A, Daneshmand S, Boorjian SA, Rouprêt M, Rink M, Shariat SF, Pradere B. Pentafecta for Radical Nephroureterectomy in Patients with High-Risk Upper Tract Urothelial Carcinoma: A Proposal for Standardization of Quality Care Metrics. Cancers (Basel). 2022 Mar 31;14(7):1781.

27. Ranti D, Pfail J, Garcia M, Razdan S, Bieber C, Rosenzweig S, Waingankar N, Hosseini A, Radros J, Mehrazin R, Lavallée E, Wiklund PN, Sfakianos JP. Neobladder creation in patients with chronic kidney disease: A viable diversion strategy. Urol Oncol. 2022 Apr;40(4):168.

28. Dovey Z, Pfail J, Martini A, Steineck G, Dey L, Renström L, Hosseini A, Sfakianos JP, Wiklund P. Bladder Cancer (NMIBC) in a population-based cohort from Stockholm County with long-term follow-up; A comparative analysis of prediction models for recurrence and progression, including external validation of the updated 2021 E.A.U. model. Urol Oncol. 2022 Mar;40(3).

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Books and Book Chapters

Eilender B, Katims A, Pfail J, Sfakianos JP. Evelving Treatment in Non-Muscle invasive bladder cancer. Springer 2022.

Bhardwaj N, Farkas A, Gul Z and Sfakianos JP. Harnessing natural killer cell function for genitourinary Cancers. Elsevier 2020

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Administrative Leadership Appointments

Internal
Not Applicable

External
Not Applicable

Publications

Invited Lectures/Presentation (2014- Present) *Presentations at scientific meetings

2023 Immunobiology of BCG unresponsive non-muscle invasive bladder cancer
Venue: AUA designee to the Congress of the SIU Istanbul, Turkey*

2023 BCG and non-muscle invasive bladder cancer: so many unanswered questions
Venue: Paul Lange Visiting Professor University of Washington, Seattle, WA

2023 BCG: Mechanisms of Action Venue: Canada Bladder Cancer Forum Kingston, ON*

2023 Dually targeting NK cells and CD8 T cells for the treatment of bladder cancer Venue: Visiting Professor Centre de Recherche des Cordeliers (CRC) Paris, France

2023 Robotic Assisted Laparoscopic Retroperitoneal Lymph node Dissection: Change of Tides?
Venue: Challenges in Laparoscopic and Robotics (CLIR) Stockholm, Sweden*

2022 Neoadjuvant program-Surgeon's Perspective Venue: SITC Boston, MA*

2022 BCG: past, present, future Venue: Congress of the SIU Montreal, CA*

2022 Immune landscape of T1 urothelial carcinoma what does it mean for future therapeutic targets?
Venue: 8th Annual symposium of the Albert Institute for Bladder Cancer and Research, Houston, TX*

2022 MIS and Pain Control in Urologic Surgery Venue: 8th ERAS world Congress Madrid, Spain*

2022 Open vs Robotic Radical cystectomy are we at a turning point? Venue: 25th Panhellenic Congress of urology Athens, Greece*

2022 Retroperitoneal Lymphadenopathy tips and tricks to avoid vascular injury. Venue: North American Robotic Urologic Symposium Las Vegas, NV*

2022 Retroperitoneal Lymphadenopathy tips and tricks to avoid vascular injury Venue: North American Robotic Urologic Symposium Las Vegas, NV*

2021 Urothelial Carcinoma: Past, Present and Future Venue: Visiting Professor Lenox Hill Hospital New York, NY

2020 Endoscopic Management of Upper Tract Urothelial carcinoma Venue: 22nd Panhellenic Congress of urology Kalamata, Greece*

2019 Unraveling the role of natural killer cells in urothelial carcinoma of the bladder. Venue: Englander Institute for Precision Medicine at Weill Cornell seminar series New York, NY *

2019 DVT prophylaxis and pain control in urologic surgery Venue: ERAS world Congress Liverpool, UK*

2018 Upper Tract Urothelial Carcinoma can “-omics” drive Care? Venue: Bladder cancer advocacy network (BCAN) think tank Denver, CO*

Media Resource Educational Materials

2021 UroToday Interview with Ashish Kamat (MD Anderson), John Sfakianos (ISMMS) and Amir Horowitz.
Novel immune checkpoint axis to understand BCG resistance and improve treatment in non-muscle-invasive bladder cancer

2018 Medscape Education
Clinical Pearls of the Management of Immune-Mediated AEs

Appendix 2

List of Expert Testimony in Last 4 Years
John Sfakianos, MD

Trial:
9/16/2024 – Rosado vs Peters (New Jersey)

Deposition:

01/22/2025 – Leonard vs Kim Jr (New Jersey)

08/21/2024 – Dery vs. Quest (Florida)

11/10/2021 – Jensen vs. Mykulak (New Jersey)

10/28/2021 – Rosado vs Peters (New Jersey)