

Exhibit 347

Specific Causation Expert Report for Jefferson Criswell Thomas Longo, M.D.

Urologic Oncologist
Associated Urologists of North Carolina, PA
160 MacGregor Pines Drive
Cary, NC 27511



Thomas Longo, MD

Date: February 7, 2025

I. Background

I am a board-certified urologist, with a subspecialty in urologic oncology. I went to medical school at the University of Nebraska, where I then continued on for my residency in urology. Immediately following my residency, I came to Duke University as the Society of Urologic Oncology Fellow. The Society of Urologic Oncology Mission statement is as follows: “The goal of an SUO fellowship is to provide additional training in Urologic Oncology above and beyond residency. This training will specifically provide a multidisciplinary exposure to Urologic Oncology and provide an opportunity to spend an extended period of time devoted to the care of patients with genitourinary malignancies and urologic oncology research.” It is a two-year fellowship roughly split in half between research and patient care.

Following the completion of my fellowship, I remained on faculty at Duke in my capacity as a urologic oncologist. My focus was solely on genitourinary malignancies with an emphasis on bladder cancer. My responsibilities and privileges included patient care and resident training. In the summer of 2023, I left Duke to join a private urology group, with a continued focus on genitourinary malignancies and bladder cancer. Given the geographic proximity of Duke to Camp Lejeune, I became increasingly familiar with the increased risk of malignancy associated with Camp Lejeune, and encountered exposed patients within my own practice.

Therefore, I was familiar with the hazards posed to human health associated with Camp Lejeune’s contaminated water prior to my involvement with the Camp Lejeune litigation. As early as 2015, when I began my fellowship, I was familiarizing myself with the Camp Lejeune water contamination with the goal of being a well-informed clinician.

Additionally, I am a bioethicist with a certificate in bioethics from the National Catholic Bioethics Center. Bioethics are intimately incorporated in my practice as a clinician, and in my decision to accept a role as an expert witness in cases involving malignancies from toxic exposure to Camp Lejeune water. The scientific dimension of each individual case represents the descriptive aspect of reality. The philosophical dimension, “sheds a light of clarity on the summit of truth” (Sgreccia). This valuation of moral acts is linked to the legal dimension. Included within the field of bioethics are the decisions the state makes regarding its citizens for the sake of their health.

II. Mandate

I have been asked to provide my opinion on the causation of Michael Criswell’s diagnosis of bladder cancer. In order to provide this opinion, I reviewed scientific and medical evidence concerning the relationship between exposure to the chemicals in the water at Camp Lejeune to bladder cancer, and specifically the relationship between persons exposed to contaminated water at Camp Lejeune and the development of bladder cancer. In addition, I have reviewed documents that pertain to Mr. Criswell, including deposition transcripts, discovery material, and medical records. To assist me, I have reviewed the bladder cancer general causation reports of Dr. Bird, Dr. Culp, Dr.

Gilbert, Dr. Hatten, and Dr. Plunkett, as well as medical and scientific literature concerning diagnosis of bladder cancer, the treatment options, and generally the risk factors for bladder cancer. Furthermore, I am relying on my training and experience.

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. Criswell'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. All of my opinions in this report are expressed to a reasonable degree of scientific and medical certainty.

III. Summary of Opinions

Bladder cancer is a disease of toxic exposure. Bladder cancer is colloquially used for the pathologic diagnosis of urothelial cell carcinoma of the bladder. Although there are different types of bladder cancer, over 90% of bladder cancer is urothelial cell carcinoma. There are known occupational risks, environmental contaminants, medicines, bladder irritants and infections, familial syndromes, and even treatments for cancer such as chemotherapy and radiation that elevate the incidence of bladder cancer. In the sections below I outline my opinions concerning the chemicals that Mr. Criswell was exposed to at Camp Lejeune as carcinogens causing bladder cancer and that exposure to these chemicals are as likely as not a cause of Mr. Criswell's diagnosis of bladder cancer.

IV. Methodology

As a specialist in urologic oncology, I am well-informed on the suspected causes of bladder cancer in humans for which I rely on my education, knowledge, training and experience. I attempt to stay up to date on these issues in my practice through review of relevant peer-reviewed journal articles and other reputable sources like IARC Monographs and the EPA. In order to rule in a new risk factor as a potential cause of bladder cancer, I review relevant sources to determine if they sufficiently establish a causal relationship between the risk factor and bladder cancer. To that end, in this case, I reviewed the Plaintiffs' general causation reports and sources such as peer-reviewed literature, ATSDR and EPA documents, and IARC Monographs. I incorporate and rely on the general causation reports for my opinions herein. I reserve the right to

supplement my opinions if additional information becomes available that may be relevant to my opinions.

As I would in my practice, after ruling in any potential risk factors as a potential cause, I conducted a differential etiology to determine what risk factor(s) contributed to cause the development of Mr. Criswell's cancer.

I am being compensated at a rate of \$750 per hour for review and \$1,000 per hour for deposition and related preparation.

V. Mr. Criswell's Factual Background

1. Mr. Criswell's Exposure at Camp Lejeune

Mr. Criswell was stationed at Camp Lejeune from January 4, 1975 until April 1, 1977 with his wife. Initially, Mr. Criswell resided with his wife's uncle at Camp Geiger before receiving his base housing.¹ Records reflect Mr. Criswell resided in Tarawa Terrace at 3155 Bougainville Drive, from July 29, 1975 until March 31, 1977 in a 2 bedroom family residence. Mr. Criswell was largely present at Camp Lejeune during this time period except for leave he took from September 1, 1976 to September 15, 1976 and during times he was off base for cross-country chasing.

While at Camp Lejeune, Mr. Criswell resided at Tarawa Terrace but mainly worked at Mainside-Hadnot Point. As a result, Mr. Criswell was exposed to the water at both Tarawa Terrace and Hadnot Point.² He would shower, wash dishes, bath, drink water and tea at Tarawa Terrace, and he would consume water Monday through Friday during PT training and work while on Mainside-Hadnot Point. Mr. Criswell's exposure to the water at Camp Lejeune was constant, consistent and cumulative. He was in the best shape of his life, with PT training at 6:30 every morning and had to stay hydrated.³

As an active marine, in the best shape of his life, Mr. Criswell was always exposed to the toxins present in the water at Camp Lejeune. Mr. Criswell drank, inhaled, and was dermally exposed the toxins everyday he was present on base. The ATSDR assume marines similar to Mr. Criswell ingested 4 liters of water per day, with up to 8 liters of water when considering marines dermal and inhalation exposures. (ATSDR 2017a). The toxins present in the water at Camp Lejeune are all absorbed into the body through ingestion, dermal and inhalation exposure pathways. (see general causation reports). PCE, TCE, VC and Benzene were all present in the water at both Hadnot Point and Tarawa Terrace during Mr. Criswell's exposure period. These toxins are all known carcinogens capable of promoting bladder cancer. Two of these toxins (PCE and TCE) were recently banned by the EPA. All these toxins form reactive/toxic metabolites after exposure that circulate in the blood though the liver. Once the reactive metabolites are present in, or formed in the kidney, basic human anatomy dictates that they may act locally in the kidney but also

¹ Criswell Dep. 71-72:16

² Criswell Dep321:18-25

³ Criswell Dep. 321:20-25

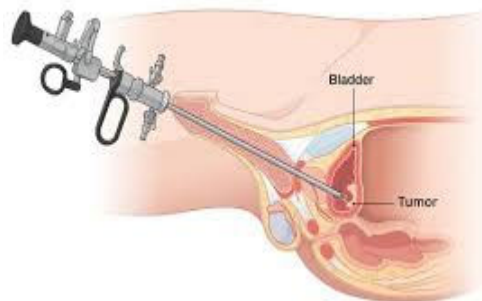
would be excreted into urine and pass from the kidney into the bladder. (Plunkett, 73). As a result, for approximately 800 days, Mr. Criswell was consistently exposed to the carcinogens with no recovery period – the toxins were at least as likely as not consistently present in his body resulting in a substantial exposure to PCE, TCE, VC and Benzene.

2. Mr. Criswell's Occupational History

Before his diagnosis of bladder cancer, Mr. Criswell was a police officer in Fayetteville, Georgia, before becoming a schoolteacher.⁴ He taught from 1986 until his retirement in 2004.⁵ He then was a franchisee for Firehouse Subs. There is no evidence Mr. Criswell was exposed to any toxins in his employment after Camp Lejeune.⁶

3. Mr. Criswell's Bladder Cancer Medical History

In October 1997, Mr. Criswell – at age 42 – presented to his primary care physician, Dr. Robertson, with complaints of pain for the last several weeks in his left lower abdominal and blood-tinged urine.⁷ The intravenous pyelogram (IVP) showed a 2 cm calcification. Mr. Criswell was referred to a urologist, Dr. Moseley. Dr. Moseley performed a cystoscopy which uncovered a 2 to 2.5 cm bladder tumor over the left lateral left posterior wall with some mucosal changes extending down over the left hemitrigone. Dr. Moseley noted at the time that Mr. Criswell did not smoke or drink.⁸ A transurethral resection of the bladder tumor (TURBT) was recommended and performed on October 20, 1997 by Dr. Moseley. A TURBT involves inserting an instrument into the urethra and bladder and cutting and cauterizing bladder tumors off the inner lumen of the bladder.



⁴ Criswell Depo 148:1-23

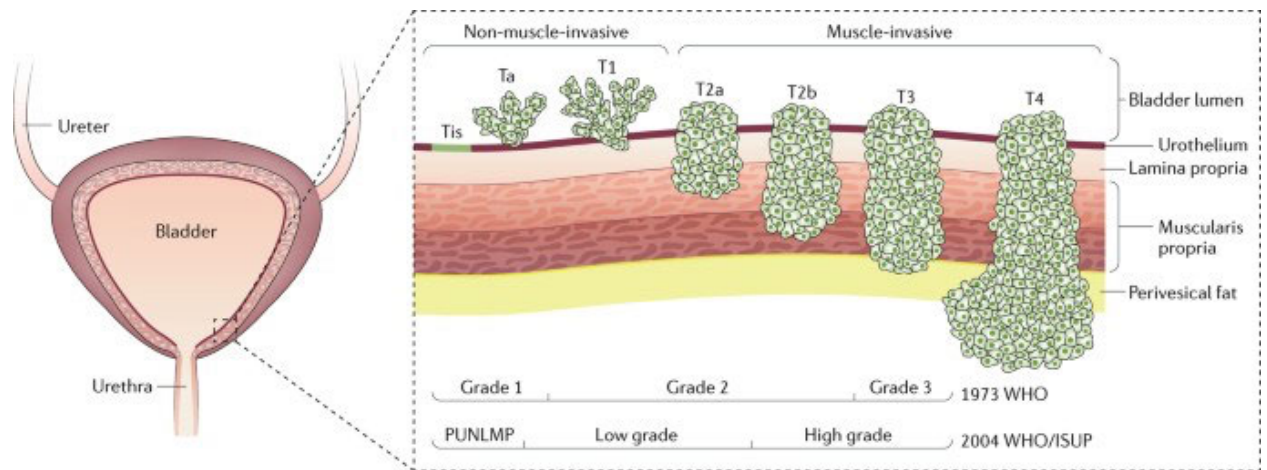
⁵ Criswell Depo 149:12-20

⁶ DPPF, Criswell Depo 147:7-10

⁷ 01482_CRISWELL_0000000095

⁸ 01482_CRISWELL_0000000101

This procedure is standard but can lead to infection, bleeding, structure, bladder perforation, and dissemination of the cancer. The pathology revealed Grade II Papillary Transitional Cell Carcinoma, Invasive, carcinoma in situ.⁹



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Nature Reviews | Disease Primers

Following his bladder cancer diagnosis and TURBT, Mr. Criswell was presented with a difficult situation at age 42. Dr. Moseley explained that with a High Grade II Superficial Muscle Invading Bladder Carcinoma, radical cystectomy of the bladder with orthotopic pouch was standard treatment.¹¹ Mr. Criswell elected to start with six courses of BCG treatment.

BCG is an attenuated live vaccine used for tuberculosis. It directly attacks cancer cells, and it stimulates the immune system to attack cancer cells. It does this by activating T cells to attack abnormal bladder urothelium. Studies have shown it to reduce the likelihood of recurrence and reduce the likelihood of tumor progression. Of all the intravesical agents, only BCG has been shown to inhibit tumor progression, which is preventing non muscle invasive bladder cancer from becoming muscle invasive bladder cancer.

Many treatment protocols exist, but the best evidence is for the Southwest Oncology Group (SWOG) protocol. This management option would consist of induction (6 weekly installations) of intravesical therapy followed by maintenance therapy (3 weekly installations at 3, 6, 12, 18,24, 30, and 36 months). Only 16% of patients will complete the entire 3 years of maintenance. Typically, the patient is unable to complete the entire regimen because of BCG toxicity (side effects are too great). A more recent study of BCG compliance is even lower in clinical practice. In particular, the SWOG protocol (induction with a maintenance program) is only completed by about 10% of patients who begin BCG

⁹ 01482 CRISWELL 0000000107

¹⁰ Chalasani V, Chin JL, Izawa JI. Histologic variants of urothelial bladder cancer and nonurothelial histology in bladder cancer. *Can Urol Assoc J.* Dec 2009;3(6 Suppl 4):S193-8.

¹¹ 01482_CRISWELL_0000000108

therapy. In addition to patient driven compliance, BCG shortages are common, and frequently occur during a treatment regimen. This is so common, that the American Urological Association has issued guidance that patients may be given a reduced 1/2 to 1/3 dose. Thus, the treatment he received is comparable to many bladder cancer patients.

The process of bladder instillation involves placing a catheter through the urethra into the patient's bladder. The medication is then injected through the catheter into the bladder and must be held for two hours without urinating. BCG has the most side effects of anything instilled into the bladder. Most common side effects are: cystitis, dysuria, hematuria, malaise, fatigue, and low grade fever. Typically, these resolve within a few days of treatment. More significant side effects a high fever, and the worst is termed BCG sepsis. This occurs in less than 1% of patients, but requires an initial hospitalization followed by 6 months of anti-tuberculosis drugs. Another rare, but devastating side effect is a contracted bladder. This can be to such a degree that the patient will lose all bladder capacity and require a cystectomy.

On December 29, 1997, Mr. Criswell underwent an additional surgery and biopsy in which the pathology revealed superficial invasion of the muscularis, but no deep invasion of the muscularis.¹² This was followed with three additional courses of maintenance BCG treatments. Following his BCG maintenance treatments, Mr. Criswell developed severe crippling irritative symptoms, mainly frequency and urgency to the point that he was unable to work. In April 1998, Mr. Criswell reported his inability to sleep at night due to his increased frequency and passing what appeared to be "tissue."¹³ Another cystoscopy was performed and biopsies revealed mild chronic follicular cystitis, consistent with BCG inflammatory response. Concern over future BCG treatment was expressed by Dr. Mosely, since Mr. Criswell was considered a "urological cripple" since his last BCG treatment.¹⁴ At the time, Dr. Moseley elected not to proceed with more BCG treatment due to Mr. Criswell's reactions. Mr. Criswell was recommended a plan of monitoring cystoscopies every 3 months.

A follow up cystoscopy was performed on July 31, 1998, and the pathology revealed no evidence of carcinoma in situ.¹⁵ In February 1999, another cystoscopy revealed a small tumor necessitating another TURBT procedure for removal of the tumor. Pathology showed mild chronic follicular cystitis, negative for malignancy.¹⁶ In August 1999, another cystoscopy was performed and there were concerns of lesions appearing to be carcinoma in situ.¹⁷ On August 18, 1999, Mr. Criswell underwent another TURBT procedure to remove the lesions. Pathology returned negative for malignancy.¹⁸ .

¹² 01482_CRISWELL_0000000113

¹³ 01482_CRISWELL_0000000123

¹⁴ 01482_CRISWELL_0000000124

¹⁵ 01482_CRISWELL_0000000141

¹⁶ 01482_CRISWELL_0000000150

¹⁷ 01482_CRISWELL_0000000152

¹⁸ 01482_CRISWELL_0000000156

Mr. Criswell stated that “they weren’t able to get it all...it was deeper than anticipated. I had reactions to treatments. I had no bladder control. I was suicidal, was drugged up on drugs to the point didn’t want to live. At one point I had a gun to my head. I couldn’t use the same bathroom as others in my house. I was that toxic. I had bags strapped to my legs because of uncontrollable bladder...I found out months later that treatment worked but I would go back to the doctor and he would say you have a tumor we need a biopsy. I couldn’t function or go anywhere; everywhere I went I had to have jugs with me for urination. I went back to the doctor and was told there was no tumor then months later was told that I have a tumor and I needed biopsy. I went through that for two years. Every three months the doctor was cutting on me. That brought on a lot of problems with depression.”¹⁹

In December 1999, Mr. Criswell sought out a second opinion from Dr. Scott Shelfo. Dr. Shelfo performed another cystoscopy which was negative. Mr. Criswell had several follow-up cystoscopies with Dr. Shelfo from 1999 to 2002 with negative findings. In October 2002, Dr. Shelfo performed a cystoscopy with negative findings of tumor or erythema. Mr. Criswell was, at that point, five years with no evidence of disease. At the time, Mr. Criswell believed he was cancer-free.

In August 2012, Mr. Criswell sought a medical opinion from the Department of Veteran Affairs regarding his bladder cancer diagnosis for disability benefits. He was seen by Dr. Ambardekar. After an examination and review of his medical history, the Department of Veteran Affairs determined Mr. Criswell’s bladder cancer is at least as likely as not caused by, or a result of, the veteran’s exposure to contaminated water at Camp Lejeune.²⁰

¹⁹ 01482_CRISWELL_0000001051

²⁰ 01482_CRISWELL_VBA_0000000551

MEDICAL OPINION

Bladder cancer

The veteran was stationed at camp Lejeune during his service with exposure to contaminated water. He denies having any family history of bladder cancer, any significant history of smoking, and denies any other exposure to chemicals prior to or after his service. The national research council published a report which included a review of studies addressing exposure to TCE and or PCE and discussion of disease conditions which were identified as having limited/suggestive evidence of an association. One of these conditions includes bladder cancer.

The veteran's bladder cancer is at least as likely as not caused by or a result of the veteran's exposure to contaminated water at camp Lejeune.

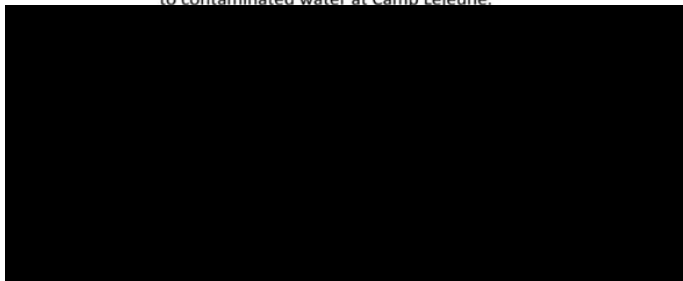
His current urinalysis, PSA and chemistry panel are normal.

NOTE: VA may request additional medical information, including additional examinations if necessary to complete VA's review of the Veteran's application.

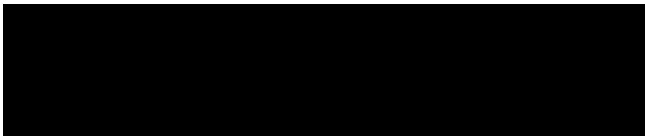
/es/ NAYANA Y AMBARDEKAR MD
STAFF PHYSICIAN
Signed: 08/20/2012 12:35

Additionally, Dr. Sherry Hills, a clinical psychologist with the Department of Veteran Affairs found Mr. Criswell's depression at least as likely as not proximately due to, or the result, of his bladder cancer. The rationale was based upon the silence of Mr. Criswell's service records regarding complaints of depression. Records in the c-file indicate that treatment for depression began shortly after treatment for bladder cancer.²¹ Furthermore, Dr. Valerie Vroon Raymond, Psy.D., evaluated Mr. Criswell and provided a report finding Mr. Criswell's bladder cancer contributes to his anxiety and depression."²² As a result of his diagnosis Mr. Criswell was prescribed with Zoloft and Wellbutrin.

c. Rationale: On August 20, 2012, the veteran was evaluated by Dr. Ambadaker who opined that The veteran's bladder cancer was at least as likely as not caused by or a result of the veteran's exposure to contaminated water at Camp Lejeune.



RATIONALE



²¹ 01482_CRISWELL_0000001057

²² 01482_CRISWELL_0000000621

In February 2014, Mr. Criswell returned to Dr. Shelfo after his life insurance application was denied. Mr. Criswell did have a lapse of several years in surveillance. It is my opinion that this did not impact his recurrence or the ultimate outcome. Surveillance of bladder cancer does not prevent recurrence of bladder cancer. It does allow for early detection, and that could lead to detecting the cancer at a lower stage before it has the opportunity to progress. Following his gap in surveillance, he resumed urologic care, and it was several years after that when he suffered a recurrence. In Mr. Criswell's case, he had a low grade, noninvasive recurrence; the gap in surveillance did not impact the outcome.

Dr. Shelfo performed a cystoscopy which revealed a recurrence of his bladder cancer with 2 small low grade pap lesions. Mr. Criswell underwent TUBRT on March 4, 2014²³ and the pathology report indicated noninvasive low-grade papillary urothelial carcinoma, muscularis propria was present but no evidence of invasion.²⁴ Following the procedure, Mr. Criswell received a one-time dose of mitomycin-c, a chemotherapy drug which works by damaging the DNA of cancer cells, preventing their replication.

Following his recurrence, Mr. Criswell's depression worsened.²⁵ He was diagnosed with major depression after developing his bladder cancer which has been attributed to the contaminated water at Camp Lejeune. His symptoms included poor sleep, impaired concentration, poor attention, distractibility, unprovoked irritability, anhedonia with intermittent passive suicidal ideations. Due to his impaired concentration and attention, his family members became frustrated with his ability to maintain effective relationships. He was prescribed Zoloft 50mg for his depressive symptoms.²⁶

Dr. Shelfo performed follow up cystoscopies in December 2014, June 2015, December 2015 with negative findings. A June 2016 cystoscopy revealed small papillary changes necessitating another TURBT.²⁷ In August 2016, Mr. Criswell underwent TURBT and the pathology returned with his second recurrence of low-grade noninvasive papillary urothelial cell carcinoma.²⁸ A dose of mitomycin-c was administered via catheter.

Monitoring cystoscopies administered by Dr. Shelfo every six months until February 2018 cystoscopy showed small 2 cm area of mild papillary changes. Mr. Criswell underwent a rigid cystoscopy with retrograde pyelogram which was negative followed by mitomycin chemotherapy.²⁹ Monitoring cystoscopies were performed with negative findings until December 2022 when there were slightly swollen red areas on the anterior wall. Retrograde pyelograms and biopsy were performed in January 2023 with

²³ 01482_CRISWELL_0000000594

²⁴ 01482_CRISWELL_VBA_0000002276

²⁵ 01482_CRISWELL_0000001914

²⁶ 01482_CRISWELL_00000000551

²⁷ 01482_CRISWELL_0000000025

²⁸ 01482_CRISWELL_0000000427

²⁹ Shelfo Dep. 87:1-11

negative results. Most recent cystoscopy of January 2024 was negative. Annual cystoscopies are necessary due to the high recurrence rate.³⁰

VI. Exposure Assessment

ATSDR performed extensive water modeling and arrived at certain conclusions as to the estimated monthly concentrations of TCE, PCE, benzene, and vinyl chloride in the water at the Hadnot Point, Tarawa Terrace, and Holcomb Boulevard water treatment plants. Dr. Kelly Reynolds, who I understand has been retained by the plaintiffs, authored a report that utilizes ATSDR's water modeling results and then develops data showing the anticipated ingestion figures for Mr. Criswell. It is important to note that Dr. Reynolds' analysis only considers ingestion and not the other two recognized routes of exposure (inhalation and dermal). As one study explained, "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."³¹ Thus, Mr. Criswell's actual exposure is higher than what Dr. Reynolds shows, but nonetheless he suffered from substantial exposure to the chemicals at Camp Lejeune.

Dr. Reynolds composed the total days of Mr. Criswell's exposure to the Camp Lejeune water at approximately 800 days of exposure. From ATSDR's estimated consumption of water Dr. Reynolds calculated the following total concentrations that Mr. Criswell was exposed to on Camp Lejeune are identified below:

³⁰ Shelfo Dep. 95:11-17

³¹ 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.

		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition/FM
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Hadnot Point				
TCE	8,357	86,022	345,628	534,241
PCE	258	2,640	23,337	34,466
VC	380	3,897	34,441	50,865
BZ	85	858	7,581	11,196
Terawa Terrace				
TCE	46	911	3,905	6,036
PCE (TechFlowMP Model)	1,147	22,510	96,472	149,118
PCE (MT3DMS Model)	1,594	31,267	134,002	207,128
VC	82	1,610	6,899	10,663
BZ	-	-	-	-
Totals HP & TT				
TCE	8,403	86,933	349,534	540,278
PCE (TechFlowMP Model)	1,405	25,151	119,809	183,584
PCE (MT3DMS Model)	1,852	33,908	157,339	241,594
VC	462	5,507	41,339	61,528
BZ	85	858	7,581	11,196

I have also reviewed the reports of Drs. Benjamin Hatten and Steven Bird, who both identified hazardous levels of exposure that demonstrate toxic levels of TCE, PCE, vinyl chloride and benzene that Mr. Criswell met or exceeded.

Viewing Dr. Reynolds' exposure numbers against these demonstrated toxic levels clearly establishes that Mr. Criswell's exposure was significant and substantial. Mr. Criswell exceeds each of the demonstrated levels set out here.

The opinions of Dr. Reynolds, Dr. Hatten, and Dr. Bird confirm that Mr. Criswell'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.

VII. General causation

Before advancing to the application of a differential etiology for Mr. Criswell 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, VC, 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...”³² EPA concluded that “TCE is carcinogenic to humans by all routes of exposure,” that is, by ingestion, inhalation, and dermal exposure.³³ Further, EPA concluded that PCE is “likely to be carcinogenic in humans by all routes of exposure.”³⁴ Similarly, the National Toxicology Program has recognized TCE as “known to be a human carcinogen”³⁵ and PCE as “reasonably anticipated to be a human carcinogen.”³⁶ 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.³⁷

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 the kidneys into urine where it sits in the bladder until voided.³⁸ Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well.³⁹ 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.

³² 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

³³ Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011

³⁴ Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012

³⁵ National Toxicology Program (NTP). 2015. Report on Carcinogens monograph on trichloroethylene. Research Triangle Park, NC: National Toxicology Program. RoC Monograph 05

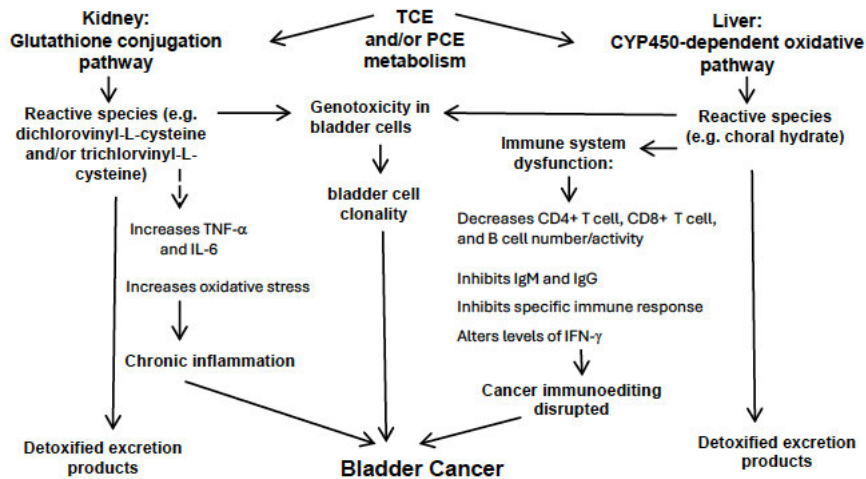
³⁶ NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service

³⁷ Hatten pp. 26-29; 31-32; Plunkett ¶¶ 47

³⁸ Plunkett ¶¶ 33, 43; Hatten p. 39; Bird pp. 17-18

³⁹ Plunkett ¶¶ 52, 56, 59

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).⁴⁰ 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.⁴¹ 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.⁴²

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 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.”⁴³ 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.

⁴⁰ Gilbert p. 30

⁴¹ Gilbert p. 32-3

⁴² Gilbert p. 19-20

⁴³ Bird Suppl. p. 1

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

VIII. Differential etiology

In order to assess whether Mr. Criswell's exposure to the water at Camp Lejeune as likely as not caused him to develop bladder cancer, I employed a differential etiology under which an expert considers the relevant potential causes of a disease and then attempts to ascertain whether any of those causes can be eliminated. I employ this analysis in my practice to assist in the treatment options I provide my patients.

During an encounter for bladder cancer, a treating 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, whose 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.”⁴⁴ These risk factors act in concert with one another, and have additive effects. In Mr. Criswell's case, as I discuss later in this report, he only had one recognized risk factor: exposure to the chemicals in the water at Camp Lejeune.

Before addressing the potential risk factors for bladder cancer, it is important to address whether the cause of bladder cancer is idiopathic. In a deposition of Mr. Criswell's treatment provider – Dr. Shelfo – the government asked whether the cause of bladder cancer is idiopathic.⁴⁵ When a disease is idiopathic it means that the cause of the disease is unknown. In my opinion, bladder cancer is rarely idiopathic in the sense that it is likely to have a known cause. In situations where an individual was exposed to a known cause of bladder cancer, considering whether the cause is idiopathic is inapplicable. As I

⁴⁴ <https://www.cancer.org/content/dam/CRC/PDF/Public/8558.00.pdf>

⁴⁵ Shelfo Dep. 33:4-12

describe in this report, I do not consider Mr. Criswell's bladder cancer as idiopathic because he has been exposed to a known and widely-accepted cause of bladder cancer.

There are a number of risk factors that have been identified as potentially causing or contributing to a diagnosis of bladder cancer. These risk factors are listed below:

1. Exposure to chlorinated solvents or others toxic chemicals.

There is a substantial body of evidence establishing that the chemicals present at Camp Lejeune are carcinogenic and cause bladder cancer. When looking at information on the carcinogenicity of chemicals, my field considers a range of reliable sources, including the United States Environmental Protection Agency (U.S. EPA), the International Agency for Research on Cancer (IARC), and the Agency for Substances and Disease Registry (ATSDR). Each of these organizations form multidisciplinary groups to systematically survey the body of available scientific literature and concluded that TCE and PCE are either human carcinogens or likely human bladder carcinogens. They are transparent and reflective of current evidence review and evaluation methods, to enhance integration of different lines of evidence and to incorporate emerging data streams from various approaches.

Regarding PCE, a systematic literature review by the U.S. EPA concluded that bladder cancer was one of the human tumor types associated with tetrachloroethylene exposure, noting a consistent and elevated risk from epidemiological studies associating PCE and bladder cancer. (EPA PCE tox review 2012). The U.S. EPA characterized tetrachloroethylene as "likely to be carcinogenic to humans" based on suggestive evidence of carcinogenicity in epidemiological studies and conclusive evidence of tumorigenicity in rodents. IARC published assessments of the association of PCE and cancer, ultimately classifying PCE within Group 2A as "probably carcinogenic to humans" (IARC 2014). IARC noted that the bladder in particular "has been identified as a target organ" for PCE. (IARC 2014). Several strong epidemiological studies have shown a clear trend toward an elevated risk of developing bladder cancer following exposure to PCE. These studies include Lynge 2006 (dry cleaners and laundry workers in Nordic countries showing a relative risk of 1.44), Blair 2003 (cohort study of dry cleaners in St. Louis, Missouri, SMR of 2.9), Aschengrau 1993 (case control stud in Massachusetts showing an odds ratio of 1.55 for any exposure, and an odds ratio of 1.16 for low exposure). In addition, Vlaanderen 2014 is a persuasive meta-analysis that showed an elevated association with bladder cancer (mRR 1.47). The strength of association has increased since EPA's 2012 review and IARC's 2014 review of the epidemiological literature. For example, Sciannameo 2019 studied male bladder cancer patients in Italy showed an increased association with low dose exposure of PCE and bladder cancer (OR 1.40). Hadkhale 2016 found an elevated association with medium occupational exposure to PCE and bladder cancer (HR 1.12).

Regarding TCE, the U.S. EPA's 2011 Toxicological Review included several studies demonstrating a positive association, including Anttila 1995 (Finnish workers exposed to TCE had a higher measure of association with bladder cancer in those followed 20 years or more; SIR 1.51); Morgan 1998 (aerospace workers in Arizona with medium and high exposure showed elevated association to bladder cancer; RR 1.41); Boice 2006 (elevated association with bladder cancer found in rocket engine workers' RR of 1.27 and 1.15 for medium and high levels of exposure); Ritz 1999 (Ohio uranium processing workers; SMR 1.17); Raaschou-Nielsen 2003 (Denmark workers showed an increase in females diagnosed with bladder cancer following TCE exposure; SIR 1.6); Morgan 1998 (pooled study showing m-SMR of 1.15)). A meta-analysis of seven studies of dry-cleaning workers observed a significantly elevated risk of bladder cancer in workers exposed to TCE (Vlaanderen 2014). The implication is this is from exposure to TCE. IARC published assessments of the association of TCE with cancer. Human studies were used to determine what specific kinds of cancer each solvent likely caused. Regarding TCE, IARC found sufficient evidence in humans and animals to conclude that TCE causes cancer.

ATSDR's Camp Lejeune studies confirm a combined exposure to TCE, PCE, and benzene can cause bladder cancer. ATSDR performed several cohort studies that compared marines stationed from 1972 to 1985 at Camp Lejeune and those stationed at Camp Pendleton. In ATSDR's 2018 morbidity study, marines at Camp Lejeune showed 64% highest morbidity than their peers at Camp Pendleton. A follow up cancer incidence study of this similar cohort (those at Camp Lejeune and Camp Pendleton from 1975-1985) showed a 9% increased risk for marines and a 10% increased risk for civilian workers. Bladder cancer is known to have a longer latency period owing to the nature of the urothelial cells that line the bladder, which means that the number of bladder cancer cases at Camp Lejeune in a younger cohort⁴⁶ further strengthens support for the chemicals' causative properties.

In addition to chlorinated solvents (such as those used at Camp Lejeune and in the dry-cleaning industry), discussed above, chemicals used in the dye and textile industry⁴⁷, rubber manufacturing industry⁴⁸, painting industry⁴⁹ and aluminum and refined products industry have been shown to have an association with bladder cancer. Mr. Criswell does not have any known occupational or environmental exposures – besides those present at Camp Lejeune – that could cause bladder cancer. Nothing in Mr. Criswell's work history, as a police officer, as a teacher, and as a restaurant owner do

⁴⁶ Bove 2024 ended its data collection in December 2017.

⁴⁷ Singh and Chadha, *Textile industry and occupational cancer* (2016), available at <https://pmc.ncbi.nlm.nih.gov/articles/PMC4986180/>

⁴⁸ IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 100F (2012), available at <https://www.ncbi.nlm.nih.gov/books/NBK304412/>

⁴⁹ Guha *et al.*, Bladder cancer risk in painters: a meta-analysis (2010), available at [https://pubmed.ncbi.nlm.nih.gov/20647380/#:~:text=The%20summary%20relative%20risk%20\(meta,only%20smoking%20adjusted%20risk%20estimates.](https://pubmed.ncbi.nlm.nih.gov/20647380/#:~:text=The%20summary%20relative%20risk%20(meta,only%20smoking%20adjusted%20risk%20estimates.)

stands out as an occupational exposure that would cause or contribute to cause bladder cancer.

2. Tobacco use;

Smoking is a known risk factor for bladder cancer, with some estimates attributing 50% of all bladder cancers to smoking.⁵⁰ However, I do not consider smoking to be a potential cause of Mr. Criswell's bladder cancer. Mr. Criswell's limited smoking history is medically insignificant. It is noted in one of Mr. Criswell's medical records that he is a former smoker, smoking less than half a pack of cigarettes per day between 1972-1974⁵¹, this reference is an outlier to the body of records suggesting far more minimal smoking⁵²⁵³. In his deposition he recounted a minimal smoking history: Mr. Criswell reports that he smoked socially on a handful of occasions as a teenager due to peer pressure in approximately 1973-1974.⁵⁴ Mr. Criswell's treating physician testified that Mr. Criswell's smoking was not significant in his development of cancer due to his minimal usage.⁵⁵

Conventional wisdom would suggest that secondhand exposure to cigarette smoke may also contribute to bladder carcinogenesis. It should be analogous to being a smoker, albeit to a lesser degree. However, there is little evidence to confirm this suspicion. Previous studies reported inconsistent results on the association between ETS exposure and bladder cancer. It may be the low levels of potential tobacco bladder carcinogens present in secondhand smoke. There is a relatively large error in measurement of secondhand smoke for a given individual over lifetime, and this may mask the moderate effect of secondhand smoke on bladder cancer risk. Mr. Criswell was exposed to secondhand smoke from his parents, who he lived with until age 17.⁵⁶

Similarly, chewing tobacco is suspected of having a causal relationship with bladder cancer, but evidence is sparse to support that conclusion. Mr. Criswell reported using chewing tobacco while coaching high school football games for a few years between approximately 1986-1990.⁵⁷ Given the insufficient data to support causality between smokeless tobacco use and development of bladder cancer, to a reasonable degree of medical certainty, chewing tobacco is unlikely a potential cause of Mr. Criswell's bladder cancer.

⁵⁰ Michael J, Matulewicz RS, Bjurlin MA. Assessment of Tobacco Screening and Smoking Cessation Recommendations among Bladder Cancer Guidelines: A Call to Action. *Journal of Urology*. 2022 Mar 1 207(3):490–2. Available from: <https://doi.org/10.1097/JU.0000000000002360>

⁵¹ 01482_Criswell_VBA_0000000288

⁵² 01482_CRISWELL_0000000095

⁵³ 01482_CRISWELL_0000004929

⁵⁴ Criswell Dep. 171:11-172:3

⁵⁵ Shelfo Dep. 77:12-16

⁵⁶ Criswell Dep. 176:1-177:7

⁵⁷ Criswell Dep. 168:1-169:9

There is some evidence that smoking cessation reduces the risk of developing bladder cancer,⁵⁸ and the Centers for Disease Control defines a “nonsmoker” as “[a]n adult who has never smoked, or who has smoked less than 100 cigarettes in his or her lifetime.”⁵⁹ Those who have quit smoking for 25 years or more have the same risk as those who never smoked.⁶⁰ Mr. Criswell’s smoking history characterizes him as a non-smoker for medical purposes. At the very least, and to a reasonable degree of medical certainty, smoking, passive smoking, and chewing tobacco is highly unlikely to be a potential cause of Mr. Criswell’s bladder cancer.

3. Alcohol Consumption

An association between high alcohol consumption and development of bladder cancer has been suggested in some studies.⁶¹ However recent meta-analyses of cohort studies did not observe a significant association between alcohol intake and the risk of bladder cancer.⁶²

Prior to his diagnosis, Mr. Criswell drank approximately two to four times per month, having one to two drinks per occasion.⁶³ Following his diagnosis with bladder cancer and resultant depression, there is evidence of increased alcohol consumption.⁶⁴ Mr. Criswell’s alcohol consumption was limited prior to diagnosis, as such I do not consider alcohol consumption to be a cause of Mr. Criswell’s bladder cancer.

4. Family history of bladder cancer;

⁵⁸ U.S. Department of Health and Human Services. *The Health Benefits of Smoking Cessation: A Report of the Surgeon General*. Rockville (MD): U.S. Department of Health and Human Services, Centers for Disease Control, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1990. DHHS Publication No. (CDC) 90-8416.

⁵⁹ https://archive.cdc.gov/www_cdc_gov/nchs/nhis/tobacco/tobacco_glossary.htm

⁶⁰ U.S. Department of Health and Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.

⁶¹ Vartolomei MD, Iwata T, Roth B, Kimura S, Mathieu R, Ferro M, et al. (2019) Impact of Alcohol Consumption on the Risk of Developing Bladder Cancer: A Systematic Review and Meta-Analysis. *World J Urol* 37(11):2313–24. doi: 10.1007/s00345-019-02825-4

⁶² Lao, Y., Li, X., He, L., Guan, X., Li, R., Wang, Y., Li, Y., Wang, Y., Li, X., Liu, S., & Dong, Z. (2021). Association Between Alcohol Consumption and Risk of Bladder Cancer: A Dose-Response Meta-Analysis of Prospective Cohort Studies. *Frontiers in oncology*, 11, 696676. <https://doi.org/10.3389/fonc.2021.696676>

⁶³ 01482_Criswell_VBA_0000000288

⁶⁴ 01482_Criswell_0000000620

While there have been positive associations between a family history of bladder cancer in first- and second-degree relatives⁶⁵, I do not consider this to be a risk factor for Mr. Criswell. Mr. Criswell has no family history of bladder cancer, though his sister was diagnosed with pancreatic cancer.⁶⁶ However, there is no positive association between family history with respect to non-bladder cancers. I do not consider Mr. Criswell's family history of non-bladder cancers to be a cause of his bladder cancer.

Other known risk factors for bladder cancer were not present in Mr. Criswell's medical or factual profile: including, but not limited to:

1. Past treatment to some anticancer drugs;

Some anti-cancer drugs, cyclophosphamide⁶⁷ and ifosfamide⁶⁸ have been associated with an increase in risk for bladder cancer. However, I do not consider this to be a risk factor for Mr. Criswell as he had no past treatment involving these anticancer drugs prior to being diagnosed with bladder cancer.

2. Other chemical exposure;

Because bladder cancer is a cancer of toxic exposure, there are other chemicals that have an association with bladder cancer. Drinking well water with high levels of arsenic has been found to increase the risk of developing bladder cancer. Saint-Jacques et al. (2014). Additionally, there is some evidence that drinking water treated with chlorine also increases the risk of bladder cancer. (IARC 1991). However, I do not consider either to be a risk factor for Mr. Criswell as he had no known exposure to well water contaminated with high levels of arsenic or a documented history of chlorinated water consumption.

3. History of bladder infections;

I do not consider this to be a risk factor for Mr. Criswell as he had no history of bladder infections prior to his diagnosis of bladder cancer.

4. Long use of urinary catheters;

⁶⁵ Yu *et al*, Family history and risk of bladder cancer: an analysis accounting for first- and second-degree relatives (2022), available at <https://aacrjournals.org/cancerpreventionresearch/article/15/5/319/694470/Family-History-and-Risk-of-Bladder-Cancer-An>

⁶⁶ 01482_CRISWELL_0000000433

⁶⁷ "Cyclophosphamide increased bladder and hematologic cancer risk". See Lim W, Moon S, Lee NR, Shin HG, Yu SY, Lee JE, Kim I, Ko KP, Park SK. Group I pharmaceuticals of IARC and associated cancer risks: systematic review and meta-analysis. *Sci Rep.* 2024 Jan 3;14(1):413. doi: 10.1038/s41598-023-50602-6. PMID: 38172159; PMCID: PMC10764325

⁶⁸ See 00897_PLG_0000083518 - Bladder Cancer - Release 45 005 301-400_Redacted

I do not consider this to be a risk factor for Mr. Criswell as he had no history of using urinary catheters prior to his diagnosis of bladder cancer.

5. Demographic considerations:

Approximately 80% of all bladder cancers are diagnosed in individuals age 65 and older,⁶⁹ and the median age for bladder cancer is 73.⁷⁰ Mr. Criswell's bladder cancer diagnosis at age 42 makes him an outlier, and suggests an unusual set of circumstances leading to his diagnosis.

In light of the known risk factors for bladder cancer, I can rule out the following conditions: significant tobacco use, family history, history of bladder infections, long use of urinary catheters, demographic considerations, pelvic radiation, parasite infections.

IX. Opinions

1. It is at least as likely as not that Mr. Criswell's exposure to chemicals in the water at Camp Lejeune caused his bladder cancer

Applying the methodology of differential etiology, there are only two risk factors for Mr. Criswell to develop bladder cancer: his exposure to the water at Camp Lejeune and exposure to passive tobacco products. Mr. Criswell had extensive exposure to the water at Camp Lejeune as a resident living in Tarawa Terrace, working at Hadnot Point as an active marine. Mr. Criswell's approximately 800 days of exposure to the toxins at Camp Lejeune was substantial. Mr. Criswell was exposed to the toxins through the water, which was present in almost all aspects of his life. He was exposed through ingestion, inhalation, and dermal to four known carcinogens consistently for approximately 800 days.

In short, Mr. Criswell was substantially exposed to the chemicals at Camp Lejeune which are known to cause bladder cancer. As such, it is at least as likely as not that his bladder cancer diagnosis was caused by the water at Camp Lejeune.

2. There are no other causes for Mr. Criswell's bladder cancer that are more likely than not to cause his diagnosis

Mr. Criswell does not have any of the risk factors traditionally associated with bladder cancer. None of his immediate family relations have a history of bladder cancer, and he had no prior history of bladder infections or use of catheters prior to his diagnosis.

⁶⁹ Saginala et al., *Epidemiology of bladder cancer (2020)*, available at <https://pmc.ncbi.nlm.nih.gov/articles/PMC7151633>

⁷⁰ SEER*Explorer: An interactive website for SEER cancer statistics [Internet]. Surveillance Research Program, National Cancer Institute; 2024 Apr 17. [updated: 2024 Nov 5; cited 2025 Jan 23]. Available from: <https://seer.cancer.gov/statistics-network/explorer/>. Data source(s): SEER Incidence Data, November 2023 Submission (1975-2021),

He has no known exposure to well water. He was diagnosed over 30 years earlier than the median age for bladder cancer. Mr. Criswell does not have any discernable occupational exposure that would contribute to his bladder cancer diagnosis.

While smoking is a known cause of bladder cancer, Mr. Criswell's personal smoking history and exposure to second-hand smoke is so minor as to be medically insignificant. However, even considering his occasional cigarette use and exposure to second-hand smoke as a potential risk factor, there is nothing to suggest that this is an overriding risk factor than his exposure to the water at Camp Lejeune. Additionally, there is nothing remarkable or discernable concerning his childhood smoking and potential second-hand smoke exposure.

Accordingly, to the extent that smoking is a risk factor for Mr. Criswell (and I don't believe it is), it is certainly not more likely than not to have caused his bladder cancer.

X. Past Burden and Future Prognosis

Mr. Criswell's life has been permanently changed by his diagnosis of bladder cancer. He will need lifelong cancer surveillance involving doctor's visits, labs, imaging (most often CT scans), and cystoscopies. As time goes by, his recurrence rate does decrease, but it will not reach zero.

There is a tremendous physical burden with the surveillance and treatment of bladder cancer. Indeed, he was given BCG and his side effects were severe enough to prompt treatment with isoniazid. Shortly thereafter he suffered a heart attack. The stress of bladder cancer, the discomfort of treatment, and the known cardiotoxicity of isoniazid likely contributed to this event.

There is an incredible psychological burden associated with bladder cancer. He had multiple cystoscopies with unusual findings that were not pathologically malignant, but he had to suffer anxiety from the time of the office cystoscopy until he had surgery for a TURBT and the subsequent pathology report. As stated above, the recurrence rate will never be zero, and his own history speaks to this fact. He went roughly 17 years without a recurrence before another tumor was found.

It is well established that a cancer diagnosis is distressing. The National Comprehensive Cancer Network defines cancer distress as, "Distress is a multifactorial unpleasant experience of a psychological (ie, cognitive, behavioral, emotional), social, spiritual, and/or physical nature that may interfere with one's ability to cope effectively with cancer, its physical symptoms, and its treatment. Distress extends along a continuum, ranging from common normal feelings of vulnerability, sadness, and fears to problems that can become disabling, such as depression, anxiety, panic, social isolation, and existential and spiritual crisis."

Patients with a diagnosis of cancer can expect to experience: fear and worry about the future, concerns about illness, preoccupation with thoughts of death, sadness about

loss of health, anger, feeling out of control, poor sleep, poor appetite, poor concentration, concerns with treatment side effects, concerns about social role (eg, as parent), spiritual/existential concerns, financial worries.

Depression may manifest itself in many symptoms including but not limited to, pain, fatigue, sleep disruption, anorexia, anhedonia, decreased interest in activities, suicidal thoughts, mood swings, inability to concentrate, and anxiety.

Depression is the most common mental disease in bladder cancer patients. A meta-analysis also demonstrated that the disease attributable mortality of bladder cancer patients with depression was 2.2 times higher, which is the highest among all kinds of cancer, than that of bladder cancer patients without mental disorders.⁷¹ Depression affects patient's ability to cope with the burden of the illness decreasing the acceptance of treatment, extending hospitalization, and reducing quality of life. Another major psychological distress of patients with cancer is anxiety. 8.8% of bladder cancer survivors had major depressive disorder according to a national database study.⁷² The prevalence of depression in these study populations was high, ranging from 4.7% to 71.3% across all cancer stages and statuses, with variation depending on the geographic region and culture studied.⁷³ younger patients with muscle-invasive BC are more likely to be diagnosed with a post-treatment psychiatric disorder potentially because of the substantial morbidity and mortality of treatment.⁷⁴ Based on Mr. Criswell's medical records, Mr. Criswell has clearly (and not surprisingly) struggled with anxiety and depression related to his diagnosis of bladder cancer. Mr. Criswell has a long history of depression arising from his initial diagnosis and treatment that was exacerbated by his subsequent recurrence after nearly 17 years.

XI. Conclusion

In conclusion, Mr. Criswell was an active Marine with approximately 800 days of exposure to known bladder carcinogens in his drinking water while stationed at Camp Lejeune. His circumstances lead to a significant level of exposure to the contaminated water through ingestion, inhalation and dermal exposure. He had an insignificant smoking history that would classify him as a non-smoker, and no other known risk factors for bladder cancer. He developed bladder cancer and suffered from the known consequences of the disease and its treatments. As a result, based upon my education, training, and experience as a practicing urologic oncologist, and my prior research into bladder cancer and Camp Lejeune, and after applying a differential etiology, it is my

⁷¹ Wang Y.-H., Li J.-Q., Shi J.-F., et al. Depression and anxiety in relation to cancer incidence and mortality: a systematic review and meta-analysis of cohort studies. *Molecular Psychiatry*. 2020;25(7):1487–1499. doi: 10.1038/s41380-019-0595-x

⁷² Park B, Youn S, Yi K-K, Lee S-Y, Lee J-S, Chung S. The prevalence of depression among patients with the top ten most common cancers in South Korea. *Psychiatry Investig*. 2017;14:618-25.

⁷³ Vartolomei L., Ferro M., Mirone V., Shariat S.F., Vartolomei M.D. Systematic review: depression and anxiety prevalence in bladder cancer patients. *Bladder Cancer*. 2018;4:319–326. doi: 10.3233/BLC-180181.

⁷⁴ Jazzar U., Yong S., Klaassen Z. Impact of psychiatric illness on decreased survival in elderly patients with bladder cancer in the United States. *Cancer*. 2018;124:3127–3135. doi: 10.1002/cncr.31404

opinion to a reasonable degree of medical and scientific certainty that Mr. Criswell's exposure to contaminated water at Camp Lejeune is at least as likely as not a cause of his bladder cancer as any other possible cause.

Appendix 1

CURRICULUM VITAE

PERSONAL DATA

Name: **Thomas Andrew Longo**
Place/Date of birth: Omaha, NE, [REDACTED]/1982
Address: 3821 Ed Drive
Associated Urologists of North Carolina
Raleigh, NC 27612
Telephone: Cell: (402) 680-5846
Work: (919) 758-8677
Fax: (919) 758-8723
E-mail: tlongo@auncurology.com

EDUCATION

Undergraduate School	Columbia University in the City of New York New York, New York Bachelor of Arts	2001-2005
Medical School	University of Nebraska Medical Center Omaha, Nebraska Doctor of Medicine	2005-2009

POSTDOCTORAL TRAINING:

Research training	University of Nebraska Medical Center Omaha, Nebraska	2000
	Brigham and Women's Hospital Boston, Massachusetts	2006
	University of Nebraska Medical Center Omaha, Nebraska	2009-2013
	GU Research Network, LLC Omaha, Nebraska	2013
	Duke University Durham, North Carolina	2015-2017
Internship/Residency	University of Nebraska Medical Center	July 2009-June 2011

Omaha, Nebraska
General Surgery

Fellowship	University of Nebraska Medical Center Omaha, Nebraska Urology	July 2011-June 2015
	Duke University Durham, North Carolina Society of Urologic Oncology	July 2015-June 2017

Work Experience

Assistant Professor of Urology, Duke University July 2017-June 2023

Associated Urologists of North Carolina July 2023 – present

LICENSURE

State of Nebraska License

License #: 28526

State of North Carolina License

License #: 208232

Federal DEA License

License #: FL1488457

NPI: 1154559904

United States Medical Licensing Exam Step 1, June 2007

United States Medical Licensing Exam Step 2 Clinical Knowledge, August 2008

United States Medical Licensing Exam Step 2 Clinical Skills, March 2009

United States Medical Licensing Exam Step 3, September 2010

American Board of Urology, Qualifying Exam, July 2015

Society of Urologic Oncology 06/01/2017

American Board of Urology 02/28/2019

HONORS & AWARDS

Adele Kalmansohn Scholarship 2005-2006

Vascular Society Student Lifeline Fellowship, Summer 2006

Society for Translational Oncology Fellows' Forum, Fall 2015

AUA Early Career Investigators Workshop, Fall 2016

MEMBERSHIPS AND PROFESSIONAL SOCIETIES

American College of Surgeons 2009-2010
American Urological Association 2010-present

Grants

“Identification of carcinogen-induced mutational signatures in human and canine bladder cancer” Consortium for Canine Comparative Oncology Pilot Projects \$100,000

“Duke-Africa Prostate study: Alternative Splicing” Cancer Control Pilot Studies Program \$25,000

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