Exhibit 349

Specific Causation Expert Report for Terry Dyer
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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 with regard to its citizens for the sake of their health.

II. Mandate

I have been asked to provide my opinion on the causation of Terry Dyer'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 Ms. Dyer, 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 Ms. Dyer'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 Terry Dyer 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 Ms. Dyer'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 Ms. Dyer'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. Terry Dyer's Factual Background

1. Ms. Dyer's Exposure at Camp Lejeune

Ms. Dyer moved to Camp Lejeune and lived at Tarawa Terrace as a child when she was a year and a half old, starting in 1958 until January 1973, when she was in high school.[1] Records reflect that Ms. Dyer and her family lived in numerous locations within the Tarawa Terrace area of the base.[2] Ms. Dyer's father served as the principal at the Tarawa Terrace II Elementary School and then later at Berkeley Manor School, both of which I understand are physically located on base.[3] Ms. Dyer's family did move to a home off base (while Ms. Dyer still attended school on base) for approximately one year but then moved back on base at Tarawa Terrace.[4] Ms. Dyer attended TT2 elementary school, Brewster Junior High School and Lejeune High School for two years, all of which were located on the base[5]. Subsequently, Ms. Dyer moved off base for her junior and senior years of high school, starting in January 1973. Even when Mrs. Dyer was not living on base, her social life regularly brought her onto the base where she continued to drink the water, visit friends and socialize.[6] All told, Ms. Dyer spent over approximately 5,000 days directly exposed to the water at Camp Lejeune.

Ms. Dyer was exposed to contaminated water throughout her childhood through all means of exposure, including dermal, inhalation and ingestion. Ms. Dyer's residential exposure was significant, with normal everyday activities such as drinking, bathing, cooking, washing food and dishes, among other things, providing exposure to contamination via ingestion, inhalation, and through dermal absorption. Ms. Dyer reports having "lived on tea and water" as well as mixing water with Kool-Aid.[7] [8] Ms. Dyer's exposure was not limited to her residence. She reports having an active childhood where she traveled all over the base where she would have ingested water.

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[1] Dyer Dep. 23:1-20; 27:6-11.
[2] Dyer Dep. 23:1-23:25, 57:7-58:3; CLJA_Housing-0000149771; CLJA_Housing-0000175670
[3] Dyer Dep. 23:1-6, 24:10-23; 00357_DYER_DPPF_0000000002
[4] Dyer Dep. 57:7-58:14.
[5] Dyer Dep. 116:5-13
[6] Dyer Dep. 58:11-21.
[7] Dyer Dep. 156:9-13
[8] Dyer Dep. 156:18-20, 157:25-158:2

Ms. Dyer reports being babysat at day-care and night-care centers at Tarawa Terrace,[9] regularly visiting on-base restaurants, being bused around to numerous activities, swimming in on-base pools, and participating in other on-base activities.[10]

Ms. Dyer was unable to escape exposure to cancer-causing carcinogens, as the contamination was present at her residence, her school, and where she played as a child and visited socially. The levels of PCE that Ms. Dyer was exposed to, as stated above, were of a sufficient level to cause bladder cancer. In particular, Ms. Dyer's PCE exposure is consistent with the PCE exposures seen in Aschengrau 1993 that showed an increase in bladder cancer diagnoses and are reflected in numerous Camp Lejeune studies (Bove 2018 morbidity study; Bove 2024 cancer incidence study). As such, Ms. Dyer's exposure to chemicals in the water at Camp Lejeune is more than a de minimis exposure – it was substantial.

2. Ms. Dyer's Occupational History

Before her diagnosis of bladder cancer, Ms. Dyer worked in various capacities, including as a personal manicurist, a school teacher, security guard, and a teller at a bank.[11] According to Ms. Dyer's recollections, she obtained a nail license and worked as an occasional manicurist from sometime in the 1980s to the early 1990s.[12] By Ms. Dyer's account, the amount of her work as a nail technician was minimal: she had two clients and assisted her sister on occasion[13], and she denies applying acetone to remove nail polish.[14] For the purposes of this report, there is nothing notable about Ms. Dyer's employment as a bank teller in the 1980s and early 1990s, or as an elementary school teacher in the 2000s[15]. Ms. Dyer worked for Corning, Inc., an optical fiber manufacturing company, as a security guard, "for a couple years." [16] As a security guard, Ms. Dyer appears not to have any direct contact with chemicals at the plant as she was tasked with walking around and monitoring the facility, driving a golf cart around the exterior of the buildings, and ensuring doors were locked.[17] Ms. Dyer does report a fire that occurred at Corning in approximately 2010[18], but it is unlikely that this fire resulted in any chemical exposure to Ms. Dyer. According to Ms. Dyer, she was positioned "pretty far away" in a security building near a back entrance and directed firefighters in through the gate to the fire.[19]

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[9] Dyer Dep. 87:8-12
[10] Dyer Dep.87:6-88:12.
[11] Dyer Dep. 89:2-7, 98:3-4, 234:2
[12] Dyer Dep. 89:2-91:14
[13] Dyer Dep. 92:9-14
[14] Dyer Dep. 90:16-24
[15] Dyer Dep. 97:25-98:7
[16] Dyer Dep. 96:23-97:13
[17] Dyer Dep. 100:5-9
[18] Dyer Dep. 141:17-24
[19] Dyer Dep. 142:21-143:10

3. Ms. Dyer's Medical History

Ms. Dyer presented to Dr. Boldizar on April 22, 2009, with complaints of side pain.[20] She was referred to a urologist, Dr. Lovett, after blood was detected in her urine.[21] Dr. Lovett performed a cystoscopy on May 12, 2009, and discovered a 5mm right anterior bladder wall tumor.[22] At this time, Ms. Dyer was 52 years old. As discussed in some detail in Dr. Culp's general causation report, Ms. Dyer's diagnosis was younger than the average bladder cancer patient by nearly 2 decades at the time of diagnosis. Dr. Lovett recommended performing a transurethral resection of bladder tumor (TURBT), which was performed on May 20, 2009[23]. A TURBT involves inserting an instrument into the urethra and bladder and cutting and cauterizing bladder tumors off the inner lumen of the bladder. This procedure is standard but can lead to infection, bleeding, stricture, bladder perforation, and dissemination of the cancer. The pathology revealed focal papillary urothelium with mild atypia, suspicious for low-grade urothelial carcinoma.[24]

A cystoscopy was performed on July 17, 2009, by Dr. Lovett[25] and Ms. Dyer returned to Dr. Lovett on July 20, 2009 with continued complaints of bladder pain and lower urinary tract symptoms.[26] Dr. Lovett performed a second TURBT. The pathology on the second resection of Ms. Dyer's bladder revealed high grade, T1 tumor and carcinoma in situ (CIS)[27]. High grade bladder cancer is more aggressive than low grade. T1 indicates that it had invaded into the lamina propria, a layer just above the muscle layer. CIS is carcinoma in situ, which is a high-grade bladder cancer, that although superficial, does have the potential to become metastatic.

The time to recurrence is a known prognostic risk factor for bladder cancer and is included in risk calculations.[28] Recurrence at 3 months was the most important predictor of progression[29]

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[20] Boldizar Dep. 19:6-12
[21] 00357_DYER_0000001875
[22] 00357_DYER_0000001876
[23] 00357_DYER_000000375-76
[24] 00357_DYER_000000380
[25] 00357_DYER_UASNC_0000000087; 00357_DYER_0000001866-00357_DYER_0000001867
[26] 00357_DYER_000001866
[27] 00357_DYER_0000000385-86
[28] Sylvester, et al. Predicting recurrence and progression in individual patients with stage Ta T1 bladder cancer using EORTC risk tables: a combined analysis of 2596 patients from seven EORTC trials. Eur Urol, 2006. 49: 466.
[29] Palou, J., et al. Recurrence at three months and high-grade recurrence as prognostic factor of progression in multivariate analysis of T1G2 bladder tumors. Urology, 2009. 73: 1313.

In the face of Ms. Dyer's "rapidly advancing urothelial carcinoma of the bladder", Dr. Lovett performed a third TUBRT on August 1, 2009;[30] the pathology revealed "no evidence of invasive carcinoma within either the lamina or muscularis propria."[31]

The treatment of choice for non-muscle invasive bladder cancer, which Ms. Dyer had, is instillation of intravesical Bacillus Calmette-Guerin (BCG). 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. At 1 year, 80% of patients who have received BCG are cancer free, and nearly 2/3rds remain cancer free at 5 years.

Many BCG treatment protocols exist that are acceptable within the standard of care, 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 therapy.

Ms. Dyer had an induction course of BCG starting on September 14, 2009[32], and reported her first fever within 3 weeks. Medical notes regarding subsequent visits evidence various symptoms: fever, blood in urine, dysuria (requiring narcotics), frequency and urgency. Ms. Dyer completed her BCG course on October 19, 2009, with instructions to return for further cystoscopy and biopsy "given CIS history[.]"[33] She returned for her fourth TURBT on November 11, 2009, and the pathology was "negative re residual urothelial carcinoma."[34] Although the pathology report did not demonstrate a malignant recurrence, it did show significant changes in the bladder. Ms. Dyer's bladder was undergoing severe changes (albeit expected changes for her diagnosis) from the resection and treatment of her bladder cancer. These changes were ongoing months after her original diagnosis and would eventually lead to her bladder becoming unsalvageable.

Ms. Dyer received a second round of BCG treatment, beginning on December 17, 2009[35]. During this second course of BCG treatment Ms. Dyer reported severe pain

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[31] 00357_DYER_000000394
[32] 00357_DYER_000001847
[33] 00357_DYER_000001837
[34] 00357_DYER_000000407
[35] 00357_DYER_0000001923-24

that required a prescription of pain medication.[36] In her third week of this second cycle Ms. Dyer reported symptoms of cystitis.[37] Cystitis in the acute setting includes dysuria (painful urination), frequency, urgency, fever, hematuria, fatigue, malaise, and suprapubic discomfort. This can progress to a chronic state. BCG cystitis has rare side effects that can include myelosuppression, ureteral reflux, ureteral obstruction, and bladder contraction.

During a February 17, 2010 exam, Dr. Lovett performed a cystoscopy and noted "there is a distinct red, slightly raised patch in the dome."[38] Discovery of a slightly raised red patch of the dome of the bladder presents a diagnostic dilemma to a treating urologist. This may be the appearance of treatment effect from BCG. A slightly raised red patch is also the appearance of CIS, a form of high-grade bladder cancer with metastatic potential.

Following her second cycle of BCG treatment, Ms. Dyer received her fifth TURBT on March 6, 2010, and the pathology revealed "no definite residual malignancy."[39] However, Ms. Dyer required a blood transfusion due to the blood loss following her fifth TURBT.[40] Following a maintenance round of BCG, Ms. Dyer underwent her sixth and final TURBT on May 25, 2010.[41] The pathology of her sixth TURBT was "negative dysplasia or malignancy."[42]

It is helpful to explain the effects of BCG on the human body. 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. Ms. Dyer's record is replete with many of these expected symptoms. More significant side effects include 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.

Ms. Dyer's symptoms progressed to where on June 8, 2010, Dr. Lovett diagnosed her with adult Hinman Syndrome.[43] Hinman Syndrome is a nonneurogenic neurogenic bladder syndrome first described in children. The term is used in adults with bladder dysfunction characterized by bladder outlet obstruction that mimics neurogenic disease without evidence of neurologic damage. It is a poorly compliant, atonic bladder that will have a small capacity. The patient is unable to recognize the high pressure or is unable to urinate leading to vesico-ureteral reflux. This means that

[36] 00357_DYER_000001921
[37] 00357_DYER_000001918
[38] 00357_DYER_000001917
[39] 00357_DYER_000000422
[40] 00357_DYER_000000414
[41] 00357_DYER_000001897
[42] 00357_DYER_000000428
[43] 00357_DYER_000001894

the pressure in the bladder is so great that the urine flows up the ureters to the kidneys. On imaging, this will be seen as hydronephrosis, or a dilated collecting system from the calyces in the kidneys and the ureters. This chronic pressure on the kidneys can lead to renal function deterioration. In fact, this is precisely what happened to Ms. Dyer, as Dr. Inman would later note acute renal insufficiency.[44]

Ms. Dyer's deterioration unfortunately progressed. On June 16, 2010, Ms. Dyer consulted with Dr. Lovett who recommended she obtain a second opinion on whether to have a cystectomy to address her blader function and eliminate long-term risk of her aggressive bladder cancer from re-emerging.[45] Dr. Lovett recognized that she was "understandably anxious" and "very concerned about impact of her disease on her employment."[46] Regarding a potential cystectomy, Dr. Lovett noted that it "would eliminate long term risk of recurrence of her aggressive bladder cancer and would provide relief of her bladder dysfunction."[47]

Ms. Dyer became a bladder cripple, as documented in Dr. Lovett's note[48] and later on in Dr. Inman's note.[49] A bladder cripple in the simplest of terms is the bladder can no longer serve in its role as a storage vessel for urine. It is a constellation of symptoms including pelvic pain, overactive bladder, incontinence, poor compliance, small capacity, recurrent infections, urinary retention, etc.

Upon his initial consult, Dr. Inman diagnosed Ms. Dyer with BCG cystitis.[50] Ms. Dyer's symptoms of BCG cystitis included pain, hematuria, fevers, chills, incontinence, and pathology consistent with BCG cystitis. She was found to have a severely contracted bladder leading to hydronephrosis. While not identifying a present need for a cystectomy, Dr. Inman recognized she may require one "simply for palliative purposes."[51]

Ms. Dyer had bilateral ureteral stent placement due to the appearance of hydronephrosis on imaging and acute renal insufficiency.[52] As described above, hydronephrosis a dilated collecting system from the calyces in the kidneys and the ureters. Rarely, it could be congenital, but this was not the case in Ms. Dyer as it was not present on prior imaging. It is caused by one of two broad categories, either reflux (urine passing from the bladder up to the kidney) causing dilation, or obstruction causing dilation. There are various ways to evaluate the cause, but stent placement could be the most definitive. If it is caused by obstruction, the stents would unblock the obstruction, and the hydronephrosis would resolve. Although stent placement is

[44] 00357_DYER_000000904
[45] 00357_DYER_000001893
[46] 00357_DYER_000001893
[47] 00357_DYER_000001893
[48] 00357_DYER_000001894-96
[49] 00357_DYER_000001049-51
[50] 00357_DYER_000000789
[51] 00357_DYER_000000789
[52] 00357_DYER_000000904

generally a simple and well tolerated procedure, it is not without burden. Most commonly, people experience symptoms from the stent including, but not limited to stent pain, urgency, frequency, and hematuria.

Ultimately, Ms. Dyer underwent radical cystectomy, which is the removal of her bladder. A radical cystectomy carries a 66% complication rate and an over 5% mortality. All of the diagnosis and therapies involve the genitalia, and this directly impacts the patient's physical intimacy through pain, embarrassment, and loss of function. A radical cystectomy with ileal conduit is considered major surgery. In a woman, the bladder, urethra, uterus, ovaries, fallopian tubes, the anterior strip of the vagina, and pelvic lymph nodes would be removed. The defect in the vagina is then sewn shut. The ureters coming from the kidneys have then been detached from the bladder, and the urine must be brought outside the body. This reconstruction portion of the operation is most commonly performed using a piece of the small bowel called the ileum to form an ileal conduit. Approximately 15 cm of the ileum is isolated from the rest of the bowel, and then bowel continuity has to be reestablished. The ureters are then sewn to one end of this portion of bowel. The other portion is brought through the abdominal wall to the skin surface where it is sewn in place forming an opening called a stoma.

This surgery has many risks which include but are not limited to bleeding, infection, adjacent organ injury, renal dysfunction, stricture formation, urine leaks, gastrointestinal tract dysfunction (bowel stricture, bowel leak), thromboembolism, myocardial infarction and death. The ileal conduit alone has long term sequelae such as stricture formation, stones, UTIs, hernias, and other metabolic issues.

Ms. Dyer's cystectomy included a large blood loss and required an ICU stay to properly care for her. Following her recovery, she developed a parastomal hernia. This is a weaking in the strength layer of her abdominal wall around the stoma of her ileal conduit. This hernia allows her abdominal contents (her bowels) to push out from her abdomen and are held back by only her skin. Most commonly, this bulge makes it difficult to get a stoma appliance (the bag to collect her urine) to properly fit and not leak. It could lead to more severe problems including an incarcerated hernia, or piece of bowel that gets so trapped that it cannot get blood supply.

A patient with an ileal conduit will now have a bag of urine attached outside their abdomen at all times. This appliance consists of a sticker that typically lasts 3-7 days, but may need to be changed more or less frequently depending upon the patient and the environmental conditions. If a patient does not have a flat location for the stoma, the appliance will leak at the site of a bulge or a fold. If the patient has excessively dry skin, or conversely, if they sweat a lot, the appliance will not achieve a good seal and will have to be changed more frequently. If the opening to the appliance is too large, skin will be exposed to urine, and this will lead to breakdown of the skin and wounds. The bags hold a standard bladder capacity and must be emptied 4-6 times a day depending upon the volume of liquid a person consumes.

Patients with ileal conduits will no longer have sterile urine; their urine will be colonized with bacteria. The bacteria has a direct route from the skin surface through the conduit, up the ureters and into the kidneys. Typically, this is a non-harmful colonization with bacteria, but it can lead to infection. The risk of renal dysfunction increases over time, and kidney function will have to be monitored. These patients typically have a minor degree of a metabolic disorder called hyperchloremic metabolic acidosis. Rarely is it severe enough that they may experience symptoms of fatigue, anorexia, weight loss, and lethargy. This acidosis can predispose patients to stone formation. This portion of bowel is responsible for vitamin B12 absorption. Over time these patients may also require vitamin B12 replacement; B12 deficiency can lead to anemia.

Following recovery from her cystectomy, Ms. Dyer recovered but experienced numerous trying health challenges. She struggled to care for her stoma and handling ostomy pouch changes.[53] Developing an ulcer near the stoma site is a common occurrence, and will likely happen to every patient with an ileal conduit at some point. It occurs when the skin is chronically exposed to urine; it is analogous to a diaper rash. Part of stoma care involves have a stoma appliance – the adhesive wafer that goes directly onto the patient's abdominal skin – fit properly. The opening has to allow the stoma itself freely protrude without being constrictive. At the same time, it must not allow any skin to be exposed. This can be especially problematic as a stomal hernia protrudes and alters the area around the conduit making it difficult to achieve a good fit without any skin exposure. Additional stoma care is required to allow it to heal, and it can occur again in the future. Ms. Dyer experienced significant weight gain, making adhesion of the ileal bag difficult,[54] and endured several leakages.[55] She developed a parastomal hernia, a common post-cystectomy complication, that caused significant pain.[56] Ms. Dyer lived with her parastomal hernia until May 24, 2023, when she had it repaired (although a repeat CT scan showed recurrence of the hernia).[57] Following her parastomal hernia surgery, Ms. Dyer was admitted to the ICU for a suspected hemorrhage and received a blood transfusion.[58] Understandably, Ms. Dyer complained of severe depression,[59] which as described below is a pervasive and well-documented issue with bladder cancer patients. Ms. Dyer was also diagnosed with several UTIs, which are common occurrences following a cystectomy with ileal conduit.[60] One of Ms. Dyer's treatment providers counseled her to expect recurrent UTIs due to her ileostomy: "she is very likely to have recurrent UTI due to ileostomy and the fact that her normal, natural barriers to UTI are no longer intact. Advised that recurrent UTI are unfortunately almost assured."[61]Another recurring issue for Ms. Dyer, common for individuals post-

[53] 00357_DYER_0000001629; 1631
[54] 00357_DYER_0000001677
[55] 00357_DYER_0000001693-96
[56] 00357_DYER_0000001677 - 00357_DYER_0000001680
[57] 00357_DYER_0000002298 - 00357_DYER_0000002375
[58] 00357_DYER_0000002298 - 00357_DYER_0000002375
[59] 00357_DYER_0000001677-79
[60] 00357_DYER_0000001677-80, 1693-96; 1703-04; 29-37; 53-60; 123-133
[61] 00357_DYER_0000000562

cystectomy, was the development of renal stones which were found following the presence of blood in her urine bag.[62]

In sum, Ms. Dyer's bladder cancer, and subsequent treatment, has led to severe pain, distress and burden. Ms. Dyer was in the urology office approximately 50 times, treated for a UTI 40 times, underwent three cystoscopies and six TURBTs. Ms. Dyer had three unplanned hospital admissions: once for a blood transfusion, once for a urinary obstruction, and once for an abscess. Ultimately, she required a cystectomy. She had three blood transfusions for a total of 10 units. She developed a parastomal hernia, painfully lived with it for over a decade, and underwent surgery to address it. The surgery to repair her parastomal hernia was also complicated. It began laparoscopic and was converted to an open case due to the level of difficulty. She had bleeding issues requiring a blood transfusion and stay in the intensive care unit. Perhaps most frustratingly, her parastomal hernia redeveloped prior to her discharge from the hospital. Her diagnosis, treatment, and follow up care involved onerous and painful surveillance.

The burden of disease involves almost every aspect of the patient's life, as they are a sick person, not just a sickness. In following Ms. Dyer's medical record after her diagnosis, it is clear that her cancer resulted in multiple burdens including, but not limited to: time, cost, anxiety, pain, infection, and body alterations. Bladder cancer, from diagnosis, and through treatment and beyond is often painful, and can bring about an undesirable physical and mental health effects beyond the pain and stress of treatment. The diagnosis and surveillance involve routine visits with cystoscopy where a camera is inserted via the urethra into the bladder several times a year for surveillance. The medical care for one individual can impact and place demands upon others. Many patients require the direct medical care or assistance from their loved ones or others. Many patients may be understandably unwilling or resistant to live with an ostomy (a piece of bowel draining urine to a bag worn on the abdomen). Although no one would volunteer to live in this condition, Ms. Dyer was willing to live with an ostomy in exchange for relief from her crippling bladder condition. While Ms. Dyer is now bladder cancer free, there are ongoing health impacts that are permanent.

It has been well established that bladder cancer is the most expensive malignancy over the lifetime of the patient.[63] In part, this is related to the high recurrence rate meaning strict surveillance is necessary to catch the disease while still contained within the bladder. Once the cancer progresses from non-muscle invasive to muscle invasive, the costs only increase; diagnosis related costs are five times non muscle invasive bladder cancer as for muscle invasive bladder cancer, and the treatment cost is three times non muscle invasive bladder cancer as for muscle invasive bladder cancer. Upon my review, Ms. Dyer's treatment for her bladder cancer and her subsequent care were medically necessary. The health issues such as recurring UTIs that Ms. Dyer experienced after removal of her bladder are common and her treatment

[62] 00357_DYER_GSR_0000000094 - 00357_DYER_GSR_0000000098
[63] Messing EM. Financial Toxicity of Having Bladder Cancer. Bladder Cancer. 2018 Jul 30;4(3):351-352.

for those conditions also was medically necessary. Ms. Dyer will continue to have complications related to her bladder cancer for the rest of her life, and will continue to need assistance from her husband or a skilled nurse with respect to use of urinary supplies and placement of her urostomy pouch. She is at low to moderate risk for developing kidney issues as a result of her bladder cancer and related treatment, and the risk of cancer recurrence while small, is still present. I reviewed Ms. Dyer's medical records and expenses and found the treatment to be medically necessary and the expenses are fair and reasonable.

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 Ms. Dyer. 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.[64] Thus, Ms. Dyer's actual exposure is higher than what Dr. Reynolds shows, but nonetheless she suffered from substantial exposure to the chemicals at Camp Lejeune.

Dr. Reynolds composed the total days of Ms. Dyer's exposure to the Camp Lejeune water at 5,376 days of exposure. From ATSDR's estimated consumption of water Dr. Reynolds calculated the following total concentrations that Ms. Dyer was exposed to on Camp Lejeune:

[64] 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.

Summed variable totals

	Cumulative ug/l-M	Chart 1: 1L Cumulative consumption (total ug= days*concentration per L)	Chart 2: ATSDR RME Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Chart 3: ATSDR CTE Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)	Chart 4: ATSDR RME; deposition ingestion age 6+ Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Hadnot Point					
TCE	3,608	26,042	40,097	13,871	93,536
PCE	-	-	14,049	4,772	35,403
VC	10	66	-	-	-
BZ	161	1,157	40,097	13,871	93,536
Tarawa Terrace					
TCE	317	7,258	3,783	3,783	24,544
PCE (TechFlowMP Model)	7,518	172,268	90,529	90,529	595,062
PCE (MT3DMS Model)	9,236	211,660	110,277	110,277	715,375
VC	612	14,050	6,948	6,948	41,177
BZ	-	-	-	-	-
Totals HP & TT					
TCE	3,925	33,300	50,956	17,655	118,080
PCE (TechFlowMP Model)	7,518	172,268	274,503	95,301	630,465
PCE (MT3DMS Model)	9,236	211,660	330,565	115,049	750,778
VC	622	14,116	19,632	6,948	41,177
BZ	161	1,157	40,097	13,871	93,536

I have also reviewed the reports of Dr. Hatten and Dr. 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 Ms. Dyer's exposure was significant and substantial. Ms. Dyer exceeds each of the demonstrated levels set out here.

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.

VII. General Causation

Before advancing to the application of a differential etiology for Ms. Dyer, 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…"[65] EPA concluded that "TCE is carcinogenic to humans by all routes of exposure," that is, by ingestion, inhalation,

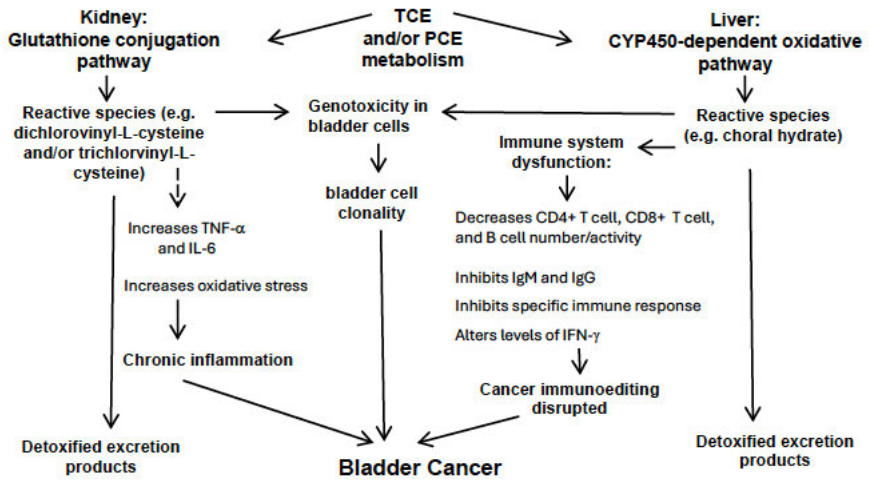
[65] International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;106:1-514

and dermal exposure.[66] Further, EPA concluded that PCE is "likely to be carcinogenic in humans by all routes of exposure."[67] Similarly, the National Toxicology Program has recognized TCE as "known to be a human carcinogen"[68] and PCE as "reasonably anticipated to be a human carcinogen."[69]. 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.[70]

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.[71] Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well.[72] 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.

[66] Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011
[67] Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012
[68] National Toxicology Program (NTP). 2015. Report on Carcinogens monograph on trichloroethylene. Research Triangle Park, NC: National Toxicology Program. RoC Monograph 05
[69] 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
[70] Hatten pp. 26-29; 31-32; Plunkett ¶ 47
[71] Plunkett ¶¶ 33, 43; Hatten p. 39; Bird pp. 17-18
[72] 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).[73] 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.[74] 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.[75]

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."[76] 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.

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[73] Gilbert p. 30
[74] Gilbert p. 32-3
[75] Gilbert p. 19-20
[76] 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 Ms. Dyer's exposure to the water at Camp Lejeune as likely as not caused her 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 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, 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."[77]. These risk factors act in concert with one another and have additive effects.

Before addressing the potential risk factors for bladder cancer, it is important to address whether the cause of bladder cancer is idiopathic. In two of the depositions of Ms. Dyer's treatment providers – Dr. Lovett and Dr. McCarthy – the government asked whether the cause of bladder cancer is idiopathic.[78] When a disease is idiopathic it means that the cause of the disease is unknown. Essentially, an idiopathic designation is one of exclusion. In my opinion, bladder cancer is rarely idiopathic in the sense that it is likely to have a known cause as bladder cancer is typically related to toxic exposures. In situations where an individual was exposed to a known cause of bladder cancer, considering whether the cause is idiopathic is inapplicable. As I describe in this report, I do not consider Ms. Dyer's bladder cancer as idiopathic because she has been exposed to a known and widely-accepted cause of bladder cancer.

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[77] https://www.cancer.org/content/dam/CRC/PDF/Public/8558.00.pdf
[78] Lovett Dep. 37:5-10; McCarthy Dep. 40: 22-41:5.

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 other toxic chemicals

There is a substantial body of evidence establishing that the chemicals present at Camp Lejeune are carcinogenic and cause blader 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. In addition, I and others in my field find it appropriate to review epidemiological literature incorporated by these organizations and following the publication of these organizations conclusions in order to stay up to date.

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.[79] 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 noted that the bladder in particular “has been identified as a target organ” for PCE.[80] 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 study 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).

[79] Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012
[80] IARC 2014

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)). 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. Since IARC's publication, additional studies have seen elevated associations with TCE and bladder cancer, including Hadkhale (2017) and Sciannameo (2019).

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% higher 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 cohort[81] 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 industry[82], rubber manufacturing industry[83], painting industry[84] and aluminum and refined products industry have been shown to have an association with bladder cancer. However, Ms. Dyer does not have any known occupational or environmental exposures – besides those present at Camp Lejeune – that could cause bladder cancer. Ms. Dyer has had several occupations – as a nail technician (though she did not work with acetone), as a security guard, as a teacher, and as a bank teller – but nothing stands out as an occupational exposure that would cause or contribute to causing bladder

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[81] Bove 2024 ended its data collection in December 2017.
[82] Singh and Chadha, Textile industry and occupational cancer (2016), *available at https://pmc.ncbi.nlm.nih.gov/articles/PMC4986180/*
[83] IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, No. 100F (2012), *available at* https://www.ncbi.nlm.nih.gov/books/NBK304412/
[84] 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.

cancer. I note that Ms. Dyer testified that as a security guard she witnessed a fire at her job but it is unclear whether it was a chemical fire; at any rate, Ms. Dyer's post was located at a back gate away from where the fire was located.

I do consider Ms. Dyer's exposure to chemicals in the water at Camp Lejeune to be a risk factor, but I see no evidence to suggest that she had any occupational exposure that would cause that to be a risk factor.

2. Tobacco use

Smoking is a known risk factor for bladder cancer, with some estimates attributing approximately 50% of all bladder cancers to smoking.[85] Here, however, I do not consider smoking to be a potential cause of Ms. Dyer's bladder cancer. There are some discrepancies in the records regarding Ms. Dyer's smoking history; however, even if the discrepancies are factored at the highest level, Ms. Dyer's limited smoking history is medically insignificant. In her deposition Ms. Dyer recounted an insignificant smoking history: Ms. Dyer reports that she might have a cigarette at a nightclub when she was dating her husband.[86] Mr. Dyer himself described her as a "casual" smoker when they dated but stopped when she got pregnant with their first child.[87] Her first child was born in [redacted] 1978, placing her cessation date approximately toward the end of 1977. Ms. Dyer's medical records are inconclusive on a precise amount of her cigarette exposure. It is noted in Ms. Dyer's medical records that she smoked 2 cigarettes a day for approximately two years while in other places it says .5 per day for two years and .25 packs for one year. A patient's medical history is sometimes inaccurate and can be re-inserted and repeated even when not medically relevant. In Ms. Dyer's case, she had ceased smoking for decades before her bladder cancer diagnosis and her limited distant history with smoking is not significant.

Ms. Dyer does not have any significant second-hand smoking exposure: she reports that her father either didn't smoke or smoked a pipe outside of the house, and her husband quit smoking soon after they met after she would "tear up his cigarettes" and then charge him $25 each time she caught him smoking.[88] Ms. Dyer's husband confirmed that he did not smoke inside the house, that he occasionally smoked inside the car, and that he was relegated to smoking (if at all) to the porch.[89] Mr. Dyer also noted that his mother remarried at the time he and Ms. Dyer dated, and that her husband was a smoker; however, Mr. Dyer reports that this individual smoked outside.[90]

Ms. Dyer's exposure to cigarette smoke is so minimal as to describe her as a never-smoker or a less than one pack a day smoker for approximately 2 years with an over 30-year history of smoking cessation. The Centers for Disease Control defines a

[85] https://www.nih.gov/news-events/nih-research-matters/smoking-bladder-cancer
[86] Dyer Dep. 163:17-24
[87] John Dyer Dep. 96:7-10
[88] Dyer Dep. 177:25-178:25
[89] John Dyer Dep. 49:3-14
[90] John Dyer Dep. 56:17-23

"nonsmoker" as "[a]n adult who has never smoked, or who has smoked less than 100 cigarettes in his or her lifetime", which fits Ms. Dyer's description of her smoking habits although reliance on her medical records would place her outside this definition.[91] Nevertheless, there is substantial evidence that smoking cessation reduces the risk of developing bladder cancer: IARC 2012 noted a 60% reduction of risk after 25 years of smoking cessation, although the risk was still higher than for never-smokers. IARC recognized that the duration and intensity of smoking, in addition to cessation, impacts the development of bladder cancer, and it appears that Ms. Dyer had a low duration and low intensity. Taking a worst case scenario of her history – that she only smoked two cigarettes a day for two years, approximately 30 years prior to diagnosis – such a history characterizes her as a non-smoker for medical purposes. At the very least, and to a reasonable degree of medical certainty, smoking is highly unlikely to be a cause of Ms. Dyer's bladder cancer. Nevertheless, it does present a potential risk factor.

3. Family history of bladder cancer

While there have been positive associations between a family history of bladder cancer in first- and second-degree relatives[92], I do not consider this to be a risk factor for Ms. Dyer. Ms. Dyer has no family history of bladder cancer.[93]

4. Past treatment with some anticancer drugs

Some anti-cancer drugs, cyclophosphamide[94] and ifosfamide[95] have been associated with an increase in risk for bladder cancer. However, I do not consider this to be a risk factor for Ms. Dyer as she had no past treatment involving these anticancer drugs prior to being diagnosed with bladder cancer.

5. 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

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[91] https://archive.cdc.gov/www_cdc_gov/nchs/nhis/tobacco/tobacco_glossary.htm
[92] 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*
[93] 0357_DYER_0000001694.
[94] "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
[95] *See* 00897_PLG_0000083518 - Bladder Cancer - Release 45 005 301-400_Redacted

with chlorine also increases the risk of bladder cancer.[96] However, I do not consider either to be a risk factor for Ms. Dyer as she had no known exposure to well water contaminated with high levels of arsenic or a documented history of chlorinated water consumption.

Polyfluoroalkyl substances (PFAS) are referred to as forever compounds. They are increasingly being found throughout the environment and are being investigated for linkages to cancer. To date, there is little evidence to suggest that they lead to an increased incidence of bladder cancer. Although there are studies of PFAS and cancer in rodents, there is yet to be epidemiological data supporting carcinogenicity in humans[97] I understand Ms. Dyer has asserted that her drinking water in Wilmington may have been exposed to PFAS for a period of time. Given the lack of sound scientific evidence linking PFAS to bladder cancer and the lack of evidence linking any amount of PFAS in Ms. Dyer's water supply to any cancer, much less bladder cancer, I do not consider this to be a risk factor for Ms. Dyer.

6. History of bladder infections

Research has suggested that a history of recurrent bladder infections may heighten future risk of bladder cancer[98] I do not consider this to be a risk factor for Ms. Dyer as she had no history of bladder infections prior to her diagnosis of bladder cancer.

7. Long use of urinary catheters

Bladder cancer risk is heightened by chronic catheterization. [99] I do not consider this to be a risk factor for Ms. Dyer as she had no history of using urinary catheters prior to her diagnosis of bladder cancer.

8. Radiation to pelvic area

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[96] IARC (1991) Chlorinated Drinking-Water; Chlorination By-Products; Some Other Halogenated Compounds; Cobalt and Cobalt Compounds. Lyon, France: International Agency for Research on Cancer

[97] Steenland K, Winquist A. PFAS and cancer, a scoping review of the epidemiologic evidence. Environ Res. 2021 Mar;194:110690. doi: 10.1016/j.envres.2020.110690. Epub 2020 Dec 30; Kirk et al, The PFAS Health Study: Systematic Literature Review. Canberra: The Australian National University. 2018.

[98] Vermeulen SH, Hanum N, Grotenhuis AJ, Castaño-Vinyals G, van der Heijden AG, Aben KK, Mysorekar IU, Kiemeney LA. Recurrent urinary tract infection and risk of bladder cancer in the Nijmegen bladder cancer study. Br J Cancer. 2015 Feb 3;112(3):594-600. doi: 10.1038/bjc.2014.601. Epub 2014 Nov 27. PMID: 25429525; PMCID: PMC4453642.

[99] Hird AE, Saskin R, Liu Y, Lee Y, Ajib K, Matta R, Kodama R, Carr L, Kulkarni GS, Herschorn S, Narod SA, Nam RK. Association between chronic bladder catheterisation and bladder cancer incidence and mortality: a population-based retrospective cohort study in Ontario, Canada. BMJ Open. 2021 Sep 2;11(9):e050728. doi: 10.1136/bmjopen-2021-050728. PMID: 34475180; PMCID: PMC8413958.

Radiation therapy to the pelvic area increases the future risk of bladder cancer.[100] I do not consider this to be a risk factor for Ms. Dyer as she had no past treatment involving radiation prior to being diagnosed with bladder cancer.

9. Demographic considerations

Approximately 80% of all bladder cancers are diagnosed in individuals age 65 and older,[101] and the median age for bladder cancer is 73.[102] Bladder cancer is four times more common in men than women. Given the higher incidences of bladder cancer diagnosis for males over 65, age and gender are counter indications for Ms. Dyer's bladder cancer diagnosis.

In light of the known risk factors for bladder cancer, I can rule out the following conditions: occupational exposures, family history of bladder cancer, past treatment by some anticancer drugs other chemical exposures, history of bladder infections, long use of urinary catheters, demographic considerations, and radiation to the pelvic area.

IX. Opinions

1. It is at least as likely as not that Ms. Dyer's exposure to chemicals in the water at Camp Lejeune caused her bladder cancer

Applying the methodology of differential etiology, a significant risk factor for Ms. Dyer to develop bladder cancer was her exposure to the water at Camp Lejeune. Ms. Dyer had extensive exposure to the water at Camp Lejeune as a resident living in Tarawa Terrace, attending high school at Tarawa Terrace, and through her social life. Ms. Dyer's family moved to Tarawa Terrace in January 1958 when she was a year and a half old and she remained on base, except for a year, until 1973. By the time Ms. Dyer arrived at Tarawa Terrace, levels of PCE in finished drinking water circulated by Tarawa Terrace's water treatment plant already exceeded the EPA's maximum contaminant level and only increased from there. Ms. Dyer and her family "lived on tea and water"; she took nightly baths and enjoyed numerous recreational activities across the base. Thus, as a child, Ms. Dyer was exposed to the contaminated water via ingestion, inhalation (water vapor), and through her skin. As noted in Dr. Plunkett's report, the EPA has concluded that PCE is "likely to be carcinogenic in humans by all routes of

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[100] Li S, Wei R, Yu G, Liu H, Chen T, Guan X, Wang X, Jiang Z. Risk and prognosis of secondary bladder cancer after radiation therapy for pelvic cancer. Front Oncol. 2022 Aug 24;12:982792. doi: 10.3389/fonc.2022.982792. PMID: 36091158; PMCID: PMC9449132. Available at https://www.frontiersin.org/journals/oncology/articles/10.3389/fonc.2022.982792/full

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

[102] 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)

exposure".[103] Ms. Dyer was also exposed to TCE as she visited different parts of the base, which has been recognized as "carcinogenic in humans by all routes of exposure." Dr. Reynolds' data also reveals significant exposures to benzene and vinyl chloride as well.

As explained by Dr. Plunkett, inter-individual differences and individual susceptibility are important considerations when assessing whether diseases are caused by toxic exposures.[104] While it is impossible to understand the full and complete nature of Ms. Dyer's individual susceptibilities, her age at exposure is an important consideration. There is wide agreement that children are highly vulnerable to chemical toxins. Children consume more water in proportion to their body size than adults, as much as seven times per kilogram of body weight than adults.[105] Young children breath in more oxygen per their body weight than adults, and as a result would have higher inhalation exposure.[106] EPA's Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens, cited by Dr. Plunkett, recognizes that "children are generally assumed to eat more food and drink more water relative to their body weight than adults."[107] Children's foods are more likely to have a higher water content than adults.[108] Lower body weight means children are disproportionately exposed to a greater amount of toxins. Children's ability to metabolize and excrete many toxins differs from that of adults, leaving them less well able to deal with chemical toxins (Rane A. Pediatr Clin N Am 1972;19:37–49). In light of the above, Ms. Dyer was uniquely susceptible to the chemical exposure at Camp Lejeune.

Ms. Dyer's age and gender are significant factors. The median age of bladder cancer diagnosis is 73 years old, and more males than females get diagnosed with bladder cancer. Increased age is associated with bladder cancer because of the accumulation of toxins over time and the length of time needed for cancer cells to grow in the urothelium. For Ms. Dyer to be diagnosed at 52 with bladder cancer, and that she is the less common gender, together indicate that she received a significant toxic exposure. Related to her age at diagnosis, the duration of time between her last exposure (approximately 1975) and diagnosis (2009) is well in line with the latency I expect from a toxic exposure-induced bladder cancer.

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[103] Plunkett ¶ 25
[104] Plunkett ¶ 121
[105] Carroquino MJ, Posada M, Landrigan PJ. Environmental Toxicology: Children at Risk. Environmental Toxicology. 2012 Dec 4:239–91; *see also* Carpenter and Bushkin-Bedient, Exposure to chemicals and radiation during childhood and risk for cancer later in life. Journal of Adolescent Health. 2013.
[106] Carroquino MJ, Posada M, Landrigan PJ. Environmental Toxicology: Children at Risk. Environmental Toxicology. 2012 Dec 4:239–91.
[107] US Environmental Protection Agency. 2005. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F, March 2005, p. 30.
[108] Hauptman and Woolf, Childhood ingestions of environmental toxics: what are the risks? Pediatr Ann. 2017 Dec 1;46(12):e466-e471.

In short, Ms. Dyer 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 her bladder cancer diagnosis was caused by the water at Camp Lejeune.

2. There are no other causes for Ms. Dyer's bladder cancer that are more likely than not to cause her diagnosis

Ms. Dyer does not have any of the risk factors traditionally associated with bladder cancer with the exception of smoking. None of her immediate family relations have a history of bladder cancer, and she had no prior history of bladder infections or use of catheters prior to her diagnosis. She has no known exposure to well water, and there is no scientific support to connect her potential exposure to PFAS to bladder cancer. She was diagnosed over 20 years earlier than the median age for bladder cancer, and as a female she is the less common gender to get bladder cancer. She has not taken anti-cancer drugs associated with bladder cancer, and had no history of bladder infections, long use of urinary catheters, or radiation to her pelvic area prior to her diagnosis. Ms. Dyer's does not have any discernable occupational exposure that would contribute to her bladder cancer diagnosis. Thus, the only remaining risk factor is smoking.

While smoking is a recognized cause of bladder cancer, Ms. Dyer's personal smoking history and exposure to second-hand smoke is either so minor as to be medically insignificant or at most a risk factor that cannot be distinguished from her Camp Lejeune exposure. Ms. Dyer is either classified as a never-smoker based on the number of cigarettes she reported smoking, or as a smoker with a brief history (1-2 years) and low intensity (.25 to .5 packs per day). Additionally, there is nothing remarkable or discernable concerning her potential second-hand smoke exposure.

Even considering smoking as a potential risk factor, there is nothing to suggest that this is an overriding risk. While smoking increases the risk of developing bladder cancer, substantial numbers of smokers with dramatically higher cigarette usage than Ms. Dyer never develop bladder cancer,[109] meaning that smoking in and of itself does not inevitably lead to bladder cancer. Importantly, the ATSDR Camp Lejeune-Camp Pendleton studies controlled for smoking and still saw increases of bladder cancer in the Camp Lejeune cohort. Dr. Bird's epidemiological review also noted a number of studies that likewise controlled for smoking and saw increases in bladder cancer following exposures to the same chemicals at Camp Lejeune. In addition, smoking often works in conjunction with other exposures to lead to cancer. Even accepting the higher cigarette usage for Ms. Dyer, this exposure would have come later in her life after she was exposed to bladder cancer-causing chemicals at Camp Lejeune. While it is not possible to know with any level of accuracy whether one of the two types of exposures were sufficient in and of themselves, there is sufficient information to conclude that both are equally legitimate risk factors that cannot be disregarded.

[109] Freedman 2012

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

X. Past Burden and Future Prognosis

As stated previously, Ms. Dyer's life has been permanently changed by her diagnosis of bladder cancer. The burden of disease involves almost every aspect of the patient's life, including their daily bodily functions, how they view themselves, their social relationships and their intimate relations. While I am hopeful that she has been cured of her disease, she will be left with the sequelae mentioned above. Regardless, she will need lifelong cancer surveillance. Ms. Dyer has a permanent urinary diversion. Ms. Dyer will require changes of her ostomy appliances several times a week. She is unable to perform this function herself and is reliant on her husband. These requirements for the application of medical care interfere with the activities and experiences of normal life. In the future, this may require nursing aide. She will always require access to medical supplies. Ms. Dyer has already suffered from a parastomal hernia. The repair was complex and involved another ICU admission and an abscess, and she continues to have a parastomal hernia. Ms. Dyer's radical cystectomy involved her genitalia. This impacts her sexual function and intimate relationship. It directly impacts the patient's physical intimacy through pain, embarrassment, and loss of function.

Ms. Dyer has struggled with urinary tract infections following her cystectomy and is expected to continue to have UTIs. There will be colonization of the urine tract with bacteria after ileal conduit urinary diversion. There is persistent colonization of this segment of ileum with bacteria even after dissociation and isolation from the rest of the small bowel. Urinary tract infections are common in patients with an ileal conduit. The colonized ileal segment with attached ureters allows for bacterial contamination of the urinary tract with an easy passageway up the ureters into the kidneys, predisposing patients to UTIs and pyelonephritis. This is also exacerbated by the fact that the ureter to bowel connection is refluxing with freely flowing urine passing back and forth across the connection point. Most patients have clinical signs and symptoms such as fevers, chills, sweats, foul smelling urine, pyuria, stomal pain or burning, or flank pain.

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.[110] 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.[111] 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.[112] 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.[113] Based on Ms. Dyer's medical records, Ms. Dyer has clearly (and not surprisingly) struggled with anxiety and depression related to her diagnosis of bladder cancer.[114]

XI. Conclusion

In conclusion, Ms. Dyer had substantial exposure to known bladder cancer-causing chemicals at Camp Lejeune. As a child, she was uniquely susceptible to these chemicals. Her diagnosis of bladder cancer at the early age of 52 is therefore not surprising. Ms. Dyer does not have any significant risk factors besides smoking, and even her smoking history is either irrelevant to her diagnosis or significantly diminished by her low intensity and over 30-year cessation. After she developed bladder cancer, Ms. Dyer 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 diagnosis, it is my opinion to a reasonable degree of medical and

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[110] 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.
[111] 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.
[112] 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.
[113] 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.
[114] Dyer Dep. 40-31, 130-138, 172, 195, 210-212, 223-225, 228, 239-240

scientific certainty that Ms. Dyer's exposure to contaminated water at Camp Lejeune is at least as likely as not a cause of her 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	
	University of Nebraska Medical Center Omaha, Nebraska Urology	July 2011-June 2015
Fellowship	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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