# Exhibit 341

**Specific Causation Expert Report for Edward Raymond**

**Vincent M. Bivins, M.D., FACS**
Urologic Oncologist
Urology Centers of Alabama
3485 Independence Drive
Homewood, Alabama 35209

Vincent M. Bivins, MD Date: 2/7/2025

## Background

I am a board-certified urologic oncologist and Fellow of the American College of Surgeons with extensive expertise in the management of cancers of the genitourinary system, including prostate, renal, and bladder cancer. I have been at the forefront of minimally invasive techniques to improve patient outcomes.

I am board-certified in Urology by the American Board of Urology (2004, renewed 2014). I completed my Urology Oncology Fellowship at the University of Washington Medical Center and his Urology Residency at the University of Oklahoma Health Sciences Center. I earned a medical degree from the University of Alabama at Birmingham (UAB) School of Medicine, where I now serve as a Preceptor for the Physician Assistant program.

Currently, I serve as the President and CEO of Urology Centers of Alabama and Director of the Van Scott Cancer Center. I have held numerous leadership positions, including President of the Alabama Urology Network and Chairman of the Department of Surgery at Brookwood Baptist Medical Center.

I actively contribute to various boards, including the YMCA of Greater Birmingham and the American Cancer Society of Jefferson County. I am a recognized leader in diversity and inclusion initiatives within the field of urology.

I am a member of several prestigious professional organizations, including the American Urological Association, Society of Urologic Oncology, and Endourology Society. My accolades include recognition by Leadership Alabama and Leadership Birmingham for my contributions to medicine and community service.

## Records and Materials

The list of materials I have considered as part of this report is included as an appendix.

## Causation Standard

The statute at issue in this case states that there are two ways to meet the causation burden:

> "(2) Standards – To meet the burden of proof described in paragraph (1) a party shall produce evidence showing that the relationship between exposure to the water at Camp Lejeune and the harm is –
>
> > "(A) sufficient to conclude that a causal relationship exists; or
> > "(B) sufficient to conclude a causal relationship is at least as likely as not."

This standard was considered throughout this opinion and all opinions contained in this report are expressed to a reasonable degree of scientific and medical certainty.

## Methodology

As a practicing urologic oncologist, I have specialized training and education in the suspected causes of bladder cancer. In my clinical practice, I treat all types of genitourinary cancer, including bladder cancers. As a member of the Society of Urologic Oncology, it is my practice to review current literature regarding potential causes of bladder cancer. This includes reviews of peer-reviewed journal articles and other well-known sources like IRAC monographs, ATSDR, and EPA.

In my clinical practice, I utilize a differential diagnosis to identify the underlying cause of an injury or symptom for best patient care. This approach involves systemically considering all potential causes of a patient's condition and available clinical information to rule out possible etiologies. For patients presenting with bladder cancer, I rule in all etiologies and conditions that may have a causal association with bladder cancer, then I rule out potential causes based on the patient's medical history, occupational history, lifestyle, and any other relevant material available to me. In reviewing Mr. Raymond's circumstances to come to a determination that a certain exposure caused his bladder cancer, I followed this traditional methodology.

I relied on my training, education and experience to determine risk factors for the development of bladder cancer. Additionally, I examined peer-reviewed scientific literature pertaining to bladder cancer risks associated with exposure to TCE and Benzene, including occupational and environmental exposures. After reviewing peer-reviewed literature, I also relied on sources such as the EPA, IARC, and ATSDR. In this case, I was also able to rely on the general causation reports of Dr. Steven B. Bird, Dr. Stephen H. Culp, Dr. Benjamin Hatten, Dr. Kathleen Gilbert and Dr. Laura M. Plunkett. Once I came to a determination that there is enough evidence to establish a causal association, I then proceeded with a differential diagnosis reviewing the exposure to the water at Camp Lejeune with other known risk factors in determining if the exposure to the water at Camp Lejeune was a cause of Mr. Raymond's bladder cancer.

## Summary of Opinions

I have concluded that Mr. Raymond was exposed to water containing significant levels of TCE and Benzene. There is scientific and epidemiological support for TCE and Benzene causality of bladder cancer. The levels of TCE and Benzene Mr. Raymond was exposed to are known to be hazardous to human health – specifically bladder cancer. Therefore, it is my opinion, to a reasonable degree of medical and scientific certainty, Mr. Raymond's exposure to the contaminated water at Camp Lejeune is as likely as not a cause of Mr. Raymond's bladder cancer.

## Mr. Raymond's Personal and Exposure History

Edward Raymond was born [redacted], 1945, and lives in Mechanicville, New York.[1] He has been married to his wife, Barbara Raymond, since 1968.[2] They have two sons, and five grandchildren.[3]

[1] Raymond Depo. 2:2-6; 11:12-22
[2] Raymond Depo. 42:19-22; 143: 14-15
[3] Raymond Depo. 25:10-13; 159:14-160:18.

Mr. Raymond served active duty in the Marines Corps from July 10th, 1963 to June 30th, 1967.[4] He enlisted shortly after graduating high school.[5] After completing boot camp at Paris Island, South Carolina, he reported for duty at Camp Lejeune on or about November 22, 1963.[6]

According to the Exposure Assessment performed by Dr. Kelly Reynolds, Mr. Raymond's service at Camp Lejeune spanned from November 22, 1963, to December 1, 1965, and included an estimated 662 days of exposure at Camp Lejeune. During this time, he was exposed primarily to the Hadnot Point water treatment plant water for the purposes of water consumption, showering, bathroom, mess hall, meals, recreation, and everyday basic living.

Mr. Raymond completed infantry training at Camp Lejeune for approximately the first six weeks he was stationed there in barracks style housing.[7] He was then assigned to the 8th Comm Battalion for his remaining two years at Camp Lejeune.[8]

Mr. Raymond worked as a Radio Telegraph Operator (MOS 2533).[9] During his time as a radio operator, he would maintain equipment and participate in training and field operations.[10] He lived in the main side barracks at Hadnot, and he ate most of his meals at the nearby mess hall.[11]

Mr. Raymond testified that he showered at least once a day for approximately 15 minutes.[12] He drank at least one canteen of water a day, sometimes two, and he would also drink from the water fountains and from a cooler in the mess hall.[13] He would fill his canteen with the water buffaloes on the main side.[14]

During his time stationed at Camp Lejeune, Mr. Raymond testified that he would spend one or two evenings a month off base going out to eat.[15] Additionally, he spent a few brief periods off the base, either on leave or deployed overseas. Mr. Raymond was transferred from Camp Lejeune on December 1, 1965.[16] He served in Vietnam, where he was presumptively exposed to Agent Orange (AO), though he has no recollection of actual AO exposure.[17]

After he was released from the Marine Corps in 1967, Mr. Raymond was employed by General Electric ("GE") for 38 years.[18] He was an operator for eight years and a control operator for thirty years.[19] During his employment with GE, he was exposed to chemicals like cooper dust

[4] 00546_RAYMOND_VA_0000000548
[5] Raymond Depo. 66:21-24.
[6] 00546_RAMOND_VA_0000000414-415; Raymond Depo. 82:5-18.
[7] 00546_RAMOND_VA_0000000414; Raymond Depo. 83:16-84:6
[8] 00546_RAYMOND_VA_0000000414
[9] 00546_RAYMOND_VA_0000000548
[10] Raymond Depo. 94:11-22.
[11] Raymond Depo. 95:22-96:20.
[12] Raymond Depo. 108:15-22.
[13] Raymond Depo. 111:12-112:8; 109:10-24
[14] Raymond Depo. 110:5-23.
[15] Raymond Depo. 99:10-14
[16] 00546_RAYMOND_VA_0000000414.
[17] 00546_RAYMOND_VA_0000000414.
[18] Raymond Depo. 149:13-158:11.
[19] Raymond Depo. 157:4-23.

and silicone dust. He wore protective equipment including safety glasses and an N95 mask or better.[20]

Mr. Raymond smoked approximately a pack of cigarettes a day from 1964-2013.[21] Mr. Raymond's brother had lung cancer, and his sister had pancreatic cancer.[22]

**Bladder Cancer Generally**

The bladder is part of the urinary tract which sits in the pelvis, its primary purpose is to store and excrete urine at appropriate times. The bladder is comprised of three layers: the urothelium, the Lamina Propia, and Musclaris Propia. The urothelium is the innermost layer of the bladder, and acts as a protective lining against toxic products and stored waste. The Lamina Propia contains the blood vessels and nerves of the bladder. The final layer is the Musclaris Propia, where the bladder muscles are located. The synergy of these three layers allows for a coordinated process of storing and voiding urine.

There are typically three types of bladder cancer, distinguished by the cell of origin. Urothelial Carcinoma is the most common, comprising 90% of bladder cancers, and is derived from the urothelial lining. Due to urothelial direct exposure, the urothelial subtype is strongly related to exposure to chemicals. (Haleseh et al. 2022). Squamous Cell Carcinoma develops from squamous cells of the bladder and is typically seen with chronic irritation and infections in areas where schistosomiasis infections occur or in patients with catheters. Adenocarcinoma, a less common subtype, is derived from the glandular area of the bladder and is seen in patients with a birth defect of a persistent urachal remnant.

Bladder cancer is caused by changes to the cells of the bladder. It is often linked with exposure to certain chemicals and is rarely idiopathic. Risk factors for developing bladder cancer include smoking, family history, pelvic radiation, occupational and environmental exposure, certain medication, chronic infection and bladder irritation, certain medical conditions such as obesity and diabetes and certain genetic conditions such as Lynch Syndrome Estimates indicate that occupational carcinogens account for up to 25% of bladder cancer diagnoses among males. (Xie et al. 2024).The application of these risk factors to Mr. Raymond's case will be detailed below.

Early detection is key to treatment. The most common presentation is painless hematuria. This can be gross (seen by the eye) or microscopic (detected by the microscope). Symptoms span from asymptomatic (without symptoms), irritative voiding symptoms; frequency, urgency or dysuria, pain, or systemic symptoms such as weight loss, fatigue or bone pain.

The workup includes a history, physical exam, imaging, and pathological assessment. The first step would be a comprehensive history and physical examination. This focuses on risk factors, family history, medical history, duration, and severity of symptoms. Urine studies are obtained

[20] Raymond Depo. 154:3-12.
[21] Raymond Depo. 116:24-117:9.
[22] Raymond Depo. 46:15-19; 48: 21-24.

evaluating for blood, infection, cytology (identifying malignant cells) and urine biomarkers. Imaging, CT urography (Fig 1) or MRI, of the entire urinary tract is obtained to assess or detect the presence and location of the tumor. Finally, cystoscopy and TURBT is performed which allows for direct visualization and biopsy of bladder tumors. This will provide both diagnostic and therapeutic analysis of the tumor to include tumor grade and staging of the cancer. Figure 2 below shows cystoscopy and TURBT.

Fig 1(Fig 1a shows CT scan of bladder tumor and Fig 1b shows tumor on cystoscopy)

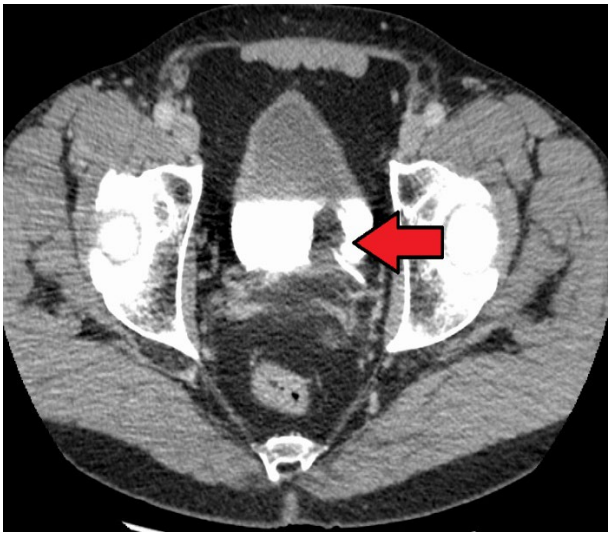


Fig 1a

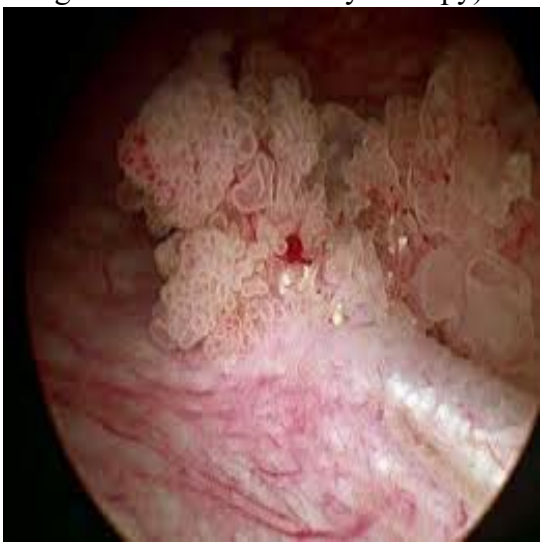
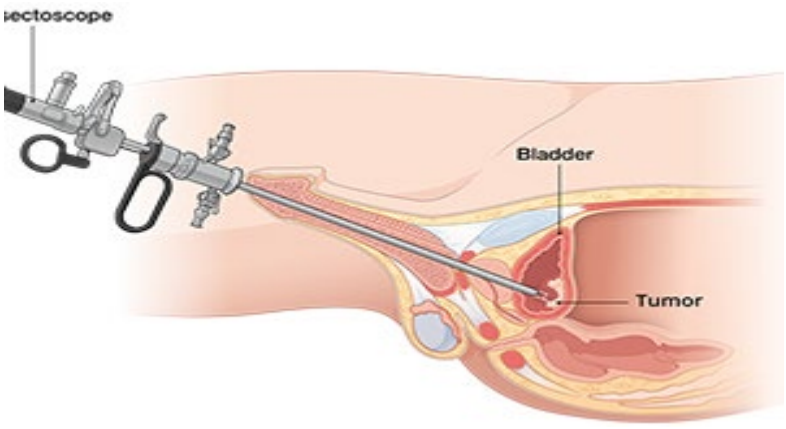


Fig 1b

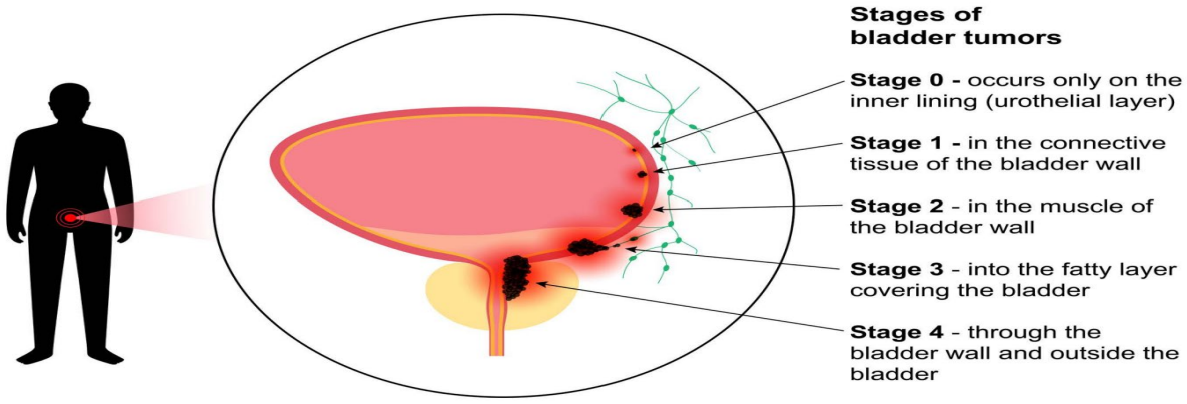
Finally, cystoscopy and Transurethral Resection of Bladder Tumor (TURBT) is performed which allows for direct visualization and biopsy of bladder tumors. This will provide both diagnostic and therapeutic analysis of the tumor to include tumor grade and staging of the cancer. Figure 2 below represents cystoscopy and TURBT.

Fig. 2



The treatment of bladder cancer depends on the pathology, staging, grading and health of the patient. As discussed above, there are three histopathological subtypes of bladder cancer. The most common, urothelial carcinoma, is historically known as Transitional Cell Carcinoma. The bladder comprises 90% of urothelial carcinoma with 10% of the kidneys and ureters. The tumor is graded as high or low according to its architectural and cytological features. This depicts how fast cancer grows and the likelihood of recurrence. The tumor is further defined if it invades the bladder muscle. The classification is Non-Muscle or Muscle invasive.

Fig. 3




Non-Muscle Invasive Bladder cancer is further classified into risk categories that help in treatment decision making. It is divided into Low, Medium and High-risk categories.

Fig. 4 (NCCN Guidelines Insights: Bladder Cancer, 2024)

**Guide 3**
**Follow-up care by risk level for non–muscle-invasive bladder cancer**

| | Low risk | Medium risk | High risk |
|---|---|---|---|
| **Cystoscopy** | **Year 1:** At 3 and 12 months<br>**Years 2–5:** Once a year<br>**After that:** As directed by your doctor | **Year 1:** At 3, 6, and 12 months<br>**Year 2:** Every 6 months<br>**Years 3–5:** Once a year<br>**After that:** As directed by your doctor | **Years 1–2:** Every 3 months<br>**Years 3–5:** Every 6 months<br>**Years 6–10:** Once a year<br>**After that:** As directed by your doctor |

Treatment is dependent on the stage and grade of the cancer. For Non-Muscle Invasive Bladder Cancer, (NMIBC) the treatment is dependent on the stage, grade and risk. For Low Risk NMIBC the treatment is surveillance. (see Fig 5).

For Medium Risk NMIBC and High risk NMIBC the treatment is intravesical BCG, which an attenuated form of Tuberculosis that creates a local immune response and reduces recurrence. (Fig 5)

Fig. 5 (NCCN Guidelines Insights: Bladder Cancer, 2024)

| Guide 2<br>Stage 0 and stage 1 treatment options | | | |
|---|---|---|---|
| **Low risk** | • Small, single, slow-growing stage 0a lesion | → | • Surveillance |
| **Medium risk** | • Large or multiple slow-growing stage 0a tumor(s)<br>• Slow-growing stage 0a tumor that comes back within 1 year<br>• Small, single, fast-growing stage 0a tumor<br>• Slow-growing stage 1 tumor | → | • Intravesical (BCG or chemo) therapy*<br>• Surveillance |
| **High risk** | • Large or multiple fast-growing stage 0a tumor(s)<br>• Fast-growing stage 0is tumor<br>• Fast-growing stage 1 tumor | → | • Intravesical BCG therapy*<br>• Radical cystectomy |
| | • Stage 0 or stage 1 tumor with very high-risk features | → | • Radical cystectomy*<br>• Intravesical BCG therapy |

**Preferred treatment*

For muscle Invasive Bladder Cancer, the treatment options are surgery cystectomy, bladder removal, radiation, chemotherapy, immunotherapy or a combination of these options.

**Mr. Raymond's Clinical History**

Edward David Raymond is a 79-year-old man with a history of metastatic bladder cancer.

Mr. Raymond has additional diagnoses of COPD, moderate pulmonary hypertension, EGD, iron deficient anemia, diastolic Congestive Heart failure, and Benign Prostatic Hyperplasia.

Mr. Raymond underwent a cystoscopy and a trans urethral resection of bladder tumor for diagnosis on December 17, 2021.[23] He was diagnosed with invasive, high grade papillary urothelial carcinoma cancer on December 17, 2021, at 76 years old and approximately 56 years after his last date of service at Camp Lejeune.[24].

[23] 00546_RAYMOND_SH_0000000305-306.
[24] 00546_RAYMOND_SH_0000000347-348; 00546_RAYMOND_VA_0000000414.

Mr. Raymond was highly anxious and upset when the bladder mass was first discovered, and he was provided with Xanax to treat his anxiety.[25] After his bladder cancer diagnosis, Mr. Raymond was subsequently diagnosed with erectile dysfunction on March 7, 2023. [26]

Since his initial diagnosis and treatment, Mr. Raymond has undergone an additional eight cystoscopies, a second transurethral resection of his bladder tumor, and six doses of intravesical Bacillus Calmette-Guerin ("BCG").

Mr. Raymond's bladder cancer journey began when he was admitted to Saratoga Hospital on October 21, 2021 with a bladder mass incidentally found on a CT scan. He was admitted with symptoms of right lower quadrant abdominal pain. He had a mass measuring 2.3 x 1 x 2.4 cm in size that would eventually be diagnosed as bladder cancer. His journey from this mass to metastatic bladder cancer is as follows:

| | |
|---|---|
| October 2021 | 2.3 cm tumor found on CT scan |
| November 15, 2021 | 1 cystoscopy shows larger sessile mass on left bladder wall |
| December 17, 2021 | Transurethral Resection of Bladder Tumor (TURBT) and pathology papillary urothelial carcinoma stage T1 (invasion into the Lamina Propia), High Grade, Musclaris propia present |
| March 2022 | Cystoscopy with biopsies all negative |
| July 2022 | Surveillance Cystoscopy shows lesions at the dome of the bladder |
| July 21, 2022, | Dr. Brian Yamada performs TURBT, and pathology reveals urothelial carcinoma stage Ta (limited to mucosa) High Grade, No Muscle seen |
| September 14 – October 22, 2022 | Weekly Intravesical bladder treatment of BCG for 6 weeks |
| November 2022 | Surveillance cystoscopy negative |
| March 7, 2023 | Surveillance cystoscopy showed a red area in bladder that was cauterized |
| June 2023 | Surveillance cystoscopy raised red area, friable, bleeding and cauterized it |
| August 2023 | Surveillance cystoscopy persistent small red area at the anterior bladder neck, cauterized |
| November 22, 2023 | Surveillance cystoscopy showed papillary lesion at anterior bladder neck, cauterized (felt cautery potentially not functional) |
| February 21,2024 | PSA 6.4 |
| May 22, 2024 | Surveillance cystoscopy showed the area at bladder neck resolved and small area on left lateral wall but not suspicious. |
| September 2024 | Admitted with shortness of breath and found to have lung and mediastinal lesions on CT scan. PET CT Scan shows diffuse metastatic disease, including prostate, seminal vesicles, base of bladder causing left hydronephrosis, iliac lymph nodes, |

[25] 00546_RAYMOND_SH_0000000170 - 0000000176
[26] 00546_RAYMOND_0000000061 – 0000000062

| | |
|---|---|
| | sclerotic lesion at T12, mediastinal nodal involvement and pulmonary metastasis. |
| October 2024 | Saw Oncologist, Talat Mahmood, MD and started on systemic therapy Pembro and Enfortumab |

**Prognosis**

Unfortunately, Mr. Raymond now has metastatic urothelial carcinoma, which carries a five-year mortality rate of 8%. Mr. Raymond was diagnosed with metastatic urothelial carcinoma in August 2024. His PET scan showed cancer in the prostate, bladder, seminal vesicles, pelvic lymph nodes, mediastinal lymph nodes, pulmonary metastasis and metastasis at T12 spinal vertebrate. He has a biopsy of lungs to confirm this metastasis of his urothelial carcinoma which makes him a TNM staging of T4N2M2.

He has suffered significant morbidity to include pain requiring narcotics. He has also had a decrease in appetite and weight loss. He states he has seen a decline where he could do 90 minutes of exercise and can no longer. He has gone into urinary retention requiring a catheter through his penis to allow drainage of the bladder. He has had chronic constipation and is also noted to have depression.

Systemic treatment of enfortumab vedotin (Padcev) and Pembrolizumab (Keytruda) have several known side effects. Padcev is an antibody drug conjugate that delivers toxic agents to the cancer cell. Side effects of Padcev include rash, fatigue, weight loss nausea, and nausea. Keytruda blocks the PD1 pathway used by cancer cells to evade the immune system. By blocking the PD 1 pathway, cytotoxic t cells (immune cells) activate the immune system to kill cancer. However, there are systemic side effects with this medication including, but not limited to: fatigue, joint pain, and renal failure.

Considering his recent diagnosis, Mr. Raymond's current treatment is evolving, and my report will be supplemented as more records become available concerning his current health.

I reviewed Mr. Raymond's medical records and expenses and found the treatment to be medically necessary and the expenses are fair and reasonable.

**Edward Raymond's Exposure to Contaminants in the Water At Camp Lejeune were At Levels Known to be Hazardous to Human Health, Specifically Bladder Cancer**

I have reviewed the specific causation opinions from Dr. Reynolds, Dr. Hatten, and Dr. Bird evaluating the exposure of Mr. Raymond to the volatile organic compounds found in the water at Camp Lejeune, as well as the water modeling reports of Dr. Maslia and the ATSDR. Mr. Raymond was exposed to TCE and benzene during his active duty at Camp Lejeune. His TCE levels ranged from 18 to 26 µg/L and benzene levels ranged from 0 to 1 µg/L. Mr. Raymond was exposed to approximately 579 µg/L-months of TCE and 21 µg/L-months of benzene.

Dr. Reynolds provided a report that estimated the cumulative ingestion amounts for Mr. Raymond during his time at Camp Lejeune:

| | | Chart 1: 1L | Chart 2: ATSDR marine in training (4.334 L consumption per day) | Chart 3: ATSDR Civilian worker RME (3.092 L consumption per day) | Chart 4: ATSDR Civilian worker CTE (1.227 L consumption per day) | Chart 5: Days on base and cumulative contaminant exposure concentrations FM 1957-1983 moderate day averages |
|---|---|---|---|---|---|---|
| | Cumulative ug/l-M | Cumulative consumption (total ug= days*concentration per L) | Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions) | Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions) | Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions) | Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions) |
| **TCE** | 579 | 14,676 | 63,606 | 45,378 | 18,007 | 97,239 |
| **PCE** | - | - | - | - | - | - |
| **VC** | - | - | - | - | - | - |
| **BZ** | 21 | 528 | 2,288 | 1,633 | 648 | 3,498 |

Dr. Reynolds was only assessing ingestion, but Mr. Raymond was also exposed via inhalation and dermal exposure routes. Dr. Bird and Dr. Plunkett have explained that the inhalation and dermal exposure may be equal to or greater than his ingestion exposure. Considering the duration (chronically for approximately 662 days) of exposure, the frequency (multiple times a day through three exposure routes) of exposure, and the intensity (at levels known to be hazardous to human health), it is my opinion that Mr. Raymond had a substantial exposure to TCE and Benzene while stationed at Camp Lejeune.

Additionally, in Dr. Bird's supplemental report on the EPA banning TCE and PCE, there is reference to the EPA stating it clear that "These risks are present even at very small concentrations." Going further, the EPA found that although "acute single exposures" to TCE can be hazardous, "other risks are incurred following long-term repeated exposures." (Federal Register Vol. 89. 102572). EPA has "identified significant health effects associated with short- and long-term exposure to TCE." (Federal Register Vol. 89. 102575). According to EPA, "TCE presents an unreasonable risk of injury to human health under the conditions of use based on acute and chronic non-cancer risks and cancer risks."(Federal Register Vol. 89. 102575). Mr. Raymond's nearly two-year long-term chronic exposure to TCE is the type of long-term repeated exposure the EPA determined was hazardous.

Additionally, I reviewed the specific causation reports of Dr. Hatten and Dr. Bird regarding Mr. Raymond's level of exposure, which found that Mr. Raymond was exposed to levels that have been shown to be hazardous to human health, specifically bladder cancer.

Altogether, Dr. Hatten, Dr. Bird and Dr. Reynolds established Mr. Raymond had a substantial exposure to TCE and Benzene, and the level of his exposure exceeded the levels that have been shown to be hazardous to human health – specifically bladder cancer.

**Bladder Cancer Associated with TCE and Benzene Exposure**

I have read the general causation reports of Dr. Bird, Dr. Culp, Dr. Gilbert, Dr. Hatten and Dr. Plunkett. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of TCE, and Benzene and bladder cancer. These reports are consistent with my review of the literature and support my opinions on this case.

My review of the literature supports that exposure to TCE and Benzene are a risk factor for development of bladder cancer.

**Trichloroethylene (TCE)**

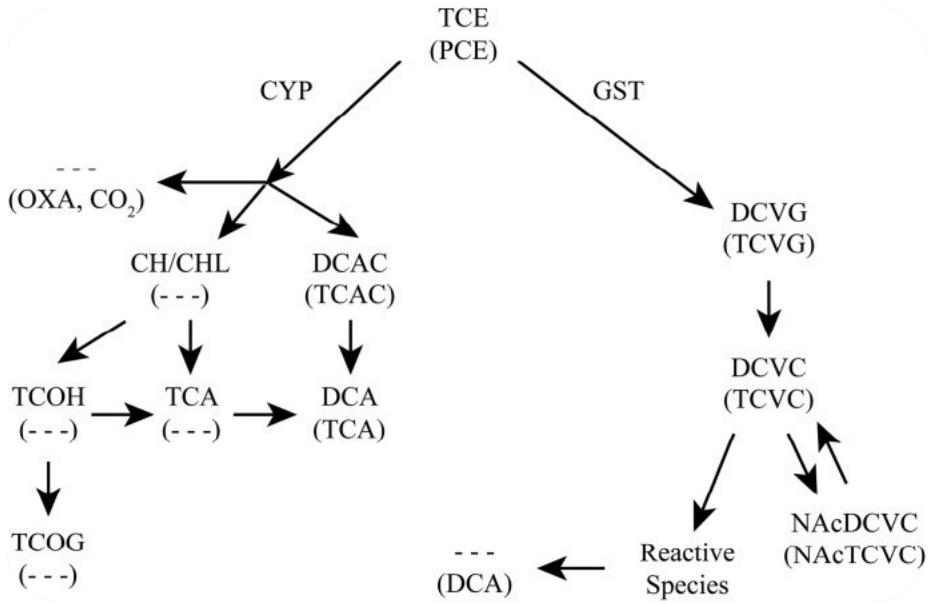
TCE was a solvent found in high concentrations at MCB Camp Lejeune. TCE has been designated as a carcinogen by the IARC and the evidence linking TCE to bladder cancer is suggestive. The following Cohort studies, Raaschou-Nielson et al, (2003), and Hansen et al (2013), showed increased risk of bladder cancer in workers exposed to TCE. The Cancer Hazard of TCE was evaluated by IARC (2014) the US EPA (2001b) National Toxicology Program, (2015). The conclusion of all three assessments was that there is sufficient evidence that TCE is a human carcinogen.

The carcinogenic pathway of TCE is similar to PCE. TCE is metabolized by oxidation into either cytochrome 450 or via glutathione conjugation into genotoxic metabolites. TCE exposure is primarily through inhalation and secondary through dermal. This is because TCE is not water soluble (Cichoki et al, 2016).

TCE is highly fat soluble and is readily taken up into fat tissue. TCE and PCE have similar metabolic pathways (see Fig 7). They are either broken down into toxic metabolites by oxidation in the liver or conjugation in the kidney.

In the oxidation of TCE, the chemical is metabolized through several steps by the Cytochrome P 450 enzyme, CYP2E1 to multiple substrates that eventually end in Dichloroacetic Acid (DCA), Trichloroethanol (TCOH), and Trichloroacetic Acid (TCA). In the Conjugative pathway TCE is then conjugated with Glutathione S-Transferase that is metabolized to di and trichlorovinyl-L-cysteine. (DCVC and TCVC) and these can further metabolize to Mercapturic Acid.

Fig 7. (Cichoki 2016)




TCE is absorbed via the lungs, dermally and to a lesser degree orally. It is subsequently absorbed in the blood stream or stored fat tissue that causes a slower and delayed release of the compound that is metabolized via oxidation through the liver in the oxidation pathway and to a lesser degree conjugation with GST into toxic metabolites, DCA, TCA and or TCOH. It is also taken up into the kidneys where it is conjugated into toxic metabolites and DCA and Mercapturic acid where it can subsequently be excreted in the urine.

Genotoxin is a chemical or agent that causes DNA or chromosomal damage. TCE has been shown to be genotoxic. (Tabrez, 2009) Damage in germ cells causes germline or heritable altered traits and DNA damage in a somatic cell may result in somatic mutation that can lead to malignant transformation. (Tung et al. 2012) Metabolism of TCE via the oxidation pathway and conjugation pathway produces toxic metabolites that are genotoxic. Studies have shown that TCE has a strong association with kidney cancer. As TCE is metabolized through the kidney and produces toxic metabolites it creates chronic irritation, and genotoxic effects to renal tubules and subsequently increase risk of Renal cell Carcinoma. (Cichoki et al. 2016) These metabolites are further secreted in the urine and presented to the bladder. Furthermore, with chronic exposure daily, and TCE being lipophilic both of which gives chronic exposure to the urinary tract irrespective of water samples and these together allow this genotoxic drug to be exposed to the bladder urothelial.

Figure 8 (Lash et al. 2000)

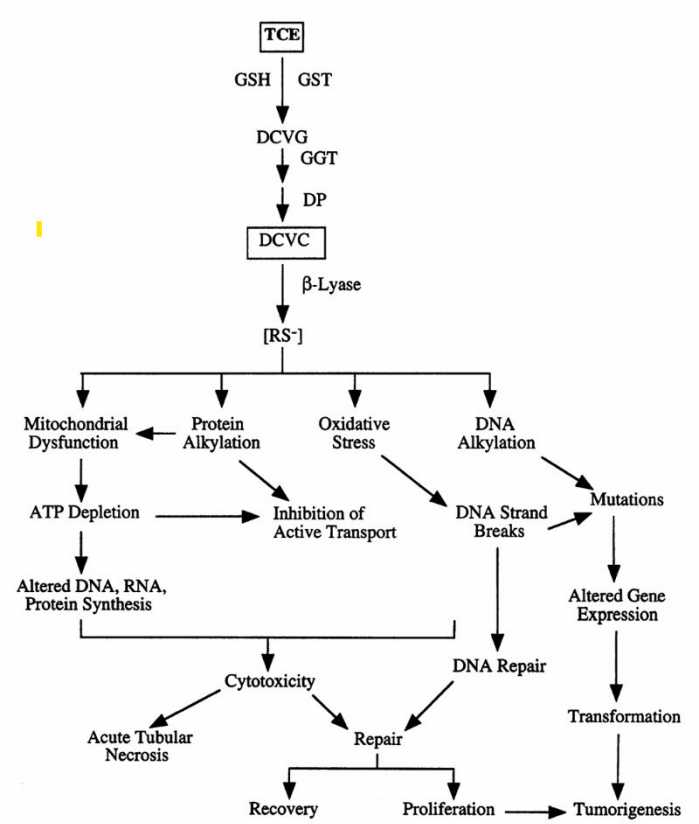



**Figure 4.** Summary scheme of the postulated modes of action of TCE via the GSH conjugation pathway for nephrotoxicity and nephrocarcinogenicity. The scheme summarized demonstrated and hypothesized modes of action of TCE in mammalian kidney, showing the various intracellular targets and the interplay between them in ultimately causing nephrotoxicity or nephrocarcinogenicity. Abbreviations used: DP, dipeptidase; RS⁻, reactive thiol and subsequent species generated from β-lyase-catalyzed metabolism of DCVC.

Urine that leaves the kidney travels through the ureters attached to each kidney and enters the bladder and stored until it is voided. The transient time can be hours. It is believed that the toxic metabolites that create the renal toxicity, genotoxicity and mutagenicity in them can have the same effect on the bladder urothelial, especially with longer storage times.

As discussed above, EPA finalized a rule banning TCE under the Toxic Substances Control Act, describing TCE as "extremely toxic." Dr. Bird provided a supplemental report that highlights the EPA's decision "that any lesser restrictions on the use of TCE would fail to adequately protect public health." (89 Fed. Reg. at 102572)

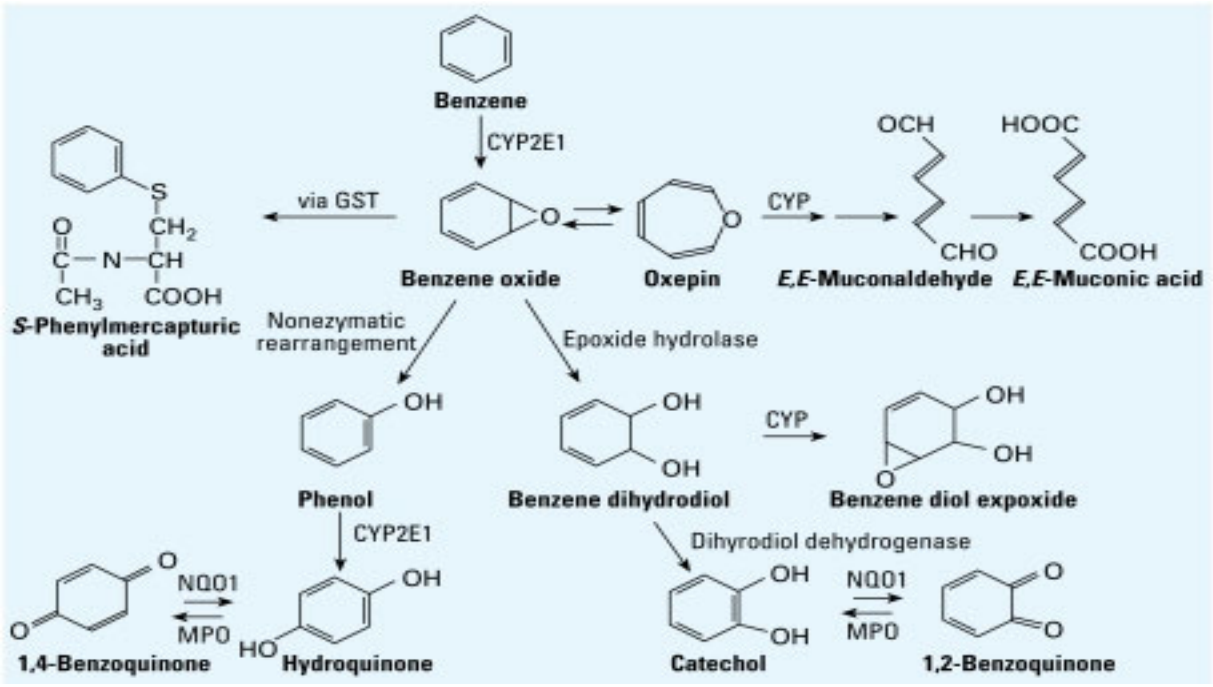
Based upon my education, training and experience, and considering the data and information identified and reviewed, it is my opinion to a reasonable degree of medical certainty that it is at least as likely as not that exposure to TCE from contaminated water at Camp Lejeune was hazardous to human health generally, that exposure to the Camp Lejeune water contaminated with TCE specifically is hazardous to human health, and, further, that the human health hazard could include the development of bladder cancer.

**Benzene**

Benzene has been classified as "carcinogenic to humans" by IARC (2012, 2018), as "carcinogenic in humans by all routes of exposure" by EPA (1998), and as "a known human carcinogen" by NTP (2021c). A recent epidemiologic study by Shala et al (2023) on male offshore petroleum workers showed that there was an increased risk of bladder cancer with benzene exposure with a HR 1.89 and cumulative benzene exposure HR of 1.6.

Benzene metabolism and its metabolites have genotoxic effect on bladder tissue that subsequently forms bladder cancer. Benzene is oxidized by the cytochrome P450 enzyme to benzene oxide. Benzene oxide is either oxidized by the Cytochrome P450 pathway to phenol which can be excreted or further oxidized by the Cytochrome P450 pathway (CYP2E1) to further metabolites to 1,4Benzoquinone or Hydroquinone. Furthermore, Benzene oxide can be oxidized via Cytochrome P450 liver enzymes to Catechol and its metabolites. which represents 70-85% and other metabolites represent the remainder.

Fig. 9 (Rappaport et al, 2009)




Genotoxin is a chemical or agent that causes DNA or chromosomal damage. (Phillips 2009) Damage in germ cells causes germline or heritable altered traits and DNA damage in a somatic cell may result in somatic mutation that can lead to malignant transformation. (Tung et al. 2012) Exposure to Benzene metabolites in particular Benzoquinone has shown in animal studies have been associated with numerous forms of genotoxic damage, including chromosome aberrations, sister chromatid exchanges, DNA and protein cross links, and DNA single and double strand breaks. These chromosome aberrations are associated with multiple forms of cancer. (Tung et al. 2012).

Mr. Raymond was exposed to this carcinogenic compound throughout his time at Camp Lejeune. He was exposed to a constant and persistent amount that tended to accumulate over time. The mechanism is that he had exposure through all three routes to include inhalation, oral and dermal. Once this chemical was absorbed it was then metabolized into metabolites either through the liver and oxidized cytochrome P-450 pathway or conjugated via conjugation by glutathione S transferase further creates toxic metabolites such as phenol and Benzoquinones.

Based upon my education, training and experience, and considering the data and information identified and reviewed, it is my opinion to a reasonable degree of medical certainty that it is at least as likely as not that exposure to benzene from contaminated water at Camp Lejeune was hazardous to human health generally, that exposure to the Camp Lejeune water contaminated with benzene specially is hazardous to human health, and, further, that the human health hazard could include the development of bladder cancer.

**Specific Causation: TCE and benzene exposure and Edward Raymond's Bladder Cancer**

As a Urologic Oncologist, I utilize differential etiology to provide the best patient care. In a differential etiology, a physician reviews known causes of a disease and attempts to rule out those causes as the cause of a patient's disease.

Bladder cancer can develop in patients with multiple risk factors. Environmental and occupational exposures are known risk factors for the development of bladder cancer. Exposures to multiple genotoxins, like smoking and workplace exposures, can have additive effects, acting together to contribute to the development of bladder cancer. Below is a discussion of known risk factors relevant to a differential etiology specific to Mr. Raymond's history.

**Relevant Risk Factors for Developing Bladder Cancer**

1. Exposure to Chlorinated Solvents and Carcinogenic Chemicals

As discussed throughout this report, there is overwhelming evidence that the chemicals present in the water at Camp Lejeune to which Mr. Raymond was exposed can cause bladder cancer. However, it is important to consider other potential exposures he may have had to possible carcinogenic chemicals.

Beyond the chlorinated solvents like those found at Camp Lejeune, some chemicals used in textiles, paint, and rubber manufacturing industries have been identified as associated with bladder cancer textile industry, rubber manufacturing industry, painting industry and aluminum and refined products industry have been shown to have an association with bladder cancer (IARC Monographs No. 100F, 2012)(Guha et al. 2010)(Singh and Chadha, 2016)

After Mr. Raymond left the Marines, he worked for approximately 38 years at the General Electric silicone manufacturing facility in Waterford, New York.[27] During his time at the GE facility, Mr. Raymond mostly worked in an office setting as a control operator, with limited interaction with any silicone or other compounds.[28] During Mr. Raymond's first eight years at the

[27] Raymond Dep. 158:2-3
[28] Raymond Dep. 152:9-10

facility, when working directly with silica equipment, he was required to wear personal protective equipment including eye protection, a helmet, and a respirator to prevent inhalation of any potentially hazardous substances.[29] There is no direct evidence that Mr. Raymond was exposed to chemicals associated with bladder cancer in his workplace, and any exposure was minimized by his use of personal protective equipment. Thus it is highly unlikely to be a cause of his bladder cancer.

2. Herbicide Exposure

Certain herbicides have been associated with the development of cancer, most notably Agent Orange, a chemical herbicide and defoliant comprising 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). Agent Orange has been most strongly associated with the development of soft-tissue sarcomas, non-Hodgkin lymphomas, and lung cancer (IARC; 2012b). There is some evidence that exposure to Agent Orange may be associated with an increased risk of bladder cancer. However, this association is slight, and studies suggesting an association are limited to those with confirmed exposure to Agent Orange, and do not tend to show an association with invasive bladder cancer.(Williams et al. 2023).

Due to his service in Vietnam, Mr. Raymond is presumed to have been exposed to Agent Orange by the VA.[30] However, Mr. Raymond was never confirmed to have been exposed, nor does he recall any exposure during his tour in Vietnam.[31] Given the staging of his bladder cancer and the lack of confirmed exposure, it is unlikely that Agent Orange is a cause of Mr. Raymond's bladder cancer.

3. Smoking

Smoking is a known risk factor for bladder cancer, with studies showing an association between the two for both men and women. (Freedman, et al. 2011).

There is evidence to support that smoking cessation decreases the risk of development of bladder cancer, the risk decreases with longer periods of abstinence from smoking. (IARC Handbooks of Cancer Prevention, Tobacco Control, Vol. 11). Risk of bladder cancer is shown to decrease 30% after 1-4 years of cessation.(Brennan et al. 2000)

Mr. Raymond experienced active and passive exposure to tobacco smoke. He reportedly smoked approximately one pack of cigarettes per day from 1963 to 2013.[32] Given his smoking history, it should be considered as an applicable risk factor in the development of his bladder cancer. Studies relating specifically to exposed Camp Lejeune veterans, which control for smoking, have still shown an increased risk in the development of bladder cancer. (Bove 2024). Additionally, Mr. Raymond had ceased smoking eight years prior to his bladder cancer diagnosis, lowering his risk compared to an individual who continued to smoke. Therefore, it is likely that

[29] Raymond Depo. 154:3-15
[30] Raymond Depo. 133:12-14
[31] Raymond Depo. 133:12-25
[32] Raymond Depo. 117:2-10

Mr. Raymond's smoking history is at least one potential risk factor in his development of bladder cancer.

4. Age

Like all cancers, the risk of developing bladder cancer increases due to age. The average age at diagnosis is 73. With respect to bladder cancer, the older age at diagnosis is indicative of a disease which requires decades between the exposure to mutagens and the mutagen overcoming the body's tumor suppressor mechanisms, finally culminating in carcinogenesis, a process termed latency. (Saginala et al, 2020). Age is best categorized as a representative of cumulative risk from other factors, namely exposures to carcinogens, and thus older age at diagnosis is consistent with a long period of latency. Studies have found latency periods of up to 40 years from exposure and diagnosis, and there is no upper limit on latency for bladder cancer. (Culp Report, p.11). Mr. Raymond was diagnosed with bladder cancer in 2021 at age 78. The age at diagnosis is in accordance with his long latency period and is reflective of the effect of his exposure to known carcinogens decades prior. Therefore, a consideration of Mr. Raymond's age alone as a risk factor is inappropriate, it can be considered in connection with other risk factors as an additive factor, but age alone is not a cause of his bladder cancer.

**Other Known Risk Factors for Bladder Cancer**

Other known risk factors which were not present in Mr. Raymond's medical or factual profile include,

1. Past treatment to some anticancer drugs and radiation

Cyclophosphamide and radiation typically given in childhood cancer carries an increased risk of bladder cancer. (Travis et al. 1995) I do not consider this to be a risk factor for Mr. Raymond as he had no past treatment involving anticancer drugs prior to being diagnosed with bladder cancer.

2. History of bladder infections.

History of bladder infections has been shown to increase the risk of bladder cancer. Patients with chronic irritation to the bladder such as recurrent urinary tract infections, bladder stones, and certain parasitic infections prevalent developing countries have an increased risk of bladder cancer (Halaseh 2022) However, Mr. Raymond did not have a history of bladder infections prior to his cancer diagnosis, so I do not consider it to be a cause.

3. Genetics/Family History.

There is evidence that family history of bladder cancer or colorectal cancer within the first-degree to second-degree is associated with increased risk of bladder cancer, as seen in Lynch syndrome disease genetic mutation.(van der Post et al. 2010) However, Mr. Raymond's family does not have a history of bladder cancer, colorectal cancer, or Lynch syndrome so I do not consider his family history or genetics to be a cause of his bladder cancer.

**Opinions**

After reviewing Mr. Raymond's personal and professional history, his exposure data, and consideration of the risk factors above I review and conclude the differentials for Mr. Raymond's bladder cancer below:

1) Exposure to water contaminated with TCE and Benzene was a risk and established a causal relationship with the development of his bladder cancer and is at least as likely as not a cause of his bladder cancer.
2) Mr. Raymond smoked 1 pack per day of cigarettes for 50 years. He quit smoking 8 years ago. As stated above, there is a 30% decrease in risk of bladder after 1-4 years of cessation. Even though his risk has gone down since his cessation, this is still part of his differential and may be additive to his water contamination.
3) The family history of bladder cancer carries a risk of developing bladder cancer. This is usually seen with genetic mutations and commonly seen in the Lynch syndrome. The Lynch Syndrome will typically manifest with family history of bladder and colorectal cancer. Mr. Raymond did not have a family history of bladder or colorectal cancer; thus, I conclude this differential is likely not a cause of his bladder cancer.
4) Herbicides are another known risk factor in the differential for bladder cancer. Although Mr. Raymond was in Vietnam during a period when Agent Orange was present, he did not give a history of Agent Orange exposure. Therefore in my opinion this is not considered a cause of in my etiology of Mr. Raymond's bladder cancer.
5) Recurrent Infections and chronic irritation are also a known risk factor. Mr. Raymond did not have a medical history of chronic infections prior to his diagnosis, and did not have a chronic catheter in his bladder and has not traveled to places where there is a high incidence of schistosomiasis and therefore chronic bladder infection, or irritation is not a cause of his etiology of bladder cancer.
6) Occupation Exposures to certain organic chemicals are associated with bladder cancer risk. Chemicals called aromatic amines, which are used in the dye, rubber, textile, and paint industries. Mr. Raymond worked for 30 years at a General Electric silicone production facility, primarily as an operator with little exposure to the silica itself, and when he was exposed, he wore protective equipment. Therefore, I conclude that Mr. Raymond's occupational exposure is not likely a cause of his bladder cancer.
7) Exposure to cytotoxic drugs, such as chemotherapy and radiation. Cyclophosphamide, a chemotherapy typically used to treat pediatric cancers, carries a risk of developing bladder cancer. Mr. Raymond did not have a history of cyclophosphamide or pelvic radiation, therefore neither are a cause of Mr. Raymond's bladder cancer.
8) Idiopathic – i.e. there is no known cause. Bladder cancer is a disease of toxic exposure that is rarely idiopathic. Approximately 82% of bladder cancer cases are attributable to modifiable risk factors. (Halaseh 2022) I have considered the potential of an idiopathic cause and ruled it out considering the presence of two common risk factors – occupational/environmental exposure and smoking.

*Opinion 1: Mr. Raymond's exposure to the water at Camp Lejeune was at least as likely as not a cause of his bladder cancer.*

Mr. Raymond was exposed to TCE and Benzene at levels that have been shown to be hazardous to human health, and this exposure is at least as likely as not a cause of Mr. Raymond's bladder cancer.

As addressed above, Mr. Raymond was exposed to known carcinogens (TCE and Benzene) that have been shown to be associated with bladder cancer. Mr. Raymond was exposed to these chemicals at Camp Lejeune, where he worked, lived, bathed, and drank contaminated water. Mr. Raymond encountered all three routes of chemical exposure – ingestion, inhalation, and dermal. His exposure was chronic, lasting approximately 662 days. As Dr. Gilbert addressed in her report, TCE can negatively affect the immune system's innate ability to search for and destroy mutated cells, thus promoting cancerous cells to grow. Mr. Raymond's combined exposures to both TCE and benzene heightened his risk. These chemicals are immunotoxic and lead to the development of bladder cancer by suppressing the body's immune system. Mr. Raymond's immune system never had time to recover from his chronic exposure.

*Opinion 2: Mr. Raymond's smoking history is at least as likely as not a cause of his bladder cancer, but not more likely than not the cause.*

Mr. Raymond's history of active and passive exposure to smoking is also at least as likely as not a cause of his bladder cancer. Smoking is a recognized risk factor for the development of bladder cancer. Mr. Raymond's cessation of smoking may have lessened his overall risk, but did not eliminate his risk entirely.

It is not possible to determine which exposure (water at Camp Lejeune or Tobacco Smoke) was more likely than not to be the cause of Mr. Raymond's bladder cancer. Although smoking is estimated to contribute to over 50% bladder tumors, it is also the easiest to control for when studying populations. People know when they are exposed to smoking but generally do not know when they are environmentally exposed to carcinogens. As a result, smoking is not a stronger association, only a more studied exposure. In fact, many epidemiological studies control for smoking and still see an increase in bladder cancer diagnoses. Hadkhale et al. (2017) explained their study's process for addressing the recognized connection between bladder cancer and smoking: "If the risk of lung cancer in a given occupation is elevated, and there are no other work-related exposures than smoking, then the risk of bladder cancer should also be elevated due to smoking…The RRs for bladder cancer clearly differ from this pattern…Though smoking is a well-established risk factor for bladder cancer, occupational differences in bladder cancer risk do not appear to be solely due to smoking."

Tobacco smoke and the chemicals at Camp Lejeune are both genotoxic. Mr. Raymond started smoking when he was a marine at Camp Lejeune, the same time he was exposed to the contaminated water. Considering bladder cancer's long latency, once you are exposed you are always exposed. A long length of time from last exposure to diagnosis is not indicative of elimination of a risk. For example, former smokers experience the effects of smoking decades after cessation.

TCE and Benzene as well as tobacco smoke have highly reactive metabolites that are genotoxic. Both lead to exposures to the urothelial cells of the bladder as the body waits to process the toxins through the urinary system. These toxic exposures equally disrupt normal cell processes. Exposure to more than one carcinogen only amplifies the risk of cancer development. Each carcinogen likely causes different types of cell damage, creating more mutations and promoting more aggressive tumor growth. Combining certain environmental pollutants with tobacco smoke significantly raises the incidence of cancers.(Cani et al. 2023). The exposure to the water at Camp Lejeune and the tobacco smoking likely were additive or even synergistic but it is simply too difficult to determine now, which exposure was more likely the cause – for this reason, the standard as likely as not applies to both exposures equally as a cause of Mr. Raymond's bladder cancer.

**Additional Required Information**

A list of the prior cases in which I have testified as an export at trial or by deposition in the last four years is attached as an Appendix to this report. I am being compensated for my services in this case at an hourly rate of $1,000 for review and analysis of medical records and other information, examinations, correspondence, preparation of a written report, consultation with legal counsel and deposition testimony.

**APPENDIX A: Curriculum Vitae**

# VINCENT MICHAEL BIVINS, MD FACS

## CONTACT INFORMATION


PHONE:
205-516-1578

ADDRESS:
3485 Independence Drive
Homewood, AL 35209

WEBSITE:
https://www.urologycentersalabama.com

EMAIL:
vbivins@urologyal.com


## LICENSURES

- Medical: State of Alabama: # 24677
- American Board of Urology, #14517, Certified 2004, 2014

## PROFESSIONAL MEMBERSHIPS & SOCIETIES

- American Urology Society
- Society of Urology Oncology
- Endourology Society
- National Medical Association
- Jefferson County Medical Society

## HONORS & AWARDS

- Leadership Alabama, Class 2013
- Leadership Birmingham, Class 2010
- UAB Physician's Assistant Outstanding Teacher Award 2020

## EDUCATION

| | |
|---|---|
| •**American College of Surgeons**, Fellow | 2019 |
| •**University of Washington Medical Center**, Urology Oncology Fellowship | 2002 |
| •**University of Oklahoma Health Sciences Center**, Urology Residency | 2001 |
| •**Vanderbilt Medical Center**, General Surgery Internship | 1997 |
| •**UAB School of Medicine**, MD | 1996 |
| •**University of Alabama**, BS in Microbiology | 1991 |

## WORK EXPERIENCE

| | |
|---|---|
| •**Urology Centers of Alabama**, Uro-Oncologist | 2002 – Current |
| •**2nd Medical Group** Maxwell AFB, Major, General Medical Officer | 2004 – 2008 |
| •**507th Medical Group**, Tinker AFB Army National Guard, Medical Detachment 5, Medical Officer, Birmingham, AL | 2002 – 2004<br>1992 – 1996 |
| •**United States Army Reserve**, Ordinance Officer 900th Maintenance Company Brundidge, AL | 1989 – 1992 |

## LEADERSHIP

| | |
|---|---|
| **President**, Urology Centers of Alabama, Birmingham, AL | 2020 – Current |
| **CEO**, Urology Centers of Alabama, Birmingham, AL | 12/2022 – Current |
| **President**, Medical Executive Committee and Medical Staff Baptist Princeton | 1/2024 - Current |
| **Chairman**, Department of Surgery, Brookwood Baptist Medical Center Princeton | 2016 – 2022 |
| **President**, Alabama Urology Network | Current |
| **President**, University of Alabama Medical Alumni Assoc. | Current |
| **Vice Chairman**, Brookwood Baptist Medical Center, Princeton | Current |
| **Diversity and Inclusion Committee**, LUGPA | Current |
| **Director**, Van Scott Cancer Center | 2010 – 2018 |
| **Preceptor**, UAB School of Physician Assistance | 2015 – Current |
| **Board Member**, Honor's College, University of Alabama | Current |
| **Member**, President's Cabinet, University of Alabama | Current |
| **Board Member**, YMCA of Greater Birmingham | 2010 – 2015 |
| **Board Member**, American Cancer Society, Jefferson County | 2008 – 2014 |
| **Board Member**, Medical Association of State of Alabama | Current |
| **Committee Member**, State Health Planning Committee | 2010 – 2014 |
| **Admissions Committee**, UAB School of Medicine, | 2005 – 2010 |

# VINCENT MICHAEL BIVINS, MD FACS

## CONTACT INFORMATION


PHONE:
205-516-1578

ADDRESS:
3485 Independence Drive
Homewood, AL 35209

WEBSITE:
https://www.urologycentersalabama.com

EMAIL:
vbivins@urologyal.com


## LICENSURES

- Medical: State of Alabama: # 24677
- American Board of Urology, #14517, Certified 2004, 2014

## PROFESSIONAL MEMBERSHIPS & SOCIETIES

- American Urology Society
- Society of Urology Oncology
- Endourology Society
- National Medical Association
- Jefferson County Medical Society

## HONORS & AWARDS

- Leadership Alabama, Class 2013
- Leadership Birmingham, Class 2010
- UAB Physician's Assistant Outstanding Teacher Award 2020

## EDUCATION

| | |
|---|---|
| •**American College of Surgeons**, Fellow | 2019 |
| •**University of Washington Medical Center**, Urology Oncology Fellowship | 2002 |
| •**University of Oklahoma Health Sciences Center**, Urology Residency | 2001 |
| •**Vanderbilt Medical Center**, General Surgery Internship | 1997 |
| •**UAB School of Medicine**, MD | 1996 |
| •**University of Alabama**, BS in Microbiology | 1991 |

## WORK EXPERIENCE

| | |
|---|---|
| •**Urology Centers of Alabama**, Uro-Oncologist | 2002 – Current |
| •**2nd Medical Group** | |
| Maxwell AFB, Major, General Medical Officer | 2004 – 2008 |
| •**507th Medical Group**, | 2002 – 2004 |
| Tinker AFB Army National Guard, Medical Detachment 5, | |
| Medical Officer, Birmingham, AL | 1992 – 1996 |
| •**United States Army Reserve**, | |
| Ordinance Officer 900th Maintenance Company | 1989 – 1992 |
| Brundidge, AL | |

## LEADERSHIP

| | |
|---|---|
| **President**, Urology Centers of Alabama, Birmingham, AL | 2020 – Current |
| **CEO**, Urology Centers of Alabama, Birmingham, AL | 12/2022 – Current |
| **President**, Medical Executive Committee and Medical Staff Baptist Princeton | 1/2024 - Current |
| **Chairman**, Department of Surgery, Brookwood Baptist Medical Center Princeton | 2016 – 2022 |
| **President**, Alabama Urology Network | Current |
| **President**, University of Alabama Medical Alumni Assoc. | Current |
| **Vice Chairman**, Brookwood Baptist Medical Center, Princeton | Current |
| **Diversity and Inclusion Committee**, LUGPA | Current |
| **Director**, Van Scott Cancer Center | 2010 – 2018 |
| **Preceptor**, UAB School of Physician Assistance | 2015 – Current |
| **Board Member**, Honor's College, University of Alabama | Current |
| **Member**, President's Cabinet, University of Alabama | Current |
| **Board Member**, YMCA of Greater Birmingham | 2010 – 2015 |
| **Board Member**, American Cancer Society, Jefferson County | 2008 – 2014 |
| **Board Member**, Medical Association of State of Alabama | Current |
| **Committee Member**, State Health Planning Committee | 2010 – 2014 |
| **Admissions Committee**, UAB School of Medicine, | 2005 – 2010 |

**APPENDIX B: Prior Testimony**

**APPENDIX B: Prior Testimony**

**Vincent Michael Bivins, MD FACS**
**3485 Independence Drive**
**Homewood, AL 35209**

Below is a list of the cases in which I have been retained and provided expert testimony at trial or in a deposition in the last four years.

*Wesley Rape and Bridgette Rape v. Dr. Mamoun Pacha, Coosa Valley Urology, PC, et. al.*;
Case No. CV-2016-900180
Circuit Court of Talladega County, Alabama
Trial Testimony

*Rushunda Williams, as Administratrix of the Estate of Zamora Dudley, Deceased vs. Jackson Hospital & Clinic, Inc., Jackson et al*.
Case No. CV-16-900773
Circuit Court of Montgomery County, Alabama
Deposition Testimony

*Cameron Murphy v. Jackson Hospital & Clinic, Inc.; Margaret Vereb, M.D*.
Case No. CV-13-900248
Circuit Court of Montgomery County, Alabama
Trial Testimony