Exhibit 438

Report of Walter Stadler, M.D., FACP

Mousser v. United States, 7:23-cv-00667 (E.D.N.C.)

Walter Stadler

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Walter Stadler, M.D., FACP

April 8, 2025

Contents

I. Expert Background and Qualifications

I am a genitourinary oncologist focused on care and treatment of patients with kidney, bladder, prostate, and testicular cancer. Full details of my academic and clinical qualification are detailed in my CV. Briefly, I received my MD from the Yale University School of Medicine in 1988, followed by an internship/residency at the University of Chicago affiliate Michael Reese Hospital and then a fellowship in Hematology/Oncology at the University of Chicago that I completed in 1994. I have been a faculty member at the University of Chicago for most of my career but have recently taken a position as Chief Clinical Officer for City of Hope, Chicago where I will continue my clinical role as genitourinary oncologist as well as my academic role conducting and leading genitourinary cancer clinical trials. I will also take on administrative responsibilities for clinical operations. I have held a number of additional administrative roles including Section Chief for Hematology/Oncology, Deputy Director for the University of Chicago Comprehensive Cancer Center and Associate Dean for Clinical Science Research, Clinical Trials.

I have conducted research on novel treatments and management of patients with genitourinary cancer and have authored over 250 peer-reviewed original manuscripts and over 120 reviews, chapters, and commentaries in this field (see CV, Attachment A). I have specifically contributed to the development of gemcitabine in bladder cancer, vascular endothelial growth factor receptor (VEGFR) inhibitors in renal cancer, and hormonal therapies in prostate cancer. Relevant to my opinions here is my role as Bladder Cancer Section Editor for the standard American College of Surgeons staging manual (CV references 104 – 107, pg 34). I routinely give presentations at national and international meetings regarding these malignancies.

From a clinical perspective, I continue to consult and manage patients with these diseases for approximately 30% of my time generating greater than the 65th percentile in prorated relative value units (RVU's) for my specialty (i.e. corrected for a full-time clinician, my clinical activity is equal to or greater than 65% of American oncologists). I have been recognized as a clinical expert through Castle Connolly Top Doctors every year since 2007.

I have testified as an expert witness at trial or deposition in the past four years only in support of Sandoz and other generic companies in their effort to invalidate Sanofi cabazitaxel patents (*Sanofi-Aventis U.S. LLC v. Sandoz Inc.*, Civ. No. 20-804-RGA, D. Del.). My hourly rate for my work here is $750/hr.

II. Basis of Opinions

My opinions are based on my extensive experience, standard guidelines from the National Comprehensive Cancer Network (NCCN), as well as generally accepted medical literature. For much of the latter, and unless referenced otherwise, I used information summarized in available textbooks including UpToDate, which is the most widely referenced source for practicing clinicians and for which I have acted as an Editor, and Holland-Frei Cancer Medicine, 10th edition, one of the major textbooks of oncology for which I also contributed a chapter. Other specific literature references are in the detailed report below and listed under References.

I have reviewed case and medical records provided by counsel including military service records, medical provider notes, and the deposition of plaintiff (1/19/2024), his wife (3/8/2024), and his employer Richard Mercer (8/1/2024), as well as Dr. Flood (8/7/2024), Dr. Mueller (6/12/2024), and Dr. Rockwood (7/24/2024). I have also reviewed and rely upon the United States's general causation reports

(Goodman Report; Shields Report, Lipscomb report), the United States's exposure report (LaKind Report), and the United States's risk assessment report (Bailey Report). Finally, I have reviewed the expert depositions of Drs. Bove and Savitz.

A complete list of the facts and data I considered are listed in Attachment B, which will be provided separately.

I reserve the right to supplement the opinions offered here as appropriate, including if new information is made available to me.

III. Summary of Opinion

It is my opinion that Mr. Mousser's upper tract urothelial cancer (UTUC) was most likely caused by a combination of smoking history and chronic inflammation and that it was unlikely caused by exposure to water at Camp Lejeune. It is furthermore my opinion that Mr. Mousser is most likely cured of his cancer and the consequences of treatment are minimal. More specifically, the nephroureterectomy has not led to any significant impacts on his kidney function, nor has it materially impacted the risk of future renal dysfunction. In terms of his future medical care, Mr. Mousser will reasonably require an additional Urology visit, cystoscopy, and CT scan as of 4/8/25. His UTUC will not affect his life expectancy.

IV. Clinical Background

A. Oncogenesis

Generally, cancer is a disease that arises from a series of genetic errors, also known as mutations, within normal tissue. Most normal tissue typically regenerates through division and replication of normal cells. While this almost always occurs faithfully, amongst the billions of base pairs making up the DNA of a human cell and the trillions of cells in the human body, errors do occur. While the vast majority of these genetic mutations are corrected or eliminated, occasionally a cell with such mutations persists and can then go on to develop additional genetic mutations, which leads to ongoing replication and division without appropriate controls. Additional mutations and alterations can then give the cells the ability to move (or metastasize) to other areas of the body. Notably, there are a number of biological processes, including the immune system, that can eliminate these mutated cells. The complex interplay between accumulation of mutations and failure to eliminate mutated cells is scientifically known as oncogenesis and in patients is the disease known as cancer (see for example (Hanahan and Weinberg)).

It follows that anything that increases the frequency of mutations or inhibits the corrective mechanisms increases the risk for cancer. The single most common factor is age. Errors in cell replication are rare and generally must accumulate to cause cancer in a patient and this simply takes time. Additionally, error correction mechanisms and the immune system become less robust as we grow older.

Exposure to certain environmental toxins known as carcinogens, the most relevant being exposure to tobacco smoke, can also increase the risk of cancer. Toxin exposure can increase the rate of genetic mutations and thus the risk of developing a cancer. Importantly, it is both the amount of toxin exposure (typically known as "dose") and the duration of exposure that determines both the odds of developing cancer causing mutations and the development of cancer (Hofseth, Weston, and Harris). The science of

toxin exposure and subsequent risk of developing cancer is generally conducted by epidemiologists and toxicologists.

While toxin exposure can increase the risk of cancer, it is important to specify that this simply increases the odds of developing this disease; not all exposed individuals will develop cancer and most individuals with cancer will not have a known exposure history (see Goodman report for further details).

Some patients are born with, or inherit, certain genetic abnormalities that make them more likely to develop a cancer. These genetic abnormalities, known as germ-line mutations, are typically tumor suppressor genes whose deletion is mechanistically related to oncogenesis. More importantly, these inherited predisposition syndromes make it much more likely that a mutated cell can accumulate the additional necessary mutations to become a clinical cancer. For example, patients that inherit a mutation of the Von-Hippel-Lindau (VHL) gene have up to a 45% risk of developing renal cancer in their lifetime (Binderup et al. 2022) as opposed to a 1.4% – 2.3% risk in the general American population (Siegel, Giaquinto, and Jemal 2024). Of note these predisposition syndromes are typically organ specific insomuch as patients with these inborn errors are more susceptible to some, but not necessarily all cancers. For example, patients born with a mutation in the VHL gene are at a higher risk of developing renal cancer, hemangioblastomas, pheochromocytomas, and pancreatic neuroendocrine tumors, but not other cancers such as upper tract urothelial cancer. Patients with such predisposition syndromes often develop multiple cancers in the same organ, with a classic example once again being the multiple independent clear cell renal cancers in patients with germ-line VHL mutations.

Finally, there are multiple families with specific cancers in multiple family members, without any common environmental exposure, but in whom a germline mutation has not been discovered. Therefore, there are almost certainly undiscovered and undescribed genetic predisposition mutations or syndromes and discovery of new cancer predisposing genes is an active area of scientific research (see for example (Roberts et al. 2016).

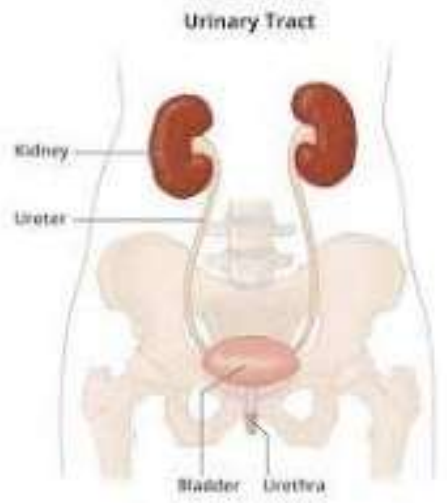
Chronic infection or inflammation has also been consistently linked to a number of different cancers. The scientific explanation for this is a bit more obscure, but two explanations have been offered (Zhao et al. 2021). First, the immune system produces a number of molecules designed to kill invading organisms and these substances can damage or mutate normal cells as well. As with toxin exposure the duration of the inflammation is critical. Second, the presence of chronic inflammation within a tissue or organ paradoxically leads to an immunosuppressive microenvironment, or in other words an environment in which the immune system is unable to function normally. This is critical because there is increasing evidence that the immune system is largely responsible for eliminating mutated cells that are destined to form a cancer. Thus, in a chronically inflamed tissue not only is the aforementioned rate and risk of developing cancer-causing mutations increased, the body's ability to eliminate such cells is compromised.

The above discussion emphasizes that development of cancer in any one individual is a random event that can occur in anyone. Certain clinical conditions or exposures can increase the odds of this random event (i.e. "cancer") occurring, but no one individual is absolutely protected or predestined to develop a cancer. Additionally, the increased odds for developing a cancer from any one condition can be low, modest or very high. Importantly, the difference between increased odds and absolute risk must be recognized. For example, if the odds of developing a specific cancer are 1/1000, even a doubling of risk means that the odds now are 2/1000 (see also Goodman report). Further, the studies determining these

risks generally apply to a population and do not necessarily reflect the increased risk in an individual patient, and more importantly do not necessarily reflect whether that factor (like a toxic exposure) is causative in that specific patient.

Finally, given these issues it must be recognized that the exact etiology, or even the most likely etiology, of any specific cancer in any one individual can be difficult or impossible to ascertain. As a result, the cause for the vast majority of cancers is idiopathic (i.e. unknown).

B. Kidney (Upper Tract Urothelial) Cancer



Kidney cancer is generally described as a single entity in broad epidemiologic studies (e.g. (Siegel, Giaquinto, and Jemal 2024)); however, it is critical to note that this broad description represents a variety of distinct malignancies composed of multiple histologic subtypes arising from the kidney. The most common are renal carcinoma and upper tract urothelial cancer (UTUC). The latter also goes by the moniker of transitional cell carcinoma of the upper tract. All urothelial carcinomas arise from the epithelial or surface lining of the renal pelvis (internal portion of the kidney), ureters (conduit from kidney to the bladder), bladder, and urethra (conduit from bladder to external world) (See figure). As such, there is a close etiologic and therapeutic relationship between all urothelial cancers, which are oftentimes described as unique due to specific surgical and local therapy options utilized when the disease is localized and has not spread or metastasized. In fact, clinicians and biologists have described cancer development anywhere in the urothelial tract and especially the development of multiple tumors in the urothelial tract as independent tumors due to a "field effect" from toxic exposures, especially smoking.

Since UTUC biology and development is more closely related to urothelial cancers of the ureter and bladder, and since many epidemiologic studies do not separate UTUC from renal cancer when assessing risk factors for kidney tumors, frequent reference to data from bladder cancer will be made here.

The global incidence of UTUC is poorly documented, but in the United States the age-adjusted annual incidence rate is 1.15/100,000 person-years. This is in comparison to global incidence rates for bladder cancer of 9.5/100,000 in males and approximately 2.4/100,000 in females. The median age of diagnosis for bladder cancers is 69 years in males and 71 years in females (Scosyrev, Yao, and Messing 2009) and is similar for UTUC. Globally, bladder cancer is the ninth and nineteenth leading cause of cancer-related deaths for males and females, respectively (Cancer 2024). Given the nearly 9-fold lower incidence rate for UTUC, and similar therapy outcomes between bladder cancer and UTUC, global mortality rates for UTUC are much lower.

The major risk factors for all urothelial cancers that I have considered are (1) tobacco smoking, (2) chronic inflammation including chronic infections, (3) industrial occupational exposure to polycyclic aromatic hydrocarbons, (4) genomic predisposition, (5) obesity, (6) drinking water chlorination, (7) phenacetin exposure, and (8) exposure to the phytotoxin aristocholic acid.

1) Tobacco smoking. Tobacco smoking is the most important etiology for urothelial cancer, for which the relative risk in smokers is 4.06 [95% CI, 3.66-4.50] (Freedman et al. 2011) Notably, an elevated risk persists even in smokers who have quit with a relative risk for former smokers who have quit more than 10 years previously in comparison to never smokers being 2.22 [CI 2.03-2.44] (Freedman et al. 2011). Smoking is associated with approximately 50% of all urothelial cancers.
2) Chronic inflammation or infection. Chronic infection or inflammation is considered a common etiologic factor in many cancers and is especially well categorized as an etiology for urothelial cancer. For bladder urothelial cancer this has been characterized in paraplegic patients with recurrent urinary tract infections due to loss of bladder control (Bejany, Lockhart, and Rhamy 1987) and in other countries in patients with chronic infection from the parasite schistosoma. For UTUC chronic kidney stones, which can cause both acute and chronic infections, have been specifically associated with UTUC with a relative risk that approximates 2.5 [95% CI = 1.8-3.3) (Chow et al. 1997).
3) Industrial exposure to polycyclic hydrocarbons. An increased risk of urothelial cancer has been demonstrated in metal workers, painters, rubber industry workers, and jobs that involve manufacture of carpets, paints, plastics, and industrial chemicals. Long term extensive exposure to polycyclic aromatic hydrocarbons, including 2-naphthylamine, benzidine, and their precursors or derivatives are thought to be an important contributor to these occupational risk factors (Pira et al. 2010). See also expert report of Dr. Julie Goodman.
4) Genomic predisposition. Genome-wide association studies (GWAS) have identified more than 20 genetic variants associated with bladder cancer risk and this risk appears to be increased by interactions between smoking status and genetic variants, especially in the NAT2 gene (Garcia-Closas et al. 2005). More recently, a polygenic risk score (PRS) based on 24 independent GWAS markers has been identified to be associated with an approximately fourfold difference in the lifetime risk of bladder cancer (Koutros et al. 2023). Germline Lynch syndrome (hereditary non-polyposis colon cancer) has been specifically associated with UTUC.
5) Obesity: Obesity has not consistently been demonstrated to be a risk factor for UTUC; although, there has been a suggestion that presence of a metabolic syndrome, defined as the presence of three or more of the following conditions: central adiposity or elevated waist circumference, impaired fasting glucose (including type II diabetes), high triglycerides, low HDL cholesterol, and hypertension, may be associated with development of UTUC (Lu et al. 2020).
6) Drinking water chlorination. Several studies have demonstrated exposure to chlorinated drinking water may increase the risk of bladder urothelial cancer with exposure to trihalomethane thought to be the etiologic agent. An overall relative risk of 1.4 [95% CI: 1.10-1.81] for those with exposure of 35 or more years in comparison to those with an exposure of less than 10 years has been reported (King and Marrett 1996). It is not known whether exposure to chlorinated drinking water increases the risk for UTUC.
7) Phenacetin use. High use and abuse of the analgesic medication phenacetin has been associated with UTUC, but this medication is not routinely available in the United States.
8) Exposure to the phytotoxin aristolochic acid. High exposure to this known mutagen, typically through ingestion of botanical supplements, can lead to nephritis, kidney failure, and subsequent UTUC.

It is estimated that approximately 82% of urothelial cancers are due to modifiable exposure related risk factors, especially tobacco smoking, which is estimated to account for 50% of all urothelial cancer (Al-Zalabani et al. 2016). It is, however, difficult or impossible to determine whether any specific cancer in an individual is due to any specific exposure or inflammatory cause.

The most common presenting symptom for UTUC is gross hematuria, i.e. visible blood in the urine. Notably, there is a long list of possible causes for gross hematuria, including kidney stones, glomerulonephritis (a specific class of non-malignant kidney diseases), infection, and exercise induced hematuria such that the majority of individuals with hematuria do not have a urinary tract malignancy. Flank pain or symptoms from cancer that has metastasized (weight loss, pain at a metastatic site, etc.) are other presenting symptoms.

Low grade upper tract urothelial cancer can be treated with endoscopic (instrument placed into the renal pelvis) resection or ablation or instillation of a mitomycin ger. Treatment of the more common high grade upper tract urothelial cancer typically involves nephroureterectomy (removal of the kidney, ureter, and a "cuff" of the bladder at the site of ureter insertion). Partial nephrectomies, or removal of only a portion of the kidney, while occasionally utilized for renal cancer, is generally not appropriate for UTUC since this disease generally impacts the entire collecting system of the kidney, which cannot be removed while sparing renal function. Additional chemotherapy can be administered prior to (neoadjuvant) or following (adjuvant) surgery; although, the value is somewhat controversial insomuch as it has been difficult to prove an overall survival advantage, and thus this is not always done.

Following nephroureterectomy, recurrence is possible in the bladder, presumably due to cancer cells washed into the bladder from the kidney that occurred prior to the nephroureterectomy, or systemically, which is termed metastatic recurrence. Post-operative installation of chemotherapy directly into the bladder following nephroureterectomy may decrease the risk of recurrent cancer in the bladder. The highest risk for recurrence is within then the first 2-3 years and declines markedly thereafter with recurrences beyond 5 years extremely rare. Recommended standard follow up and monitoring of UTUC following nephroureterectomy is thus CT scans of the chest, abdomen and pelvis every 6 months for 3 years followed by annual scans up to 5 years post-surgery. Cystoscopy (visual evaluation of the bladder) is recommended every 3 months for the first year and then at the same frequency as the CT scans thereafter.

Prognosis and survival of UTUC is highly dependent on stage. Staging is based on both extent and size of the tumor within the kidney, presence of spread to lymph nodes, and presence of metastatic disease (TNM system).

TNM classification 2017 for upper tract urothelial cell carcinoma		
T - Primary tumour		
TX	Primary tumour cannot be assessed	
T0	No evidence of primary tumour	
	Ta	Non-invasive papillary carcinoma
	Tis	Carcinoma *in situ*
T1	Tumour invades subepithelial connective tissue	
T2	Tumour invades muscularis	

T3	(Renal pelvis) Tumour invades beyond muscularis into peripelvic fat or renal parenchyma (Ureter) Tumour invades beyond muscularis into periureteric fat
T4	Tumour invades adjacent organs or through the kidney into perinephric fat
N - Regional lymph nodes	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single lymph node 2 cm or less in the greatest dimension
N2	Metastasis in a single lymph node more than 2 cm, or multiple lymph nodes
M - Distant metastasis	
M0	No distant metastasis
M1	Distant metastasis

Adapted from the AJCC Cancer Staging Manual, Eighth Edition (2017) published by Springer International Publishing. Corrected at 4th printing, 2018.

The five-year cancer-specific survival rates for patients with pT0/Ta/Tis, pT1, pT2, pT3, and pT4 disease are 94, 91, 75, 54, and 12 percent. Grade has historically been categorized on a 3-pt scale but is currently generally broken down into low grade (historical grade 1) and high grade (historical grade 2 and 3) cancers. Ta tumors are the only ones that can be low grade, which confers a better prognosis than high grade Ta. The risk of cancer recurrence drops markedly at 3 years post-resection with the overall risk of recurrence for individuals who have not recurred by then being less than 10%; patients who have not recurred by 5 years post resection have a risk of recurrence of less than 5% (Margulis et al. 2009).

A potential major long-term complication of nephroureterectomy is decreased renal function. Renal dysfunction, or chronic kidney disease (CKD), is generally graded by the estimated glomerular filtration rate (GFR), which is based on serum creatinine levels:

- G1 = ≥ 90 ml/min/1.73 m^2 (normal)
- G2 = 60-89 ml/min/1.73 m^2 (mildly decreased)
- G3a = 45-59 ml/min/1.73 m^2 (mildy to moderately decreased)
- G3b = 30-44 ml/min/1.73 m^2 (moderately to severely decreased)
- G4 = 15-29 ml/min/1.73 m^2 (severely decreased)
- G5 = <15 ml/min/1.73 m^2 (kidney failure)

The implications and outcomes for patients with CKD include hypertension, fluid retention, electrolyte imbalances and in the worst-case scenario need for dialysis. Overall, and depending on the patient's age, the relative risk for all-cause mortality among patients with isolated G3a CKD is 1.2 (CI: 1.0 – 1.5) – 1.9

(CI: 1.4 – 2.5), with a specific relative risk of cardiovascular mortality of 1.3 (CI: 0.6 – 3.2) – 1.4 (CI: 1.2 – 1.8), and risk of developing the need for dialysis of 3.1 (CI: 1.1 – 8.3) – 3.4 (CI: 1.6 – 7.2), which for the former reflects an increased risk from 48/10,000 to 65/10,000 and the latter reflects an increased risk from 5.7/10,000 to approximately 18.6/10,000 (Levey et al. 2011).

Notwithstanding the aforementioned, the most common causes for CKD are hypertension, diabetes, hypercholesterolemia and smoking. Chronic non-steroidal anti-inflammatory drug use, with medications such as ibuprofen, may also increase the risk of chronic renal disease. However, in patients with modest renal dysfunction, it is only high dose use, on the order of 3.5 or more 200mg ibuprofen pills daily (i.e. 700 mg or more), that is significantly associated worsening renal function (Gooch et al. 2007).

V. Overview of Plaintiff Case

Plaintiff Mr. Mousser is a veteran who first developed genitourinary issues when he experienced gross hematuria on 11/25/83 while stationed in Beirut, Lebanon approximately 16 months after initial enlistment and 13 months after residency at Camp Lejeune (see LaKind report). Work up with cystoscopy in Landstuhl, Germany and Naples, Italy in 12/83 reportedly revealed an unremarkable intravenous pyelogram (IVP) and evidence of cystitis. The hematuria was ascribed to possible bacterial cystitis and a follow up cystoscopy in 1/84 was unremarkable. He experienced recurrent flank pain and hematuria on 11/7/13 and was admitted to Peterson Regional Medical Center; although, details of the work up are unavailable. A urology note from 4/25/18 (HCM Medical Group, Michael Speck) notes that this was a 6-month follow up visit for hematuria that occurred on or around 9/17. At this time, urine cytology noted only rare atypical urothelial cells, a CT urogram was negative for upper tract lesions but a 2 cm right renal parapelvic cyst was noted, and a cystoscopy revealed a normal bladder and urethra with no tumors or stones.

It is not clear how frequently Mr. Mousser experienced hematuria between 1984 and 2013, or thereafter; however, he did present with recurrent pain and hematuria on 8/3/20. As part of this hematuria work up he underwent a 24-hour urine protein estimation on 8/21/20 that showed 2112 mg of protein, which is a high level of proteinuria. At that time he also had a glomerulonephritis work-up (C3/C4, Hep C, Hep B, Anti PR3/Anti MPO Ab's, rheumatoid factor, ANA, anti-GBM, cytoplasmic antibody, basilar antibody, SPEP) that was unremarkable. Follow up CT urogram at that time revealed a 3.7 cm right renal hilar mass highly suspicious for a urothelial cancer. Cystoscopy was unremarkable, but ureteroscopic brushings revealed a urothelial cancer. He underwent a robotic assisted right nephroureterectomy on 10/20/20, including intravesical gemcitabine administration, without significant complications. Pathology report revealed:

DIAGNOSIS:

A. LYMPH NODE, RIGHT, INTERNAL ILIAC, EXCISION:
FIVE BENIGN LYMPH NODES NEGATIVE FOR MALIGNANCY (0/5).
B. KIDNEY, RIGHT, NEPHROURETERECTOMY:
NON-INVASIVE HIGH GRADE PAPITLLARY UROTHELIAL CARCINOMA PTA PNO (SEE CANCER CASE SUMMARY BELOW).
NEGATIVE RESECTION MARGIN.
SURGICAL PATHOLOGY CANCER CASE SUMMARY
RENAL PELVIS AND URETER: RESECTION SPECIMEN

PROCEDURE: NEPHROURETERECTOMY
SPECIMEN LATERALITY: RIGHT
TUMOR
TUMOR SITE: RENAL PELVIS
HISTOLOGIC TYPE: PAPILLARY UROTHELIAL CARCINOMA, NONINVASIVE
HISTOLOGIC GRADE: HIGH-GRADE
TUMOR EXTENSION: NONINVASIVE PAPLLLARY CARCINOMA
MARGINS: UNINVOLVED BY INVASIVE CARCINOMA AND CARCINOMA IN SITU/NONINVASiVE UROTHELIAL CARCINOMA
LYMPH NODES
NUMBER OF LYMPH NODES INVOLVED: 0
NUMBER OF LYMPH NODES EXAMINED: 5
PATHOLOGIC STAGE CLASSIFICATION (PTNM, AJCC 8THEDITION)
PRIMARY TUMOR (PT): PTa
REGIONAL LYMPH NODES (PN): PNO
ADDITIONAL FINDINGS
PATHOLOGIC FINDINGS IN IPSILATERAL NONNEOPLASTIC RENAL TISSUE: NEPHROSCLEROSIS, CHRONIC PYELONEPHRITIS, HYDROURETER (IRON AND FONTANA MASSON STAINS PERFORMED)

This pathology is considered to be a Stage 1 cancer with an approximately 95% cure rate.

From a renal function perspective, he had a creatinine of 1.1 on 8 October 2020 and 2 days following surgery creatinine rose to 1.2 and further increased to 1.6 on 7/28/21. Mr. Mousser's renal function continued to decrease until March 2022, when he had a creatinine level of 1.9 and a GFR of 36. In his deposition, Dr. Flood attributed this further decline to Mr. Mousser's blood pressure medication (Lisinopril) and reduced blood flow to his kidney rather than intrinsic kidney disease. After lowering the dose of this medication, Mr. Mousser's creatinine level rebounded to 1.3 as of November 2023. Mr. Mousser has continued to have creatinine levels and GFR scores representative of CKD stage 2/3a. His most recent results in January 2025 indicate a GFR of 63 and a creatinine level of 1.3.

Additionally, follow up Nephrology evaluation in 4/21, post resection of his urothelial cancer, suggested that Mr. Mousser may have an underlying "loin pain-hematuria" syndrome or an underlying IGA nephropathy, but since renal tissue from the nephroureterectomy was not sent for light microscopy/immunofluorescence or electron microscopy a definitive diagnosis could not be made. Further work up has not been contemplated, because prior proteinuria, which was likely related to the underlying cancer at the time, has resolved and no further hematuria has been noted.

Mr. Mousser does have a smoking history, but the medical record is somewhat inconsistent on the extent, and he testified that he smoked only while in the Marines. Mr. Mousser's former employer and good friend, Mr. Richard Mercer, also testified that Mr. Mousser smoked daily for approximately a year when he worked at National Care Sales in 2012. Mr. Mousser also has a history of being overweight (BMI 28.7), hyperlipidemia, diabetes, and hypertension.

In February 2025, Mr. Mousser reported being diagnosed with a new bladder cancer. This new cancer is not addressed in Plaintiff's experts' reports and limited medical records describing the new cancer have been made available to me. My opinions are limited to Mr. Mousser's UTUC described above. I reserve

the right to supplement these opinions based on new information, including information related to any new malignancy.

VI. Opinion

In order to assess likely and unlikely causes for any one individual's cancer I assess whether any of the known causes apply to the individual, whether that individual's cancer presentation is typical for known causes, and to what degree other potential causes need to be considered in this context. Additionally, I consider the complex multi-step and somewhat random nature of oncogenesis discussed above, as well as the increased risk attributable to and the clinical significance of any known potential causative factor. For example, a known factor, such as smoking, is likelier to be a cause if that factor is persistent and long standing. Conversely, a known factor might still be unlikely to be a cause if exposure is brief and clinically minimal.

Given the medical history and aforementioned discussion regarding UTUC etiology, it is my opinion that Mr. Mousser's UTUC was more likely than not due to a combination of smoking history and chronic upper tract inflammation and was less likely than not due to any exposure to Camp Lejeune drinking water. This is based on evaluation of the aforementioned etiologic factors for UTUC:

1) Smoking. As noted above, tobacco smoking accounts for 50% of all urothelial cancer, which is much more a smoking-related disease than RCC. Mr. Mousser was a smoker, especially while in the Marines, and the increased risk of urothelial cancer is not eliminated even when smoking is discontinued. Additionally, Mr. Mousser developed his cancer at age 57, which is well within the range of smoking related urothelial cancer.
2) Chronic inflammation or infection: The first episode of Mr. Mousser's intermittent hematuria occurred during his military service and far too soon after being stationed at Camp Lejeune for any exposure there to be a causative factor. As such there is clinical evidence for non-malignant kidney pathology present long before the diagnosis of UTUC. Furthermore, the pathology report described the kidney not involved by cancer to exhibit underlying nephrosclerosis and pyelonephritis, which suggests long standing renal inflammation, which a Nephrologist suggests may be due to an underlying "loin pain-hematuria" syndrome or an underlying IGA nephropathy. It is likely that this inflammation also contributed to Mr. Mousser's UTUC.
3) Industrial exposure to polycyclic hydrocarbons. There is no evidence for the long term extensive exposure to industrial chemicals or polycyclic aromatic hydrocarbons that has been linked to urothelial cancer
4) Genomic predisposition. There is no known familial or genetic predisposition for Mr. Mousser's cancer.
5) Obesity: While obesity has not been consistently demonstrated to be a risk factor for UTUC, it is associated with renal cancer and there is some suggestive evidence that an associated metabolic syndrome may have a causative role. Mr. Mousser was obese and did have hyperlipidemia, diabetes, and hypertension and thus by definition has a metabolic syndrome. Obesity may thus be contributory to Mr. Mousser's cancer
6) Drinking water chlorination. While it is impossible to rule out chlorinated drinking water as an etiologic factor in Mr. Mousser's UTUC, any such exposure would have occurred over decades and most of it not during residency at Camp LeJune.

7) Phenacetin use. This drug is generally not available in the United States and there is no evidence that Mr. Mousser used it.
8) Exposure to the phytotoxin aristolochic acid. This generally occurs through use of botanical supplements and there is no evidence of such use in Mr. Mousser's case.

Additionally, Mr. Mousser has not suffered any significant long term consequences of his UTUC. More specifically:

1) His post-operative renal function was only mildly abnormal, but with modification of his medications has stabilized with an estimated GFR of approximately 60, and the prior proteinuria, which was most likely due to the underlying cancer that was resected has resolved. As such, the increased risk of overall mortality, cardiovascular mortality, or end-stage renal failure requiring dialysis is essentially nil (Levey et al. 2011).
2) The biggest risks for any worsening renal function are his underlying diabetes, hypertension, and hypercholesterolemia, which are also much more significant risks for development of important medical manifestations such as cardiovascular morbidity/mortality, stroke, or peripheral vascular morbidity than the loss of a single kidney.
3) The aforementioned risks for worsening renal function can be mitigated through appropriate medical management of diabetes, hypertension, and hypercholesterolemia, which he is receiving.

Based on his 2020 UTUC diagnosis and nephroureterectomy, Mr. Mousser may reasonably require surveillance and follow-up care. Specifically, Mr. Mousser may require urology follow-up, including CT scans of the chest, abdomen and pelvis, for up to one more year (5 years post-surgery); he may also require cystoscopy at the same frequency.

As such, Mr. Mousser's UTUC is unlikely to be related to Camp Lejeune exposures and is far more likely than not related to prior smoking and possibly an undiagnosed chronic inflammatory condition of the right kidney. Furthermore, its successful treatment has minimal to no impacts on his current or future health status.

VII. Responses to Plaintiff's Experts

A. General Responses

It is my opinion that plaintiff's experts make some fundamental errors in their causation assessment. First, plaintiff's experts refer to the Bradford-Hill criteria assessing whether an association between exposure and subsequent health event (in this case UTUC) is likely to be causative. For example, Dr. Del Pizzo states "[t]he Bradford Hill considerations are employed here for structural analysis to determine wither this association with Mr. Mousser is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Mousser's kidney cancer." (Del Pizzo report at 18-19). These criteria, however, focus on potential causation within a ***population*** and do not necessarily apply to causation in an ***individual***. This becomes especially important when the relative risk associated with such an exposure is small and less than 2, which is acknowledged by plaintiff's experts even under a worst-case scenario. In other words, within even an exposed population most of the patients with a specific cancer would have gotten that cancer even in the absence of exposure. To thus attribute any single individual's

cancer to such an exposure, even if there are no other obvious known causative conditions, is not accurate.

Secondly, and relatedly, plaintiff's experts ignore the implications of the multi-step and random events for oncogenesis in general and renal cancer specifically. For example, Dr. Del Pizzo claims to have "analyzed all of the potential risk factors" in his differential diagnosis. (Del Pizzo report at 16). Similarly, Dr. Smith claims to have "considered all of the risk factors that are medically relevant for Mr. Mousser." (Smith report at 15). As I have noted and plaintiff's experts acknowledge, multiple genetic and other events must occur for a clinical cancer to develop and there is a certain randomness to each event and oncogenesis in general (a phenomenon known as "stochastic"). As such, clinicians like myself consider most cancers in any specific individual to be idiopathic, which is not even considered by plaintiff's experts. In other words, even if his smoking and chronic inflammation did not cause his UTUC as I opine, it is far more likely that Mr. Mousser's UTUC was idiopathic in origin than caused by a toxic exposure. Plaintiff's experts not only fail to convincingly "rule out" idiopathic cause; they ignore it altogether.

B. Specific Responses

I agree with plaintiff's experts that smoking-related UTUC risks decrease with smoking cessation. For example, Dr. Del Pizzo notes "a substantial reduction in risk for long-term former smokers." (Del Pizzo report at 16). Dr. Smith similarly notes that "[f]ormer smokers have lower risks compared to current smokers, with risk declining further over time after quitting." (Smith report at 14). As I explain above, however, an elevated risk persists even in smokers who have quit with a relative risk for former smokers who have quit more than 10 years previously in comparison to never smokers being 2.22 [CI 2.03-2.44] (Freedman et al. 2011).

Plaintiff's experts simply discount Mr. Mousser's smoking-related UTUC risk despite the fact that smoking is in fact associated with approximately 50% of all urothelial cancers (Freedman et al. 2011). I agree that smoking-related UTUC risk is dose-dependent. For example, Dr. Del Pizzo recognizes that "[s]tudies demonstrate a strong dose-dependent increase in risk associated with numbers of cigarettes smoked per day." (Del Pizzo report at 15). Nevertheless, to then simply discount the contribution of smoking when the record is somewhat inconsistent on the historical smoking history, does not reflect the literature on the role of smoking in urothelial cancer.

Dr. Smith correctly recognizes that "chronic inflammation and infections are recognized as risk factors for urothelial cancer," and that "it is not likely that the limited and intermittent bleeding Mr. Mousser experienced starting in 1983 was due to an existing cancer." (Smith report at 15). Dr. Cooper opines that "[f]or hematuria to have any association with cancer or to have been from cancer that exists, it would have had to be more significant than the history Mr. Mousser gave and the medical records show at those times when hematuria was noted." (Cooper report at 14). As I explain above, it is my opinion that the prior hematuria was due to an underlying inflammatory condition and not due to the cancer itself until shortly prior to diagnosis. While the exact nature of this inflammation is unclear, pathologic evaluation of the nephroureterectomy specimen revealed underlying nephrosclerosis and pyelonephritis, which suggests long standing renal inflammation. This conclusion is similar to that of treating nephrologist Sandipani Sandilya, MD, who opined on 8/11/21 that Mr. Mousser had and underlying "loin pain-hematuria" syndrome or an underlying IGA nephropathy.

I agree with plaintiff's experts that the care Mr. Mousser received was appropriate.

I disagree with Dr. Cooper's opinions that Mr. Mousser's future medical care likely includes renal replacement therapy. (Cooper report at 3). Dr. Cooper appears to base his assessment upon Mr. Mousser's eGFR following his nephroureterectomy. As I describe above, Mr. Mousser's more recent testing shows only mild impairments to kidney function. Indeed, Dr. Smith correctly describes Mr. Mousser's CKD as Stage 3a. (Smith report at 15). In the absence of ongoing proteinuria there is no significant increased risk of developing end stage renal disease and need for dialysis (Levey et al. 2011).

VIII. References

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