Exhibit 473

Expert Report of Harry Paul Erba, M.D., Ph.D.

Karen Marie Amsler v. United States
7:23-cv-00284
U.S. District Court for the Eastern District of North Carolina

Prepared By:

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Harry Paul Erba, M.D., Ph.D.

April 8, 2025

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Plaintiff: Karen Marie Amsler

Case: *Karen Marie Amsler v. United States*, No: 7:23-cv-00284 (U.S. District Court, Eastern District of North Carolina).

I. Executive Summary

I prepared this report in response to the United States' request for my opinion as a board-certified hematologist and oncologist on whether Ms. Karen Marie Amsler's acute lymphoblastic leukemia (ALL) with a genetic abnormality of her KMT2A gene was caused by her exposure to the water at the Camp Lejeune military base. I was also asked to comment on Dr. Lukasz P. Gondek's February 7, 2025 report.

In formulating my opinions in this case, I have determined the following:

1. Based on the biology of ALL associated with KMT2A gene rearrangements, I do not believe that any exposure Ms. Amsler may have had to the water supply at Camp LeJeune over 50 years earlier is responsible for the development of her leukemia.
2. Ms. Amsler's prognosis at this point after her bone marrow transplant is very good with low risk of relapse and low risk of mortality related to the bone marrow transplant.
3. I believe Ms. Amsler would be able to return to her prior occupation or other employment at this time.

I base these opinions on my review of the relevant case materials and literature, decades of training and experience in hematology and oncology, and over 30 years of clinical diagnosis and treatment of patients with acute leukemia. I reserve the right to supplement these opinions if additional information is given to me after the date of this report. I hold all of these opinions to a reasonable degree of medical certainty.

II. Qualifications

I am a board-certified hematologist and oncologist at Duke University School of Medicine and a Member of the Duke Cancer Institute in Durham, North Carolina. I graduated in 1979 from Yale University with a Bachelor of Science degree in Biology. I earned my Medical Degree and Doctor of Philosophy Degree in Biophysics from Stanford University School of Medicine in California in 1988. I completed my internal medicine internship, internal medicine residency, and hematology and oncology fellowship at the Brigham and Women's Hospital, Dana Farber Cancer Institute, and Harvard Medical School. I am a Professor of Medicine in the Division of Hematologic Malignancies and Cellular Therapy in the Department of Medicine at Duke University. I serve as the Director of the Leukemia Program at Duke University. I have served as the Chair of the Southwest Oncology Group Leukemia Committee (SWOG) since 2012.[1] I have been a member of

[1] SWOG is one of the cooperative groups funded by the NCI to perform clinical research for people with cancer in the United States and its territories.

the National Cancer Institute (NCI) Leukemia Steering Committee since 2012. I am also the Co-Chair of the Senior Scientific Council of the NCI-sponsored MyeloMATCH precision medicine initiative in acute myeloid leukemia (AML) and myelodysplastic syndromes, a role that I have occupied since 2019.

Since 1996, my clinical and research career has focused on the diagnosis and treatment of adults with acute leukemias, myelodysplastic syndromes, and myeloproliferative neoplasms, first at the University of Michigan (1996-2012), then at the University of Alabama at Birmingham (2012-2018), and currently at Duke University (2018-present). I currently care for over 100 patients annually with AML, acute lymphoblastic leukemia (ALL), myelodysplastic syndromes, chronic myeloid leukemia (CML), and other myeloproliferative diseases in the Duke Blood Cancer Center outpatient clinic and on the Hematologic Malignancies Inpatient Service at Duke University Hospital. Before relocating to Duke University on July 1, 2018, I also cared for patients with chronic lymphocytic leukemia (CLL) for over 20 years at the University of Michigan and the University of Alabama at Birmingham. I am certified in Hematology by the American Board of Internal Medicine.

My current *curriculum vitae* is attached as Appendix A. It includes a list of my publications for the last ten (10) years. A list of the materials I considered in drafting this report is attached as Appendix B.

I receive $500 for each hour of service, including for my time spent testifying in a deposition or at trial. I have never been retained as an expert witness before my work in this and related cases in the Camp Lejeune litigation. I have never been deposed or testified at trial.

III. An Overview of Leukemia

Leukemia is a cancer arising from cells in the blood and bone marrow. To understand the origin of leukemia, one must be familiar with normal blood cell formation. There are two major subtypes of blood cells, myeloid and lymphoid. The myeloid cells include granulocytes (neutrophils, monocytes, eosinophils and basophils), platelets, and red blood cells. The lymphoid cells include B lymphocytes, T lymphocytes, and natural killer cells. The mature myeloid cells all have a finite life span in the blood: neutrophils remain in the blood for hours, platelets for 7-10 days, and red blood cells for 100 days. These cells are essential to human life. The granulocytes provide an innate defense against bacterial, fungal, and parasitic pathogens (in other words, infectious organisms). Platelets are essential for the first stages of blood clotting to prevent exsanguination due to disruption of the blood vessels. Finally, red blood cells carry oxygen from the lungs to the tissues of the body which is essential for energy production in these cells. B and T lymphocytes provide defense against viral pathogens, producing antibodies or cellular responses, respectively.

The bone marrow is responsible for constantly producing these mature blood cells throughout life. There is a finite pool of bone marrow stem cells and progenitor cells that are capable of self-renewal and differentiation into these myeloid or lymphoid cells. Leukemia is due to acquired changes in the genes (mutations) that govern the normal differentiation of the stem and progenitor cells into the mature blood cells. These cells may also acquire mutations in genes that lead to the

accumulation of leukemic cells. Depending on the type of progenitor cell (myeloid or lymphoid), these cancers are classified as myeloid (myelogenous) or lymphocytic (lymphoblastic) leukemia. In both cases, the bone marrow will ultimately not be able to continue its normal function of blood formation.

Myeloid and lymphocytic leukemia are also classified as either chronic or acute based on the rate of disease progression, the type of cancer cells that accumulate, and treatment methodology. Chronic leukemias progress slowly. In chronic leukemias, patients have a greater number of mature blood cells. In general, these more mature cells can carry out some of their normal functions. The two major types of chronic leukemia are chronic myeloid leukemia (CML) and chronic lymphocytic leukemia (CLL).[2]

Acute leukemias are cancers that rapidly expand in the bone marrow and require immediate chemotherapy (within days of initial presentation of the patient). In most adult patients with acute leukemia, hematopoietic stem cell transplant (also known as bone marrow transplant or blood and marrow transplant) after achieving an initial complete remission is the only potentially curative option. The two major types of acute leukemia are acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL). Ms. Amsler was diagnosed with ALL with a genetic abnormality of her KMT2A gene.

A. Acute lymphoblastic leukemia (ALL)

Acute lymphoblastic leukemia (ALL) is a cancer of the progenitor cells that create mature B or T lymphocytes. The median age at diagnosis is 17 years in the Surveillance, Epidemiology, and End Results (SEER) registry; 53% of ALL patients are less than 20 years old at diagnosis. However, there is a second peak in incidence later in life; 8.4% of ALL patients are between the ages of 55 and 64 years at diagnosis (https://seer.cancer.gov/statfacts/html/alyl.html).

The etiology of ALL in most patients is unknown ("idiopathic"). However, there are some known factors associated with increased risk of ALL development. The identical twin of a patient with ALL is at higher risk of developing ALL. Prior exposure to ionizing radiation, chemotherapy, and radiotherapy have been implicated in the development of ALL. Hereditary disorders such as Down syndrome, Li-Fraumeni syndrome, Fanconi anemia, and Bloom syndrome are associated with increased risk of developing ALL. There is also an increased risk of ALL in patients with hereditary immunodeficiency syndromes. However, over 90% of cases are idiopathic without an obvious etiology.

In ALL, leukemic cells called lymphoblasts accumulate rapidly in the bone marrow, leading to bone marrow failure. Given the finite survival of all peripheral blood cells, the number of normal myeloid cells in the blood will decrease if the marrow fails to continue to produce new blood cells. The rate of decrease will be proportional to the normal life of each cell; the number of neutrophils and platelets will decrease more rapidly than the red blood cells. The number of granulocytes declines rapidly, placing patients at risk of bacterial and fungal infections. Neutropenia (low

[2] Chronic lymphocytic leukemia and small lymphocytic lymphoma have been re-classified by The World Health Organization as CLL/SLL.

neutrophil count) is associated with opportunistic infections by bacteria normally found in the upper respiratory tract, the gastrointestinal tract, and the skin. Neutropenia may also make patients susceptible to lung, sinus, and deep tissue infections by airborne fungal spores. The patient's platelet count declines (a condition called thrombocytopenia), increasing the risk of spontaneous bruising, nose bleeds, and more serious hemorrhages. Anemia is the term used for a low red blood cell count (or low hemoglobin or low hematocrit). Anemia can cause fatigue, shortness of breath during activities, and headaches.

There can also be serious consequences of the increasing number of leukemic lymphoblasts in the blood. As the total white blood cell count in the blood increases over 100,000 per microliter, blood flow may become compromised in smaller blood vessels, negatively impacting the function of multiple organs. This clinical situation is termed leukostasis. Patients may develop encephalopathy. The clinical manifestations of encephalopathy include global decrease in cognitive function, confusion, sedation, expressive aphasia (an inability to speak), ataxia (inability to coordinate movement), loss of sight, and other neurologic findings. Lung function may become compromised resulting in impairment of gas exchange in the lungs and decrease oxygen delivery to tissues.

Therefore, clinical leukostasis is a medical emergency, requiring rapid reduction in the total white blood cell count with chemotherapy and/or leukapheresis. Leukapheresis is a procedure in which the blood of the patient is circulated through a machine that can remove the white blood cells (predominantly the leukemic blasts) and return the red blood cells, platelets, and plasma to the patient. Leukapheresis requires the emergent placement of a large bore catheter into a major vein of the body (subclavian, internal jugular, or femoral vein) under local anesthesia.

The rapid accumulation of leukemic lymphoblasts may have other serious consequences. The bone marrow and other tissues often involved by acute lymphoblastic leukemia such as lymph nodes, liver and spleen may not have adequate blood supply to allow the rapid accumulation of these malignant cells. The cancer cells will turn to anaerobic metabolism for energy production. However, this process will result in the accumulation of lactic acid in the blood. Normal cell function in other organs will be negatively impacted by the acidic environment. Also, the leukemic lymphoblasts themselves will undergo spontaneous lysis (breakdown) and necrosis (cell death). Tumor lysis results in the accumulation of uric acid and phosphate that will impair kidney function. Plasma potassium levels can increase rapidly due to both tumor lysis and impaired kidney function, resulting in fatal cardiac arrhythmias. Tumor lysis syndrome is a common complication of acute lymphoblastic leukemia presenting with leukocytosis (high white blood cell count) and may require urgent leukapheresis as well as artificial kidney function (forms of hemodialysis).

B. Treatment of ALL

There are multiple different chemotherapy regimens that are used to treat adults with ALL. All these regimens have several features in common. The goal of the first cycle (or course) of chemotherapy is to achieve a complete remission. Complete remission is defined as clearance of the leukemic cells from the blood and bone marrow and recovery of normal bone marrow function. The rate of complete remission in ALL is very high, approaching 90%. Unless a patient undergoes

allogeneic hematopoietic stem cell transplantation (allo HSCT, see below) in first remission, these chemotherapy regimens are continued for 2-3 years to decrease the risk of relapse. For the first 6-9 months, there are multiple different courses of intensive chemotherapy. Each course of therapy typically will include 4-5 different chemotherapy drugs. After completion of 9 months of intensive chemotherapy regimens, patients may continue maintenance therapy with oral, less toxic chemotherapy for up to 2-3 years from the time of diagnosis. We will also add immunotherapeutic agents to chemotherapy. If the leukemic blasts express a cell surface marker CD20, the addition of the monoclonal antibody rituximab to the chemotherapy has been shown to improve survival. Rituximab binds to the leukemic cells and directs the immune system to attack and kill the cancer cells. Recently, other immunotherapies (such as inotuzumab ozogamicin and blinatumomab) have been added as chemotherapy regimens. However, these newer therapies were not available at the time of Ms. Amsler's treatment in 2020.

Finally, therapy directed at the prevention of relapse in the central nervous system (CNS, including brain, spinal cord, cerebrospinal fluid, and membranes covering the brain and spinal cord) is essential. Most chemotherapy agents do not penetrate or accumulate in the CNS; the CNS acts as a sanctuary site for these leukemia lymphoblasts. Therefore, certain chemotherapy drugs (methotrexate, cytarabine, hydrocortisone, and dexamethasone) must be administered directly into the cerebrospinal fluid (CSF) surrounding the brain and spinal cord. Commonly, these chemotherapy agents are delivered into the CSF below the termination of the spinal cord. This procedure is known as a spinal tap or lumbar puncture. Chemotherapy delivered into the CSF is known as intrathecal chemotherapy. Lumbar puncture can be complicated by transient headaches and rarely by more serious transient neurologic events such as seizure or confusion. Some of the systemic chemotherapy regimens include higher doses of cytarabine and/or methotrexate that can penetrate the CNS as well.

Allo HSCT (bone marrow transplant) is considered for a subset of ALL patients who achieve a complete remission. The goal of allo HSCT is to decrease the risk of relapse further than that achievable with chemotherapy alone. There are two basic indications for allo HSCT in first complete remission: first, the genetic subtype of ALL, and second, the persistence of disease at very low levels in "complete remission" as measured by very sensitive tests (termed measurable residual disease, MRD). There are several ALL subtypes based on DNA analysis of the leukemic cells that are known to be resistant to chemotherapy and to have a high risk of relapse after first remission. Mutations involving the lysine methyltransferase 2A gene (KMT2A, previously known as Mixed Lineage Leukemia, MLL) is one of the more common high-risk genetic changes, occurring in approximately 5-15% of adult and pediatric ALL patients. Although the preferred treatment, many adult ALL patients cannot receive allo HSCT due their advanced age, poor function status, comorbid illnesses (heart or lung disease), and inadequate social support.

The first step in allo HSCT is to give the recipient (patient) a "preparative regimen," also known as a conditioning regimen. The preparative regimen consists of chemotherapy with or without total body radiation treatment. The purpose of the preparative regimen is to eradicate the bone marrow cancer (leukemia) and to suppress the recipient's immune system to allow the donor stem cells (bone marrow cells) to engraft (re-populate the bone marrow to produce healthy blood). The stem

cells are obtained from a donor who is closely matched to the recipient based on the major human leukocyte antigens (HLA). Using stem cells from a donor that is closely matched with the recipient reduces the risk of graft rejection (the infused new bone marrow cells not engrafting and not making blood) and graft-versus-host disease (GVHD). Most patients will have a donor identified from their immediate family or a registry of volunteer donors.

GVHD is an iatrogenic disease (i.e., a disease caused by the treatment). The donor immune system will engraft and identify the recipient cells as foreign. GVHD can occur even if the donor and recipient are "100%" HLA matched. This is due to minor HLA differences between the recipient and the donor. The stem cells can be taken from either related or unrelated donors and either from the peripheral blood or the bone marrow. The stem cells are infused into the recipient following the preparative regimen. Typically, the new bone marrow stem cells will engraft, and the blood counts will begin to improve within approximately 10-14 days. The recipient will be at risk for organ damage (lungs, liver, gastrointestinal tract) from the preparative regimen, graft failure (no engraftment), acute or chronic GVHD, and infections.

GVHD is a serious risk for leukemia patients who receive bone marrow transplants. There are two general types of GVHD, acute and chronic. Acute GVHD occurs within the first 3-12 months of the stem cell infusion. It is characterized by inflammation in the skin (rash), gastrointestinal tract (mouth sores, nausea, vomiting, loss of appetite, and diarrhea), and liver (liver damage leading to jaundice and liver failure). Complications of chronic GVHD may include recurrent bacterial infections (pneumonia, sinusitis), skin changes (scleroderma or thick, fibrotic skin), joint contractures, sicca syndrome (dry mouth and dry eyes), osteoporosis, infertility, early menopause, and increased risk of other cancers.

The efficacy of allo HSCT is not just related to the intensive chemotherapy and radiation therapy received prior to the stem cell infusion. In addition to targeting normal tissues in the recipient (patient), the immune system of the donor can recognize and eradicate residual leukemia cells in the recipient. This process is called graft-versus-leukemia effect. The relapse rate at 3 years following allo HSCT for ALL in first complete remission is approximately 30%; later relapses are very uncommon (see Greil C, et al. *Bone Marrow Transplant* 2021; 56: 841-52). In other words, the patient can be considered cured if there is no evidence of relapse within 3-5 years of the allo HSCT.

IV. Summary of Exposure

Ms. Amsler's father was stationed at Camp Lejeune from 1965-1967. From the fall of 1965 until May 1966, the Amsler family lived in off-base housing. Ms. Amsler attended a private kindergarten that was also located off base during the 1965-1966 school year. In May 1966, the family moved to a home located on base. Ms. Amsler attended first grade at a school on base for the 1966-1967 school year. In June 1967, the family left Camp Lejeune when Ms. Amsler's father was deployed to Vietnam (Karen Amsler Deposition, pages 94-97; see also Exposure Report of Judy S. LaKind).

V. Ms. Amsler's Relevant Medical History

Ms. Amsler (DOB [redacted]1960) was 60 years old at the time of her diagnosis of ALL with a KMT2A rearrangement (also known as 11q23 rearrangement or MLL rearrangement) on 09/12/2020. She has a relatively benign medical history, including prior treatment for high cholesterol, migraine headaches, rosacea, acne, and osteoporosis (see, e.g., 00284_AMSLER_0000010492-0000010891). She has benign liver cysts which have been either removed, drained, or injected. Other surgical interventions include bilateral tubal ligation (surgical sterilization), rhinoplasty, and right knee arthroscopy. There is no family history of leukemia. Family members have had prostate, breast, and colon cancer. She has never smoked. She worked in a microbiology laboratory at Johnson & Johnson until 2019. She was then employed at St. Joseph's Chandler Hospital in March 2020 after her move to Pennsylvania. As a microbiologist, she handled cultures of infectious agents and was exposed to culture media and antibiotics. Antibiotics and culture media are not associated with an increased risk of leukemia. Infectious agents are not known to cause any form of acute leukemia. Like most patients with acute leukemia, a definite causative condition was not identified at the time of her diagnosis.

Ms. Amsler's medical history shows that she was generally in good health until one day before her diagnosis of ALL in September 2020. Ms. Amsler lived in Pennsylvania before moving to South Carolina in 2019. She received primary care in Pennsylvania at Lehigh Valley Internal Medicine Clinic between 2015 and 2019. On 09/23/2015, complete blood counts and white blood cell differential were ordered as part of a routine health maintenance visit; all values were within the normal range (00284_AMSLER_0000010519).

When Ms. Amsler moved to South Carolina, she became the patient of primary care physician, Dr. John Moore. Her first visit with Dr. Moore occurred on 09/09/2019, one year before her diagnosis with ALL. The visit is described as a wellness visit without any specific symptoms noted on the entire review of systems (i.e., series of questions to gather information about a patient's overall health) (00284_AMSLER_0000000110 – 0000000120). Dr. Moore reviewed blood counts from Pennsylvania (from 07/26/2018); the complete blood counts and white blood differential were all within the normal ranges (00284_AMSLER_000000066 – 000000067). Ms. Amsler's complete blood counts were completely normal in September 2019 during her first visit with Dr. Moore as well (00284_AMSLER_0000010898-0000010909). Dr. Moore reviewed her history of osteoporosis, migraine headaches, rosacea, and liver cysts. He ordered a referral to Gastroenterology for follow up of the liver cysts.

On 09/08/2020, four days before her visit to the Emergency Room for evaluation of blurry vision, she had a video visit with Dr. Lancaster, MUSC Gastrointestinal Surgery, for her history of benign hepatic (liver) cysts (00284_AMSLER_0000007554). She was "asymptomatic" at the time. His comment may only have been related to symptoms due to liver cysts such as pain and early satiety. However, there is also no mention of blurry vision or leg swelling. Dr. Lancaster reviewed the MRI performed on 10/05/2020 of Ms. Amsler's abdomen. Dr. Lancaster only notes the presence of "multiple benign cysts without enhancing or nodular components." Although I could not find the actual report of this MRI, Dr. Lancaster made no mention of other changes typical of ALL,

such as enlarged lymph nodes or enlarged spleen. Ms. Amsler's prior abdominal MRI radiology reports also did not mention enlarged lymph nodes or enlarged spleen.

Ms. Amsler's first symptom related to ALL was blurry vision while at work on Friday, 09/11/2020. She was concerned, but she decided not to seek medical attention until the next day, Saturday, 09/12/2020. The following details of her presentation are found in the initial Emergency Department evaluation (00284_AMSLER_0000000325 as well as 00284_AMSLER_0000009133 - 0000009144) and Dr. Jennifer Yannucci's initial consultation (00284_AMSLER_0000000318 - 000000324). On presentation to the Emergency Department, Ms. Amsler had two symptoms: blurry vision only in the right eye and left leg swelling. She did not report any other symptoms that can be observed in patients with acute leukemia with a more indolent course, such as fever, drenching night sweats, weight loss, and loss of appetite. She was evaluated by an ophthalmologist in the Emergency Department. There was evidence of leukemic retinopathy (disease of the retina due to leukemia such as bleeding) and subretinal fluid. Her white blood cell (WBC) count was 608,800 (60 times the upper limit of normal), hemoglobin 8.7 gram/dL, and platelet count 138,000 (00284_AMSLER_0000009119). Her neutrophil count was normal. Most of the nucleated cells in the blood were blasts (64%) or atypical lymphocytes (35%, potentially also blasts). Her LDH (a marker of cell proliferation and turnover) was 26 times the upper limit of normal (5,235). Ultrasound Doppler examination of the legs demonstrated a distal (lower) left popliteal (posterior knee) deep venous thrombosis (00284_AMSLER_0000000339).

Ms. Amsler received oral hydroxyurea chemotherapy capsules and underwent leukapheresis with rapid reduction in the total white blood cell count over a few days. With these interventions she did not suffer clinical consequences of a tumor lysis syndrome. The leukemia diagnosis was made by both peripheral blood and bone marrow examination. The peripheral blood flow cytometry detected B lymphoblasts (cancer cells) with absence of CD10 expression, strongly suggestive of the KMT2A gene rearrangement. The bone marrow was packed with acute leukemia. The bone marrow was hypercellular for age (95% cellularity). Typically, the cellularity of the bone marrow from the posterior iliac crest (the site of the bone marrow biopsy) is 100-patient age. In other words, normally Ms. Amsler's bone marrow should have only been 100-60, or 40% cellular with the remaining 60% of the space filled with fat cells. Almost all these cells were the B lymphoblasts (90% involvement). The diagnosis was confirmed by staining the bone marrow biopsy and detecting expression of CD34, TdT, and PAX5, markers of B lymphoblasts, on the cancer cells in the bone marrow (00284_AMSLER_0000009405). Cytogenetic analysis (chromosome analysis) demonstrated an abnormal female karyotype in six cells with a translocation between chromosome 4, band q21 and chromosome 11, band q23 (00284_AMSLER_0000009407). The chromosomal change was due to fusion of the KMT2A gene on chromosome 11q23 to the AFF1 gene on chromosome 4q21. This KMT2A rearrangement was confirmed by Fluorescence In Situ Hybridization, or FISH (00284_AMSLER_0000009403).

Ms. Amsler then started a standard chemotherapy regimen for ALL known as "hyper CVAD.[3]" The records note that she also received a monoclonal antibody, rituximab. The addition of this antibody to chemotherapy has been shown to improve the survival of ALL patients. However, her ALL cells did not express (have) the target for rituximab, CD20 (noted in a physician note on 00284_AMSLER_0000008714). Therefore, she did not receive rituximab as documented, for example, on chemotherapy order and chemotherapy verification forms for cycle #2A Hyper CVAD (0284_AMSLER_0000002928 and 0000002929). The confusion in the medical record may have been created by the chemotherapy flow sheets (00284_AMSLER_000000245 – 000000272; 00284_AMSLER_000000348 – 000000375) which note "Hyper CVAD + R" as the chemotherapy regimen (R stands for the rituximab antibody). The chemotherapy flow sheets were likely pre-populated. For example, the doses of chemotherapy for cycles 6 (3A), 7 (4A), and 8 (4B) were entered on the flow sheets, but never administered.

The first four days of each hyper CVAD chemotherapy cycle are administered in the hospital as an inpatient because the schedule of drug administration does not allow for outpatient administration in most clinics. A peripherally inserted central venous catheter (PICC) is inserted in the arm for each admission for convenience and safety of administration. Two of the medications (vincristine and doxorubicin) may cause severe tissue damage if they leak out of a peripheral intravenous catheter. The odd number cycles and the even number of cycles are different from each other. During the odd number cycles (also known as cycles #1A, 2A, 3A, and 4A), patients receive intravenous cyclophosphamide, doxorubicin, vincristine, and dexamethasone. During the even number cycles, patients receive high dose methotrexate and high dose cytarabine (cycles #1B, 2B, 3B, and 4B). The dose of the cytarabine was appropriately attenuated (decreased by 67%) for Ms. Amsler based on her older age. She had two lumbar punctures with each cycle of the hyper CVAD regimen. Chemotherapy was administered directly into the cerebrospinal fluid during each procedure (called intrathecal administration). She received intrathecal methotrexate on 09/17, 10/08, 10/29, 11/19, and 12/10/2020. She received intrathecal cytarabine on 09/22, 10/08, 11/03, 11/24, 12/15/2020. The first lumbar puncture was performed on 09/18/2020, during her first admission; there were no leukemia cells seen in the cerebrospinal fluid. Up to 5% of ALL patients may have leukemic involvement of the central nervous system and/or the CSF at diagnosis; this is especially true of patients like Ms. Amsler with high white blood cell count at diagnosis and with KMT2A-r ALL. Involvement of the central nervous system by any acute leukemia at the time of diagnosis is considered a poor risk feature.

She received cycle #1A hyper CVAD during the first admission starting on 09/16/2020. She started cycle #1B on 10/07/2020; cycle #2A on 10/28/2020; cycle #2B on 11/18/2020; and cycle #3A on 12/09/2020. She did not receive further cycles of hyper CVAD, because the plan was to proceed with the allo HSCT (bone marrow transplant) as soon as possible. She experienced fatigue and nausea during chemotherapy. She required blood transfusions, since the chemotherapy caused

[3] Hyper CVAD is a commonly-prescribed high-dose, intensive chemotherapy regimen for ALL and aggressive B cell lymphomas that delivers multiple cycles of four chemotherapy drugs [typically cyclophosphamide (Cytoxan), vincristine sulfate, doxorubicin hydrochloride (Adriamycin), and the steroid dexamethasone] over a four day period (the A cycles) rapidly alternating every three weeks with cycles of high dose methotrexate and high dose cytarabine over three days (the B cycles).

transient suppression of the bone marrow function with each cycle. The chemotherapy cycles are given every 3 weeks, to allow time for the bone marrow to recover from the prior cycle. Her only unexpected complication occurred during cycle #2B hyper CVAD. She developed rapid palpitations in her chest due to atrial fibrillation, an abnormal heart rhythm associated with decreased heart function and risk of strokes, during cycle #2B. She required electrical cardioversion (electric shock to her chest) to terminate the atrial fibrillation. She was then treated with an anti-arrhythmic medication, flecainide and metoprolol. More recently, she only takes flecainide if she feels the palpitations return (pill-in-the-pocket method). She had minor transient, self-limited bleeding from her ear after cycle #3A on 12/18/2020. She never required re-admission for treatment of a complication of the chemotherapy regimen. Re-admission for treatment of fever and infection due to a low white blood cell count is very common, especially after the B cycles of treatment. The details of these admissions for hyper CVAD treatments can be found in the Memorial Hospital records (00284_AMSLER_0000009115 – 0000009545).

Other than the above-noted heart palpitation complication, Ms. Amsler tolerated hyper CVAD chemotherapy remarkably well. The chemotherapy drugs, cyclophosphamide and doxorubicin, have both been associated with a low risk (1%) of developing a different bone marrow cancer years later, either myelodysplastic syndrome or acute myeloid leukemia.

To document complete remission, Ms. Amsler underwent repeated bone marrow biopsies before and after the allo HSCT. She first saw Dr. Praneeth Baratam on October 27, 2020, after remission had been documented for consideration of allo HSCT during first remission. Acute leukemia must be in remission prior to allo HSCT; otherwise, the risk of relapse is very high (nearly 100%). He agreed with Dr. Yannucci that allo HSCT would provide her with the greatest chance of the disease not recurring and the greatest chance of cure. During the next two months, she continued with hyper CVAD therapy to maintain the remission, while Dr. Baratam's team prepared for the allo HSCT. A stem cell donor had to be identified and evaluated to be an acceptable stem cell donor. Ms. Amsler underwent several studies before the allo HSCT to ensure she was eligible for the procedure (such as repeat bone marrow biopsy, echocardiogram, and pulmonary function tests) because prior authorization for the allo HSCT had to be secured from her third-party insurance plan.

She enrolled on the Blood and Marrow Transplant Clinical Trial Network (BMT-CTN) 1703 clinical trial; she was randomly assigned to the standard-of-care GVHD regimen, tacrolimus and methotrexate. She received a reduced intensity conditioning (RIC) preparative regimen with fludarabine and melphalan chemotherapy. On 01/14/2021, she received an infusion of hematopoietic stem cells collected from the peripheral blood of an unrelated donor through a Hickman catheter in her chest. The donor was HLA-identical with the patient at all 10 HLA loci (i.e., a perfect match). She remained in the hospital for one month after the transplant and stayed in Charleston for frequent evaluation at MUSC for 100 days following her discharge (standard post allo HSCT procedure). On April 26, 2021, a repeat bone marrow biopsy was negative for any evidence of the ALL; there was 100% donor chimerism (i.e., the stem cell transplant had engrafted and was producing blood and immune cells).

In early May 2021, she developed a rash on her arms that spread to her legs and chest. The rash did not improve with the topical high-potency steroid, clobetasol (00284_AMSLER_0000000387). She was felt to have grade 2 (25-50% of body surface area) skin only acute graft-versus-host disease (GVHD). The dose of the tacrolimus was increased. By May 17, 2021, the rash had improved. By 06/01/2021, the rash had resolved (00284_AMSLER_0000003584). The tacrolimus could be tapered and ultimately discontinued. She did not have any significant infectious complications following the allo HSCT.

She now appears to have a normal quality of life. She is continuing her health maintenance evaluations for osteoporosis, liver cysts, hyperlipidemia, paroxysmal atrial fibrillation, and arthritis. She has been able to travel internationally.

VI. Analysis

A. Based on the biology of ALL associated with KMT2A gene rearrangements, Ms. Amsler's ALL is not related to any exposure to the water supply at Camp Lejeune over 50 years earlier.

KMT2A rearrangements can be seen in both acute myeloid leukemia and acute lymphoblastic leukemia. In fact, the prior name for the KMT2A gene was MLL, an abbreviation for myeloid lymphoid leukemia. KMT2A rearrangements can be found in therapy-related AML and ALL as well as de novo AML and ALL. In the MD Anderson series, 40% of KMT2A-r AML were therapy-related (Issa G, et al. Blood Cancer J. 2021; 11: 162). In a series of patients from five academic centers, 14 of 22 adult patients (64%) with KMT2A-r ALL were therapy-related (Saygin C, et al. Blood Adv. 2019; 3: 4228).

To understand the cause of Ms. Amsler's ALL, we must first review the biology of this subtype of ALL with KMT2A mutations. The mechanism by which KMT2A genetic changes lead to acute leukemia has been elucidated. In a subset of patients with acute leukemia, the KMT2A gene is broken, and the first part of the gene is then fused to one of over 100 other genes. This genetic event is known as a rearrangement, leading to the abbreviation KMT2A-r to describe this class of mutations. In turn, the rearranged gene produces a fusion protein. Regardless, of the partner gene, a fusion protein produced from the KMT2A-r is responsible for the development of acute leukemia. The KMT2A fusion protein leads to the expression of genes that block the normal differentiation of immature cells in the bone marrow (such as HOX and MEIS1) and cause progenitor cells to proliferate and accumulate, leading to leukemic transformation. KMT2A-r in ALL usually occurs as a single mutation and often does not require a cooperative mutation to trigger the leukemia pattern (see Andersson AK, et al. Nat Genet. 2015; 47; 330-337; Agraz-Doblas A, et al. Haematologica 2019; 104: 1176-1188).

There is evidence from pre-clinical studies (laboratory experiments) that the KMT2A rearrangement is sufficient to cause acute leukemia within a very short latency period. When a KMT2A fusion gene is introduced into mouse bone marrow cells, the mice develop leukemia

within 4-12 months. Introduction of several different KMT2A fusion genes into hematopoietic precursor cells induce transformation to leukemia immediately upon transplantation of these precursor cells into recipient mice. The very short latency between development of a KMT2A rearrangement and development of acute leukemia is also highlighted by two specific clinical examples: (1) ALL in neonates (infants less than 1 year of age) is associated with KMT2A rearrangement in 80% of cases; and (2) clinical observations of patients with therapy-induced acute myeloid leukemia (AML). In this second example, acute leukemia with KMT2A rearrangement is known to occur within 1-2 years of exposure to leukemogenic chemotherapy, such as doxorubicin, daunorubicin, and etoposide. These chemotherapy drugs are inhibitors of topoisomerase II, which leads to breakage of the KMT2A gene and fusion with other genes. These two clinical scenarios clearly indicate the very rapid development of acute leukemia following the appearance of the KMT2A rearrangement, the latter related to exposure to a known leukemogenic toxin.

Clinical observations also strongly suggest that the KMT2A mutation occurs as a single mutation, and that this mutation is all that is required to trigger the leukemogenic event. For example, in AML associated with KMT2A rearrangements, more than half of the patients in one cohort study did not have any other pathogenic driver mutation (Issa G, et al. Blood Cancer J. 2021; 11: 162). Issa and colleagues also show that KMT2A-r AML almost never have mutations that have been associated with prior DNA damage (e.g., DNMT3A, TET2, and ASXL1 mutations). In other words, additional genetic events preceding or following the occurrence of the KMT2A rearrangement are not required for the development of acute leukemia due to KMT2A rearrangement. The KMT2A rearrangement creates a fusion protein that rapidly induces abnormal proliferation of the marrow progenitor cell, blocks differentiation of these cells, and results in the appearance of acute leukemia (either myeloid or lymphoblastic) within a few months, not decades later.

In summary, when ALL is induced by the KMT2A rearrangement (mutation), other recurring, cooperative mutations are not required. The patient will present clinically with symptoms and abnormal blood counts related to ALL shortly after the KMT2A fusion gene has occurred. Ms. Amsler's exposure to the contaminated water supply at Camp Lejeune occurred more than 50 years before her diagnosis of KMT2A rearranged ALL, in effect excluding the VOCs in the Camp Lejeune water supply as a causative agent.

In Ms. Amsler's case, she was working in her lab in September 2020 when she noticed blurry vision in one eye. This was the first indication that something was wrong with no earlier prodrome symptoms (meaning no early signs or symptoms of the illness that preceded the characteristic manifestations). Her WBC was 609,000, which is incredibly high (a WBC count of 10,000 is normal). Her initial bone marrow biopsy showed 90% leukemic blasts. However, her platelet count was 138,000, which is relatively normal (approx. 150,000 per microliter blood is normal). Her neutrophil count was also still normal. Both WBC and platelets are produced by bone marrow. Platelets have a life span of about 7 days. WBC spend less than one day in the blood before exiting the blood to enter tissues to prevent infectious organisms from entering the body. The relatively preserved blood platelet and neutrophil counts despite a bone marrow completely packed with non-functional leukemia cells indicate that the disease came on suddenly, within a few days to a

week. Her illness exploded out of nowhere. She had not been sick with it for months or years, and it certainly was not caused by a genetic mutation related to exposure to water at Camp Lejeune over 50 years earlier. To state it plainly, this leukemia will not sit and do nothing for decades.[4]

There was a 53-year gap between when Ms. Amsler's family left Camp Lejeune and the onset of her leukemia. It is highly improbable, bordering on impossible, that any possible chemical exposure more than five decades earlier could be a direct cause of Ms. Amsler's specific subtype of ALL with KMT2A rearrangement.

My analysis in this case does not require looking at exposure and risk assessment data due to my opinion that Ms. Amsler's ALL was induced by the spontaneous occurrence of a KMT2A rearrangement mutation in her hematopoietic stem cells shortly before her clinical presentation and was not caused by a genetic mutation related to exposure to Camp Lejeune water over 50 years earlier. Nonetheless, the reports of Drs. Judy LaKind and Lisa Bailey demonstrate that Ms. Amsler had minimal exposure to the chemicals at issue, particularly benzene (see reports of Judy S. LaKind and Lisa A. Bailey for Karen Amsler).

B. Ms. Amsler's prognosis more than 4 years after her bone marrow transplant is very good with low risk of relapse and low risk of mortality related to the bone marrow transplant.

Ms. Amsler's two symptoms at presentation to the emergency department on 09/12/2020, blurry vision and deep venous thrombosis (DVT), were both directly related to ALL. The blurry vision in just one eye was due to leukostasis of blood flow in the retina. Her left leg swelling was due to the DVT of the left popliteal vein, likely triggered by ALL. Treatment of a single provoked DVT is typically only 3-6 months of an anticoagulant ("blood thinner"). She was on rivaroxaban (Xarelto) for the DVT. However, she now continues an anticoagulant (apixaban, Eliquis) to prevent thromboembolic events (e.g., strokes) associated with paroxysmal atrial fibrillation.

She already had osteoporosis prior to the diagnosis of ALL, and she has remained on appropriate therapy to slow the progression of osteoporosis and prevent fractures. The several courses of steroids during hyper CVAD chemotherapy and for treatment of the acute GVHD may contribute to osteoporosis.

I agree with Dr. Baratam as he stated in June 2024 (3.5 years post-transplant) that Ms. Amsler's chances of long-term survival were excellent at that point because she had made it past the 2-year mark (see Baratam Deposition, page 39). Although we cannot be certain, we generally consider

[4] Dr. Baratam (one of Ms. Amsler's treating doctors for the allo HSCT) asserted that "acute leukemias only tend to happen within days to weeks" (see Baratam Deposition, page 26). Dr. Yannucci (another of Ms. Amsler's treating doctors) indicated that the disease becomes clinically evident within a few weeks at most (see Dr. Yannucci Deposition, page 57). I agree with these statements. However, there is a difference between the clinical onset of a cancer and the first genetic abnormalities that ultimately lead to the cancer. In Ms. Amsler's case, the latency between the causative genetic change in the KMT2A gene and the clinical presentation with acute leukemia would only be 1-2 years, not over 50 years.

patients cured of any acute leukemia if there has been no evidence of relapse five years after initial remission. She is now four years post allo HSCT with no recurrence of the disease. She is likely cured since most recurrences are within the first year or so after stem cell transplant.

C. I believe Ms. Amsler would be able to return to her prior occupation or other employment at this time.

I also agree with Dr. Baratam that she may return to work in her chosen profession as a microbiologist (see Baratam Deposition, pages 41-43). Dr. Yannucci also stated that she did not ask Ms. Amsler to modify her lifestyle due to the bone marrow transplant (see Yannucci Deposition, page 37). Ms. Amsler is, by her own account and that of her husband (see Deposition of Michael Wukitch, e.g., pages 54, 56-57), engaging in activities such as exercise, travel, and occasional employment outside the home.

VII. Response and Rebuttal to Dr. Gondek's Expert Report

I disagree with some of the assertions made by Dr. Gondek in his report (dated 02/07/2025).

A. Causation

First, Dr. Gondek states that the specific genetic rearrangement of Ms. Amsler's B-ALL (i.e., (4;11)(q21;q23)) supports the notion that her disease was caused by exposure to cytotoxic agents, such as benzene. Dr. Gondek's assertion that 11q23 rearrangements are more likely to be associated with exposure to cytotoxic agents "such as benzene, TCE, and PCE" is incorrect. There is no data on the specific cytogenetic abnormalities observed in acute leukemia following exposure to benzene, TCE or PCE. Moreover, a recent analysis of one hundred seventy-two AML patients with KMT2A rearrangements treated at M. D. Anderson Cancer Center (Issa G, et al. Blood Cancer J. 2021; 11: 162) found therapy-related disease (i.e., AML related to exposure to certain cytotoxic chemotherapy drugs that are inhibitors of topoisomerase II) in only 40% of their cohort (i.e., not the majority). In a series of patients from five academic centers, 8 of 22 adult patients (36%) with KMT2A-r ALL were NOT therapy-related (Saygin C, et al. Blood Adv. 2019; 3: 4228).

While I agree that there is a strong association between 11q23 translocations in therapy-related AML and prior exposure to topoisomerase II inhibitors such as anthracyclines and etoposide, Dr. Gondek fails to mention the very short latency period between such exposures and the onset of KMT2A rearranged acute leukemia in all these studies. Both AML and ALL with KMT2A rearrangements occur within 1-3 years of the exposure to the topoisomerase II inhibitor exposure. The very short latency has also been my personal experience as a treating oncologist for the last 30 years.

Second, Dr. Gondek cites to two studies to support his statement that 11q23 rearrangements were seen in white blood cells obtained by people exposed to benzene: (1) Zhang (Zhang L, et al. Environ Mol Mutagen. 2007; 48: 467); and (2) Vaughan (Vaughan A, et al. Chemico-Biological

Interactions 2005; 153-154:179). See Gondek report, page 7. In the Zhang study involving Chinese workers exposed to benzene, the manuscript clearly states that there was NO increase in two of the most common 11q23 translocations, t(4;11) and t(6;11), in these people, even at high levels of benzene exposure **(see table below)**. These were the only two KMT2A translocations tested in this study. The specific KMT2A rearrangement in Ms. Amsler's ALL was the t(4:11).

TABLE III. Structural chromosome aberrations (SCAs) in workers exposed to different levels of benzene

Benzene exposure (n)[a]	t(11; 4, 6, ?)[b]	t(14;18)	del(6q)	Other SCAs
Controls (44)	0.043 ± 0.018[c]	0 ± 0.0	2.91 ± 0.31	0.06 ± 0.02
Exposed (43)	0.034 ± 0.020	0.025 ± 0.013***[d,e]	4.61 ± 0.44**[d]	0.19 ± 0.03***[d]
≤31 ppm (21)	0.005 ± 0.005	0 ± 0.0	3.67 ± 0.61	0.16 ± 0.04**[d]
>31 ppm (22)	0.061 ± 0.038	0.049 ± 0.025***[d,e]	5.51 ± 0.58***[d]	0.22 ± 0.05***[d]
P_{trend}	0.28	NA[f]	0.0002	0.0003

In the Vaughan study, Vaughan and colleagues suggested that inhibition of topoisomerase II by benzene metabolites may result in acute leukemia via dysregulation of apoptosis (i.e., cell death) associated with novel KMT2A rearrangements. However, Vaughan and colleagues were able to induce KMT2A translocations by irradiation or exposure to an anti CD95 monoclonal antibodies in cell cultures. Their studies did not actually use benzene or its derivatives to create these KMT2A fusion genes. Furthermore, the fusion genes identified in their studies were NOT any of the previously described fusions observed in human leukemia, including the KMT2A::AF4 fusion gene of the t(4;11) translocation found in the leukemic blasts of Ms. Amsler.

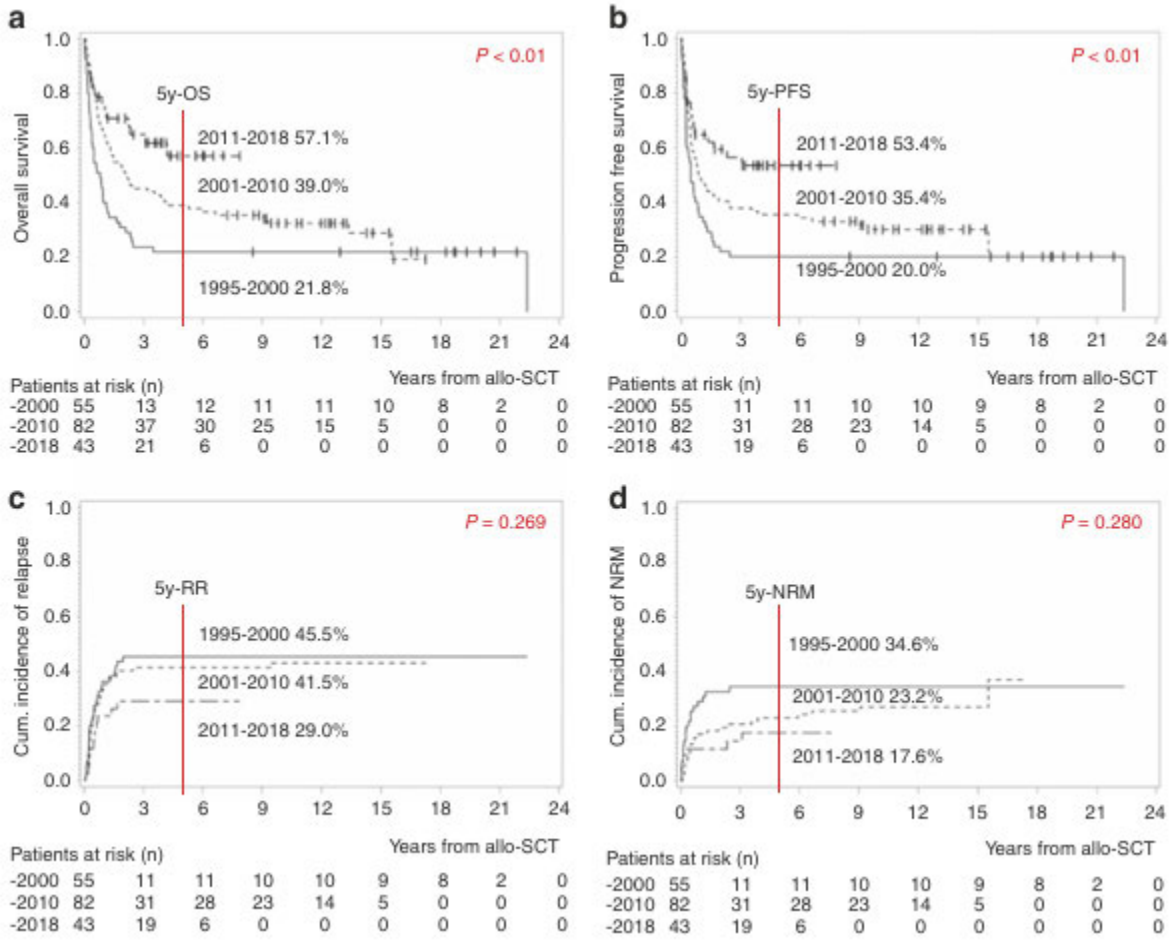
Further, Dr. Gondek cites the New Jersey study of exposure to VOCs in drinking water and the incidence of leukemia and non-Hodgkin lymphoma (Cohn P, et al. Environ Health Perspect. 1994; 102: 556-561) to support his opinion that there is a relation between TCE exposure and the development of ALL. Due to small numbers of events (ALL cases), the confidence intervals are wide (i.e., 95% CI: 1.03-5.45). Moreover, the data from the New Jersey Cancer Registry extended only a few years beyond the time of the water contamination, not decades as in the case of Ms. Amsler. Given the limited timing of the collection of leukemia cases from the New Jersey State Cancer Registry, this analysis does not allow us to conclude that an exposure to VOCs may be a cause of ALL decades later.

Finally, although Dr. Gondek relies heavily on the International Agency for Research on Cancer (IARC) (see Gondek report, page 10), he fails to mention that IARC does not classify benzene, TCE, PCE, or vinyl chloride as having sufficient or limited evidence of association with ALL. See Report of Peter Shields at page 60; see also Report of Julie E. Goodman on Leukemia (epidemiological evidence does not support an association between benzene, TCE, PCE, and vinyl chloride and ALL).

B. Damages

Since Ms. Amsler has not had significant acute or chronic graft-versus-host disease, she has not required prolonged courses of steroids. Therefore, I believe her risk of progressive osteoporosis will return to that expected for an average post-menopausal woman. She also has not had further episodes of atrial fibrillation and has not required ongoing daily anti-arrhythmic therapy or anti-coagulation.

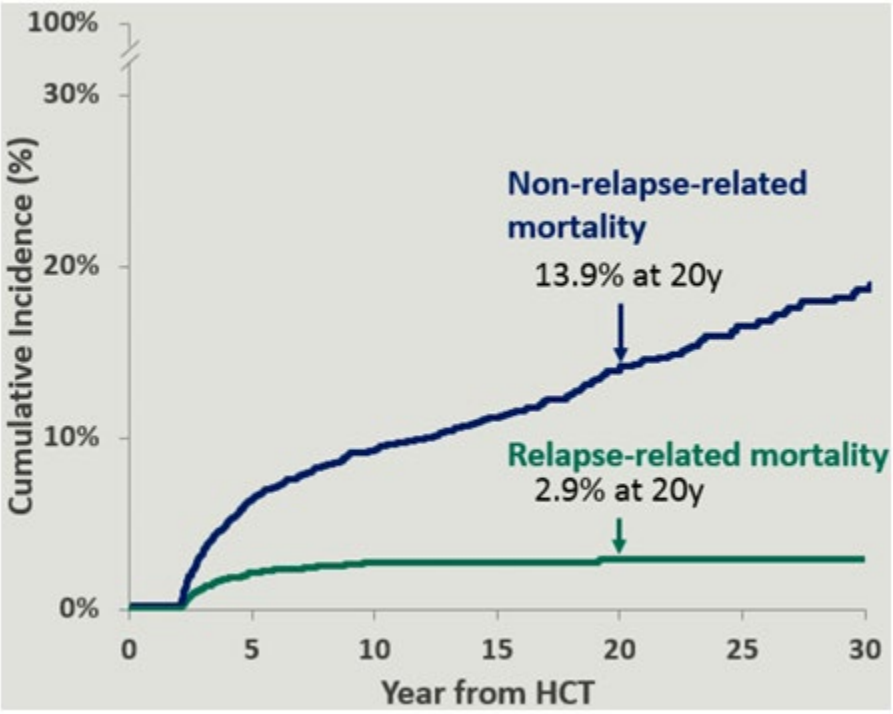
On the possibility of a relapse of Ms. Amsler's ALL, Dr. Gondek cites the manuscript by John Wingard and colleagues regarding the outcome of ALL patients undergoing allo HSCT (see Wingard JR, et al. J Clin Oncol. 2011; 29: 2230). This analysis of the Center for International Center for Blood and Marrow Transplant Research (CIBMTR) included 10,620 ALL patients transplanted between 1980 and 2003. This is old data. The outcomes following allo HSCT have improved since this time. For example, the data from the University Hospital of Freiberg clearly shows the improvement in survival over time for ALL patients undergoing allo HSCT (Greil C, et al. Bone Marrow Transplant 2021; 56:841). Five-year survival improved from 21.8% in the 1995-2000 period, to 39.0% in the 2001-2010 period, to 57.1% percent in the most recent period 2011-2018. There were improvements in progression free survival as well as risk of relapse between 1995 and 2018. Furthermore, the data below in **panel C** indicates that the risk of non-relapse related mortality (e.g., death due to graft-versus-host disease and infection) as well as the risk of relapse plateaus by 4 years following the allo HSCT. This more recent data illustrates the advances in allo HSCT which have resulted in improvement in non-relapse related mortality.



Dr. Smita Bhatia, an NIH funded expert in patient outcomes following allo HSCT, reviewed the life expectancy of patients following allo HSCT (see Bhatia S. Cause Specific Late Mortality after Allogeneic Stem Cell Transplant. American Society of Hematology Educational Book, 2019: 626-629). The following is the abstract from her manuscript:

Conditional on surviving the first 2 to 5 years after allogeneic blood or marrow transplantation (BMT), the 10-year overall survival approaches 80%. Nonetheless, the risk of late mortality remains higher than the age- and sex-matched general population for several years after BMT. The higher mortality rates in transplant recipients translate into shorter projected life expectancies compared with the general population. Risk of relapse-related mortality reaches a plateau within 10 years after BMT. With increasing time from BMT, nonrelapse-related mortality becomes the leading cause of death, and continues to increase with time after BMT. The major causes of nonrelapse mortality include infection (with or without chronic graft-versus-host disease), subsequent neoplasms, and cardiopulmonary compromise. In this review, findings from large cohorts are summarized, identifying opportunities for risk-based anticipatory intervention strategies to reduce mortality.

The cumulative risk of relapse-related mortality at 10-20 years following allo HSCT in patients surviving greater than 2 years post allo HSCT is less than 5%, and this risk reaches a plateau by 4-5 years after the allo HSCT. On the other hand, the risk of non-relapse mortality increased over time in registry analyses performed decades ago; an analysis of the Bone Marrow Transplant Survivor Study reported a 13.9% risk of non-relapse mortality (NRM) at 20 years post allo HSCT (Francisco L, et al. Blood 2016; 128: 691). The most common causes of NRM are infection, cardiovascular disease, pulmonary disease, and second primary cancers. However, the risks of infections, pulmonary disease, and second malignancies are related to the use of total body irradiation during the preparative regimen for the allo HSCT and chronic graft-versus-host disease. Ms. Amsler did not receive total body irradiation and has only minimal evidence of chronic graft-versus-host disease (dry eyes). The risk of developing post allo HSCT cardiovascular disease is related to independent cardiovascular risk factors the patient may have and the use of anthracycline chemotherapy during leukemia treatment. Ms. Amsler has controlled hyperlipidemia as her only cardiovascular risk factor. She is unlikely to develop anthracycline-induced cardiomyopathy at this point, since she received doxorubicin (Adriamycin) five years ago.



Ms. Amsler underwent allo HSCT four years ago. She has remained in remission without evidence of infectious complications and only minimal evidence of chronic graft-versus-host disease (dry eyes), not requiring any immunosuppressive therapy. The infectious risks cited by Dr. Gondek including cytomegalovirus (CMV) and invasive fungal infections such as Aspergillus are most likely to occur in the first two years following allo HSCT. These risks continue until recovery of normal immune function. Although I have not seen an extensive evaluation of her immune function, there was full donor chimerism (engraftment of the donor immune system) in 2021. The immune function generally recovers within 3 years post allo HSCT in the absence of ongoing immunosuppressive therapy or significant chronic graft-versus-host disease. Ms. Amsler has neither of these risks. She did not receive a preparative regimen for the allo HSCT that increases the risk of cardiac injury; that is, she did not receive total body irradiation or cyclophosphamide. Therefore, her risk of cardiac failure following the hyper CVAD regimen (exposure to doxorubicin) is very low at this point.

VIII. Conclusions

Based on my review of this case, I have reached the following conclusions concerning Ms. Amsler's case to a reasonable degree of medical certainty:

1. Any exposure Ms. Amsler may have had to the water supply at Camp LeJeune was not responsible for the development of her ALL.
2. As of today, Ms. Amsler is likely cured of ALL; the risk of relapse is less than 5%, at most. Her risk of non-relapse mortality related to the bone marrow transplant is also low because she did not receive total body irradiation, has minimal evidence of chronic-graft-versus host disease, and is unlikely to develop anthracycline-induced cardiomyopathy since she received doxorubicin (Adriamycin) five years ago.
3. Ms. Amsler has not endured any toxicity that would preclude her from returning to a normal life and from pursuing her chosen career.

APPENDIX A

DUKE UNIVERSITY MEDICAL CENTER

CURRICULUM VITAE

Harry Paul Erba, MD PhD

Primary academic appointment: Hematologic Malignancies and Cellular Therapy

Primary academic department (not DUAP): Medicine

Secondary appointment (if any) - (department): N/A

Present academic rank and title (if any): Professor of Medicine

Date and rank of first Duke Faculty appointment: 07/01/2018

Medical Licensure: North Carolina License, # 2018-01322

Date of License: 05/25/2018

Specialty certification(s) and dates (Month/Day/Year):

ABIM, Board Certified, Internal Medicine (09/25/1991-12/31/2011)

ABIM, Board Certified, Hematology, (11/10/1994-Present)

ABIM, Board Certified, Medical Oncology, (11/09/1995-12/31/2015)

Citizen of United States

Education and Training

Education	Institution	Date (Year)	Degree
High School	North Haven	1975	HS Diploma
College	Yale University	9/1975-5/1979	BS, Biology
Graduate or Professional School	Stanford University School of Medicine	9/1981-6/1988	MD, Ph.D.

Scholarly societies (Alpha Omega Alpha, Sigma Xi, Phi Beta Kappa; etc.):

1977 Sigma Xi
1978 Phi Beta Kappa

Professional training and academic career (chronologically, beginning with first postgraduate position):

Institution	Position/Title	Dates
Internships and Residencies:		
Harvard Medical School	Clinical Fellow in Medicine	6/1988-6/1991
Brigham and Women's Hospital	First-Year Resident Physician	6/1988-6/1989
Brigham and Women's Hospital	Second-Year Resident Physician	7/1989-6/1990
Fellowships:		
Brigham and Women's Hospital	Clinical Fellow in Medicine	7/1990-6/1991
Brigham and Women's Hospital	Research/Clinical Fellow in Medicine	6/1991-7/1992
Harvard Medical School	Research Fellow in Medicine	6/1991-6/1993

Academic, Administrative, and Clinical Appointments

Instructor in Medicine, Harvard Medical School	7/1993-6/1996
Assistant Professor of Internal Medicine, University of Michigan Medical School	6/1996-8/2005
Associate Director, Hematology/Oncology Fellowship, University of Michigan	6/1996-12/05
Associate Professor of Internal Medicine, University of Michigan Medical	9/2005-6/2012
Professor of Internal Medicine, University of Alabama at Birmingham (UAB)	7/2012-4/2018
Director, Hematologic Malignancy Program, UAB Division of Hem & Oncology	7/2012-4/2018
Chair, Hematologic Malignancy Working Group, UAB Cancer Center	7/2012-4/2018
Associate Director, Clinical Research, UAB Comprehensive Cancer Center	3/2013-4/2018
Alfred F. LoBuglio Endowed Chair for Translational Cancer Research, UAB	9/2012-4/2018
Professor of Medicine, Duke University	7/2018-Present
Medical Director, Hematologic Malignancies Inpatient Services, Duke Hospital	1/2019-5/2023
Director, Leukemia Program, Duke Cancer Institute	7/2019-present

Hospital Appointments

7/1993-6/1996	Assistant Physician, Harvard University Health Services
7/1993-6/1996	Associate Staff Physician, Department of Medical Oncology, Dana-Farber Cancer Institute
7/1992-6/1996	Associate Physician, Brigham and Women's Hospital
7/1996-6/2012	Attending Physician, University of Michigan Health Systems
7/2012-4/2018	Attending Physician, University of Alabama at Birmingham Hospital
7/2018-Present	Attending Physician, Duke University Medical Center

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Chapters in books

1. Kelley's Essentials of Internal Medicine, Second Edition. Humes, H. David, ed. in- chief. **Erba, Harry P.**, Associate ed., Lippincott, Williams and Wilkins, 2001.

2. Conn's Current Therapy. Rakel, RE and Bope, ET, eds. **Erba, Harry P.**, W.B. Saunders Company, 2002.

3. Conn's Current Therapy. Rakel, RE and Bope, ET, eds. **Erba, Harry P.**, W.B. Saunders Company, 2003.

4. Hematology for the Medical Student. Schmaier, A and Petruzzelli, L, eds. Myeloid Stem Cell Disorders. **Erba Harry P.**, Lippincott Williams and Wilkins, March 2003:153-171.

5. Hematology for the Medical Student. Schmaier, A and Petruzzelli, L, eds. Acute Leukemia. **Erba, Harry P.**, Lippincott Williams and Wilkins, March 2003:173-184.

6. Hematology: Basic Principles and Practice. Hoffman R, Benz EJ, Silberstein LE, Heslop HE, Weitz JI, Salama ME, Abutalib SA, eds. Clinical Manifestations and Treatment of Acute Myeloid Leukemia. **Erba, Harry P.** Elsevier 2023: 950-976.

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Consultant appointments: (Include US government, state, private organizations, etc.)

Daiichi Sankyo Pharmaceutical
Kura Oncology
Servier
Sumitomo Pharma

Honors and Awards

1978 -1979	Science and Engineering Awards, Yale University
1979	B.S. (summa cum laude), Yale University
1979 -1980	Fulbright-Hays Fellowship, University of Leicester, Leicester, England
1995	Scholar Award, American Society of Hematology
2002	Teacher of the Year, 2002, Division of Hematology/Oncology, Department of Internal Medicine, University of Michigan
2003	Teacher of the Year, 2003, Division of Hematology/Oncology, Department of Internal Medicine, University of Michigan
2004	Teacher of the Year, 2004, Division of Hematology/Oncology, Department of Internal Medicine, University of Michigan
2004	Outstanding Clinician Award, University of Michigan
2005	Teacher of the Year, 2005, Division of Hematology/Oncology, Department of Internal Medicine, University of Michigan
2007	Special Recognition and Appointment to the National Comprehensive Cancer Network (NCCN) Board of Producers
2008	Special Recognition and Appointment to the National Comprehensive Cancer Network (NCCN) Board of Producers
2012	League of Clinical Excellence, University of Michigan
2016	Teacher of the Year, 2016, Division of Hematology/Oncology, Department of Internal Medicine, University of Alabama at Birmingham
2017	Teacher of the Year, 2016, Division of Hematology/Oncology, Department of Internal Medicine, University of Alabama at Birmingham

Organizations and participation (Offices held, committee assignments, etc.):

Intramural Committee and Administrative Service

Harvard Collaborative Oncology Group, Lymphoma Committee (member)
Intern Selection Committee, Department of Medicine, Brigham and Women's Hospital
Protocol Review Committee, University of Michigan (member)
Leukemia Conference, University of Michigan (Organizer, 7/96 - 6/12)
Hematologic Malignancy Working Group, University of Alabama (Chair, 7/12 - 4/18)
Clinical Trials Operations Committee, University of Alabama at Birmingham (Chair, 3/13 - 4/18)

Extramural Committee, Organizational, and Volunteer Service

National Comprehensive Cancer Network, Clinical Guidelines Committee, Chronic Myelogenous Leukemia
National Comprehensive Cancer Network, Clinical Guidelines Committee, Myelodysplastic Syndromes
National Comprehensive Cancer Network, Clinical Guidelines Committee, Acute Leukemia
National Comprehensive Cancer Network, Clinical Guidelines Committee, Myeloid Growth Factors
Southwest Oncology Group (SWOG), Executive Officer (4/2005 – 10/2012)
SWOG Leukemia Committee, Chair (10/2012 - present)
NCI Leukemia Steering Committee, Member (10/2012 – present)
NCTN Myelo MATCH Initiative, Co-Chair, Senior Scientific Council (1/2019-present)

Scientific Steering Committees and DSMB Positions

Genzyme Oncology, Acute Myeloid Leukemia Steering Committee.
Sunesis Pharmaceuticals, VALOR Steering Committee
Janssen Research and Development, Chair, Independent Data Monitoring Committee, CNTO328 (siltuximab) MDS2001 protocol
Celgene, Chair, Scientific Steering Committee, AML/MDS Registry
Glycomimetics, Inc. Chair, Data and Safety Monitoring Committee, GMI-1271 Phase Ib Study
Daiichi Sankyo Inc., Co-Chair, Scientific Steering Committee, QuANTUM – First
AbbVie, Chair, Independent Review Committee for VIALE A and VIALE C

Memberships in Professional Societies

Active Member, American Society of Hematology

Active Member, American Society of Clinical Oncology

Member of the National Comprehensive Cancer Network (NCCN) Practice Guidelines Committees for acute myeloid leukemia, chronic myeloid leukemia, myelodysplastic syndromes, and myeloid growth factors (until 3/31/10).

Teaching Activities

1976 -1978	Teaching Assistant, Molecular Cytogenetics, Cold Spring Harbor Laboratory, Cold Spring Harbor, New York
1980	Teaching Assistant, Animal Cytology, School of Biological Sciences, University of Leicester, England

Stanford University:

1982	Teaching Assistant, Advanced Molecular Biology, department of Biochemistry, Stanford University
1982 -1983	Teaching Assistant, Cells and Tissues, Department of Structural Biology, Stanford University
1983	Teaching Assistant, Human Anatomy, Department of Structural Biology, Stanford University
1983 -1984	Tutor, Anatomy and Physiology, Primary Care Associate Program, Stanford University
1985	Lecturer, Anatomy of the Kidney, Renal Physiology, Stanford University

Harvard Medical School:

1989 -1990	Instruction of interns and students on the General Medicine Service, Brigham and Women's Hospital
1990 -1993	Instruction of students on Hematology Elective, Brigham and Women's Hospital
1993	Attending Physician, Hematology/Oncology Service, Brigham and Women's Hospital

University of Michigan:

1996 - 2012	Attending Rounds, Department of Internal Medicine
1996 - 2012	Clinical Outpatient Teaching, Department of Internal Medicine
1999 - 2012	M2 Hematology Course, Department of Internal Medicine, annually
2005 - 2002	Chief Rounds, Department of Internal Medicine, annually

Clinical activity - type of practice and estimate of time commitment:

1. AML Clinic (2 days/week)
2. AML disease focus clinical trials (5 days/week)
3. Attending, hematologic malignancies inpatient teaching service (8 weeks/year)

Participation in academic and administrative activities of the University and Medical Center:

1. Attend Grand Rounds for CME credits.
2. Medical Director, Hematologic Malignancies Inpatient Service.
3. Director, Leukemia program in the Division of Hematologic Malignancies and Cellular Therapy at Duke.

APPENDIX B

A. References

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B. Materials Considered

General Materials

- Expert Report of John C. Lipscomb, Ph.D., DABT (February 7, 2025)
- Expert Report of Julie E. Goodman, Ph.D., DABT, FACE, ATS – Trichloroethylene, Perchloroethylene, Benzene, Vinyl Chloride, and trans-1, 2-Dichloroethylene Exposure and NHL Risk (February 7, 2025)
- Expert Report of Julie E. Goodman, Ph.D., DABT, FACE, ATS – Trichloroethylene, Perchloroethylene, Benzene, Vinyl Chloride, and trans-1, 2-Dichloroethylene Exposure and Leukemia Risk (February 7, 2025)
- Expert Report of Michael J. McCabe, Jr., Ph.D. – Camp Lejeune Water Volatile Organic Chemicals and Non-Hodgkin's Lymphoma and Leukemia (February 7, 2025)
- Expert Report of Peter G. Shields, MD (February 7, 2025)
- Expert Report of Jay L. Brigham, Ph.D. (December 9, 2024)
- Expert Report of Dean W. Felsher, M.D., Ph.D. – Leukemia & Non-Hodgkin Lymphoma (December 9, 2024)
- Expert Report of Howard Hu, M.D., M.P.H., Sc.D. (December 9, 2024)

- Expert Report of Kathleen M. Gilbert, PhD – TCE, Non-Hodgkin Lymphoma, and Leukemia (December 9, 2024)
- Expert Report of Lukasz Gondek, MD, PhD – Leukemia (December 9, 2024)
- Expert Report of Steven B. Bird, MD – Hematopoietic Cancers: Leukemia & Non-Hodgkin's Lymphoma (December 9, 2024)
- Expert Report of Timothy M. Mallon, M.D., M.P.H, MS. – Leukemia (December 9, 2024)

Case-Specific Materials

Amsler v. United States

- Amsler Complaint (March 4, 2023)
- Amsler Short-Form Complaint (November 5, 2023)
- Amsler Discovery Pool Profile Form [00284_AMSLER_DPPF_0000000001-17]
- Amsler Track 1 Trial Plaintiff Damages Assessment [00284_AMSLER_0000011143-11145]
- Deposition Testimony and Exhibits of Dr. Jenniffer Yannucci (June 3, 2024)
- Deposition Testimony and Exhibits of Dr. John Moore (August 7, 2024)
- Deposition Testimony and Exhibits of Dr. Praneeth Baratam (June 6, 2024)
- Deposition Testimony and Exhibits of Karen Amsler (April 16, 2024)
- Deposition Testimony and Exhibits of Michael Wukitch (August 8, 2024)
- Expert Report of Judy S. LaKind, Ph.D., *Amsler v. United States* (April 8, 2025)
- Expert Report of Lisa A. Bailey, Ph.D., *Amsler v. United States* (April 8, 2025)
- Expert Report of Lukasz P. Gondek, MD, PhD, *Amsler v. United States* (February 7, 2025)
- Expert Report of Dubravka Tosic, Ph.D., *Estimated Economic Loss of Mrs. Karen Marie Amsler* (April 8, 2025)
- Report by The Center for Forensic Economic Studies, Chad L. Staller & Stephen M. Dripps, *Amsler v. United States* (February 7, 2025)
- Medical Records and other documents produced by Plaintiff [00284_AMSLER_0000000043-523, 00284_AMSLER_0000000654-7913, 00284_AMSLER_0000007923-7961, 00284_AMSLER_0000008029-8038, 00284_AMSLER_0000008047-9894, 00284_AMSLER_0000009907-9938, 00284_AMSLER_0000010300-10476, 00284_AMSLER_0000010492-10998]
- Medical Records from Adult Primary Care Waters [00284_AMSLER_APCW_0000000001-358]
- Medical Records from Lehigh Valley Health Network [00284_AMSLER_LVH_0000000001-70]
- Medical Records from Low Country Cancer Care Clinics [00284_AMSLER_AON_0000000001-272]
- Medical Records from MUSC Health [00284_AMSLER_MUSC_0000000001-777, 00284_AMSLER_MEDRECS_0000000002-2187]
- Medical Records from the Office of Dr. John Moore [00284_AMSLER_JM_0000000002-359]

Connard v. United States

- Connard Short-Form Complaint (November 11, 2023)
- Connard 1st Amended Short-Form Complaint (February 14, 2024)
- Connard 2nd Amended Short-Form Complaint (July 19, 2024)
- Connard Discovery Pool Profile Form [01557_CONNARD_DPPF_0000000001-14]
- Connard Track 1 Trial Plaintiff Damages Assessment [01557_CONNARD_DPPF_0000001670-73]
- Deposition Testimony and Exhibits of Vivian Connard (February 26, 2024)
- Deposition Testimony of Dr. Aaron Rapoport (May 7, 2024)
- Deposition Testimony of Dr. Gorgun Akpek (July 19, 2024)
- Expert Report of Judy S. LaKind, Ph.D., *Connard v. United States* (April 8, 2025)
- Expert Report of Lisa A. Bailey, Ph.D., *Connard v. United States* (April 8, 2025)
- Expert Report of Lukasz P. Gondek, MD, PhD, *Connard v. United States* (February 7, 2025)
- Expert Report of Dubravka Tosic, Ph.D., *Estimated Economic Loss of Stephen M. Connard* (April 8, 2025)
- Report by The Center for Forensic Economic Studies, Chad L. Staller & Stephen M. Dripps, *Connard v. United States* (February 4, 2025)
- Medical Records from UMMS [01557_CONNARD_UMMS_0000000001-3351]
- Medical Records produced by Plaintiff, including records from University of Maryland Greenebaum Comprehensive Cancer Center, University of Maryland Medical System, and Washington Medical Center [01557_CONNARD_0000000001-1673]

Fiolek v. United States

- Fiolek Complaint (March 31, 2023)
- Fiolek Short-Form Complaint (November 6, 2023)
- Fiolek 1st Amended Short-Form Complaint (March 15, 2024)
- Fiolek 2nd Amended Short-Form Complaint (January 28, 2025)
- Fiolek Discovery Pool Profile Form [00062_FIOLEK_DPPF_0000000001-14]
- Fiolek 1st Amended Track 1 Trial Plaintiff Damages Assessment (September 19, 2024)
- Fiolek 2nd Amended Track 1 Trial Plaintiff Damages Assessment (December 27, 2024)
- Deposition Testimony and Exhibits of Robert J. Fiolek (Vols. I and II)
- Deposition Testimony of Jeraldine Fiolek (April 25, 2024)
- Deposition Testimony and Exhibits of Dr. John Cho (May 3, 2024)
- Deposition Testimony and Exhibits of Dr. Walter Wardell (August 13, 2024)
- Deposition Testimony and Exhibits of Dr. Jeffrey Goodwin (January 8, 2025)
- Expert Report of Judy S. LaKind, Ph.D., *Fiolek v. United States* (April 8, 2025)
- Expert Report of Lisa A. Bailey, Ph.D., *Fiolek v. United States* (April 8, 2025)
- Expert Report of Max Kates, M.D., *Fiolek v. United States* (April 8, 2025)
- Expert Report of Dubravka Tosic, Ph.D., *Estimated Potential Offsets of Mr. Robert J. Fiolek* (April 8, 2025)

- Expert Report of Damian A. Laber, M.D., F.A.C.P., *Fiolek v. United States* (February 7, 2025)
- Medical Records and other documents produced by Plaintiff [00062_FIOLEK_0000000001-11678]
- Medical Records from VBA [00062_FIOLEK_VBA_0000000001-2689]
- Medical Records from VHA [00062_FIOLEK_VHA_0000000001-82]
- Medical Records from Coastal Carolina Health Care [00062_FIOLEK_CCHC_0000000001-915]
- Medical Records from CarolinaEast Medical Center [00062_FIOLEK_CEMC_0000000001-842, 00062_FIOLEK_CEMC_000000942-4436, 00062_FIOLEK_CEMC_0000004538-10874]
- Medical Records from UNC Hospitals [00062_FIOLEK_UNC_0000000001-324]

Gleesing v. United States

- Gleesing Short-Form Complaint (November 3, 2023)
- Amended Short-Form Complaint (June 18, 2024)
- Gleesing Discovery Pool Profile Form [01486_GLEESING_DPPF_0000000055-76]
- Deposition Testimony of Joseph Gleesing (April 12, 2024)
- Deposition Testimony of Charlene Gleesing (June 4, 2024)
- Deposition Testimony of Dr. Min Luo (July 22, 2024)
- Deposition Testimony of Dr. Muhammad Usman (September 26, 2024)
- Expert Report of Judy S. LaKind, Ph.D., *Gleesing v. United States* (April 8, 2025)
- Expert Report of Lisa A. Bailey, Ph.D., *Gleesing v. United States* (April 8, 2025)
- Expert Report of Damian A. Laber, M.D., F.A.C.P., *Gleesing v. United* States (February 7, 2025)
- Expert Report of Dubravka Tosic, Ph.D., *Estimated Economic Loss of Joseph M. Gleesing* (April 8, 2025)
- Report by The Center for Forensic Economic Studies, Chad L. Staller & Stephen M. Dripps, *Gleesing v. United States* (February 7, 2025)
- Medical Records from [01486_GLEESING_BMH0000000001-2395]
- Medical Records produced by Plaintiff, including records from Borgess Medical Center, Bronson Healthcare Group, University of Michigan Cancer Center (UMCC), and Ann Arbor VA Hospital [01486_GLEESING_0000000001-7186]

Hill v. United States

- Hill Complaint (November 20, 2023)
- Hill 3rd Amended Short-Form Complaint (August 16, 2024)
- Hill Discovery Pool Profile Form [00028_HILL_DPPF_0000000001-23]
- Deposition Testimony and Exhibits of Bruce Hill (April 9, 2024)
- Deposition Testimony of Kristie Hill (June 14, 2024)
- Deposition Testimony and Exhibits of Dr. Maxim Norkin (June 6, 2024)

- Deposition Testimony and Exhibits of Dr. Bradley Fletcher (June 13, 2024)
- Deposition Testimony and Exhibits of Dr. Jessica Schmit (July 15, 2024)
- Deposition Testimony and Exhibits of Stephen McCready, PA-C (May 8, 2024)
- Expert Report of Judy S. LaKind, Ph.D., *Hill v. United States* (April 8, 2025)
- Expert Report of Lisa A. Bailey, Ph.D., *Hill v. United States* (April 8, 2025)
- Expert Report of Deborah A. Navarro, MA, *Rehabilitation Evaluation and Life Care Plan of Bruce Hill* (April 8, 2025)
- Expert Report of Dubravka Tosic, Ph.D., *Estimated Economic Loss of Bruce Hill* (April 8, 2025)
- Expert Report of Dean W. Felsher, M.D., Ph.D., *Hill v. United States* (February 7, 2025)
- Report by The Center for Forensic Economic Studies, Chad L. Staller & Stephen M. Dripps, *Hill v. United States* (February 7, 2025)
- Medical Records from VBA [00028_HILL_VBA_0000000024-9561]
- Medical Records from VHA [00028_HILL_VHA_0000000001-1411]
- Medical Records from University of Florida Health Shands Cancer Hospital [00028_HILL_MEDRECS_0000000015-71]
- Medical Records and other documents produced by Plaintiff [00028_HILL_0000000005-15, 00028_HILL_0000004844-64]