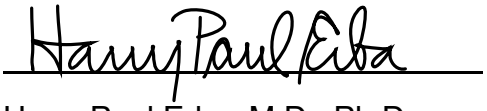
Exhibit 474

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

Vivian Connard, Representative of
Stephen Matthew Connard, deceased v. United States
7:23-cv-01557-D-RN
U.S. District Court for the Eastern District of North Carolina

Prepared By:



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

April 8, 2025

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Plaintiff: Vivian Connard for her husband Stephen Connard (date of death 05/14/2010)

Case: *Stephen Matthew Connard v. United States*, No: 7:23-cv-01557-D-RN (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 Mr. Stephen Matthew Connard's acute myeloid leukemia (AML) was caused by his exposure to the water at the Camp Lejeune military base. I was also asked to comment on Dr. Lukasz Gondek's 02/07/2025 report.

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

1. It is highly unlikely that Mr. Connard's limited exposure to water at Camp Lejeune caused his AML.
2. Mr. Connard's death was not directly caused by AML, which had been in remission for nearly a decade prior to his death.

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 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 (MDS), a role that I have occupied since 2019.

[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.

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 (MDS), 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 CLL for over 20 years at the University of Michigan and the University of Alabama at Birmingham.

The scope of my research activity has been limited to the clinical evaluation of new therapies for people with acute myeloid leukemia, acute lymphoblastic leukemia, chronic myeloid leukemia, chronic lymphocytic leukemia, myelodysplastic syndromes, and myeloproliferative neoplasms. 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 peer-reviewed publications during my entire academic life, including 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 by 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). Mr. Connard was diagnosed with AML.

A. Acute Myeloid Leukemia (AML)

Acute myeloid leukemia (AML) is a cancer of the myeloid progenitor cells that create mature blood cells.[3] The median age at diagnosis is 69 years in the Surveillance, Epidemiology, and End Results (SEER) registry. However, AML can be diagnosed at any age; 14.4% of AML patients are younger than 45 years at diagnosis. In 2000, the annual incidence of AML was 3.9 AML cases per 100,000 people (https://seer.cancer.gov/statfacts/html/amyl.html).

The etiology of AML in most patients is unknown ("idiopathic"). However, there are some known factors associated with increased risk of AML development. The incidence of AML increases with advancing age, due to the acquisition over time of specific mutations in DNA that lead to the abnormal growth of cells. Other risk factors associated with AML include (1) having a close family member with leukemia; (2) prior exposure to ionizing radiation, chemotherapy, radiotherapy, and immunosuppressant drugs; (3) exposure to cigarette smoke and benzene; (4) Mendelian AML predisposition syndromes due to germline mutations of RUNX1, DDX41, GATA2, and other genes; and (5) congenital bone marrow failure syndromes such as Schwachman-Diamond syndrome, dyskeratosis congenita, Fanconi anemia, Down syndrome and others. The Danish national population-based database estimated only 5% of AML are therapy-related (Granfeldt-Ostgard LS, et al. J Clin Oncol. 2015; 33: 3641). The proportion of adult AML

[2] CLL has been re-classified as a subtype of non-Hodgkin Lymphoma. The World Health Organization now classifies this subtype as CLL/SLL.

[3] Acute myeloid leukemia has had multiple different names over the last century including acute non-lymphocytic leukemia, acute myeloblastic leukemia, and acute myelocytic leukemia. The multiple subtypes of AML add to the confusion. The following are just a few of the terms currently used for subtypes of AML: acute promyelocytic leukemia, acute monoblastic leukemia, acute myelomonocytic leukemia, acute erythroid leukemia, acute megaloblastic leukemia, blastic plasmacytoid dendritic cell neoplasm, and myeloid sarcomas.

cases due to germline AML predisposition mutations is unknown; however, 8% of cancers in adults harbor germline cancer predisposition mutations (Huang KL, et al. Cell 2018; 173: 355-370). Congenital bone marrow failure syndromes are very rare. Therefore, most AML cases (85-90%) are idiopathic.

AML due to prior exposure to DNA damaging agents such as alkylating chemotherapy and ionizing radiation typically occurs within 10 years of exposure. In a series of 65 patients exposed to alkylating agent chemotherapy and/or radiation therapy, the latency ranged from 11 to 192 months with a median of 58 months (Michels SD, et al. Blood 1985; 65: 1364). However, only 6 of 65 cases (14%) had a latency over 120 months (10 years).

Blood cells have a finite existence, with red blood cells living approximately 100 days before being removed by the spleen. Platelets remain in the blood for about 7-10 days, and neutrophils remain in the blood for less than one day before exiting into tissues to prevent infection by bacterial and fungal organisms. In AML, normal myeloid progenitor cells are prevented from developing into mature blood cells. Therefore, the natural consequence of AML is for these vital blood cells to decrease rapidly. Anemia, the term used for a low red blood cell count (or low hemoglobin or low hematocrit), can cause fatigue, shortness of breath during activities, and headaches. If the platelet count is low (a condition that is called thrombocytopenia), there may be unexplained bruising, nose bleeds, or more serious abnormal bleeding such as bleeding from the gastrointestinal tract or even bleeding in the brain, which can be rapidly fatal. Neutropenia is a low neutrophil count and is associated with opportunistic infections by bacteria in the upper respiratory tract, the gastrointestinal tract, and the skin. Neutropenia can also make people susceptible to infections by various airborne fungal spores, which can be life-threatening.

B. Treatment of AML

Patients with AML are typically administered a chemotherapy regimen to achieve complete remission followed by further chemotherapy to eradicate any residual disease. In most cases, this treatment will not be curative alone. If remission is achieved but the chance of relapse is high, we will recommend an allogeneic hematopoietic stem cell transplant ("allo HSCT") if the patient is healthy enough to receive this therapy. Since 2018, less intensive therapeutic options have become available for older AML patients, who may be unable to tolerate intensive chemotherapy. These regimens can extend survival but are not curative and must continue to maintain the response as long as possible. However, these less intensive treatment regimens are effective even in relapsed AML and AML with adverse cytogenetic analysis, such as monosomy 7 and other monosomal karyotypes, and are now considered as appropriate treatments even for younger patients with adverse risk AML.

The first step in allo HSCT is to give the recipient (patient) a "preparative regimen," also known as conditioning regimen. The preparative regimen consists of chemotherapy with or without total body radiation. The purpose of the preparative regimen is to eradicate cancer (leukemia) from the body 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. Stem cells obtained from the umbilical cord of volunteer donors are also used for this purpose.

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.

IV. Summary of Exposure

According to his military records, Mr. Stephen Connard was stationed at Camp Lejeune from October 1977 until July 1981, except for an overseas deployment from February 1979 until March 1980. (See Exposure Report of Judy S. LaKind). Mr. Connard's surviving spouse, Mrs. Vivian Connard, testified that she believed Mr. Connard lived at Mainside Barracks while at Camp Lejeune, but she did not know any details about his water usage.

Drs. LaKind and Bailey have calculated Mr. Connard's exposure to Camp Lejeune water conservatively, such that there is little to no likelihood that his actual exposure was greater than their estimates. According to their assessments, assuming a military high-end exposure to benzene, Mr. Connard's increased lifetime risk of AML was only 0.0001%, or 1 AML case in 1,000,000 exposed people. (See Risk Assessment of Lisa Bailey, p. 33).[4]

[4] Dr. Bailey explained that Mr. Connard's estimated increased lifetime risk of cancer generally of 0.01%, or 1 cancer case in 10,000 exposed people, is driven primarily by TCE and vinyl chloride in drinking water, which are not predictive of leukemia (AML) risks. (Risk Assessment of Dr. Bailey, p. 33). In any event, Dr. Bailey also notes that Mr. Connard's estimated increased risk of developing any cancer during his lifetime did not exceed the EPA's target excess cancer risk range.

V. Mr. Connard's Relevant Medical History

Mr. Connard (DOB 07/15/1959) was 41 years old at the time of his diagnosis of AML on March 25, 2001, which was approximately 20 years after he left Camp Lejeune. Although I do not have any records from prior to his diagnosis, Mr. Connard appears to have been in good health. Mr. Connard had a family history of cancer. His mother was a smoker and had both lung and throat cancer. (01557_CONNARD_0000000091). He also had a family history of hypertension and heart problems. (01557_CONNARD_0000000211). There was no family history of leukemia.

The indication for the bone marrow biopsy on 03/26/2001 was "New Onset Pancytopenia." (01557_CONNARD_0000001668). Pancytopenia refers to a condition in which the number of white blood cells, red blood cells, and platelets are all below the normal range. Pancytopenia often suggests a pathologic process in the bone marrow, requiring a bone marrow biopsy for diagnosis. Depending on the degree to which the blood counts are depressed, a patient may experience fatigue, dizziness, fever, infection, shortness of breath, and easy bruising. Mr. Connard had undifferentiated AML by the French - American – British classification (FAB M0). (01557_CONNARD_0000000021).[5] Cytogenetic analysis demonstrated monosomy 7 (loss of one of the two #7 chromosomes), a recurring, somatically acquired chromosomal abnormality in AML blasts. The presence of monosomy 7 has been associated with a poor prognosis in adults with AML. His diagnosis pre-dated the routine use of Next Generation Sequence analysis for detection of either somatic or germline pathogenic mutations.

Mr. Connard received a timed sequential chemotherapy regimen, which is aimed at improving the rate of complete remission. The initial chemotherapy treatment in this approach is intended to reduce the number of leukemia cells in the body but can also trigger more quiescent leukemic blasts to enter the cell cycle.[6] A second round of chemotherapy with a cell cycle specific drug (typically high dose cytarabine) is then administered before the patient recovers from the first chemotherapy treatment. Although Mr. Connard's leukemic blasts were initially cleared by this regimen, the leukemia was already recurring as his bone marrow was recovering from the therapy.

Next, Mr. Connard received an investigational salvage chemotherapy regimen consisting of mitoxantrone, cytarabine, and bevacizumab. Bevacizumab is a monoclonal antibody that blocks vascular endothelial growth factor, preventing the formation of new blood vessels needed for growth of tumors. Mr. Connard did not respond to this therapy, and developed high blood pressure (hypertension, abbreviated in the medical record as HTN) related to the bevacizumab. Therefore, Mr. Connard had primary refractory chemotherapy, which is defined as having persistent leukemia despite two cycles of intensive chemotherapy.

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[5] Undifferentiated AML-M0 is a rare subtype of AML in which the blasts do not resemble any particular line of normal myeloid differentiation (e.g. granulocytic, monocytic, erythroid, etc.). M0 AML is not indicative of the etiology of the AML.

[6] Chemotherapy agents such as cytarabine are cell-specific drugs, that must be incorporated into DNA during cellular replication to cause subsequent cell death by apoptosis. Quiescent leukemic blasts are not in cell cycle (so-called, G0 phase) and are not replicating their DNA. Therefore, these leukemic cells are resistant to chemotherapy and can cause relapse of the leukemia.

The intensive course of two separate therapeutic chemotherapy regimens was complicated by fungal pneumonia, which was treated with amphotericin, and renal insufficiency, both of which were resolved prior to further therapy for the AML. (01557_CONNARD_0000000021-22). Mr. Connard then proceeded with allo HSCT.

Mr. Connard's allo HSCT began with a preparative regimen of cyclophosphamide 60 mg/kg IV daily for 2 days (specifically, 07/23/2001 and 07/24/2001). The purpose of this chemotherapy was to eradicate the disease as well as suppress his immune system before receiving a transplant. To prevent GVHD, he received an immunosuppressive regimen consisting of the calcineurin inhibitor tacrolimus, mycophenolate, and daclizumab (an investigational CD25 blocking monoclonal antibody, not FDA approved for this indication). He then received intravenous infusions of hematopoietic stem cells from an HLA-matched unrelated donor on 07/26/2001 and 07/27/2001. (01557_CONNARD_0000000023-24). Despite initial complications during his allo HSCT,[7] Mr. Connard fortunately achieved complete remission shortly after his AML treatment, which he maintained for the remainder of his life. The absence of relapse over a nine-year period is quite remarkable, since he underwent allo HSCT with active AML, and he received a reduced intensity preparative regimen. It is very likely that the allograft (transplanted stem cells) was able to immunologically eradicate any residual leukemic cells (graft-versus-leukemia effect).

By December 2005, Mr. Connard had developed chronic graft versus host disease (cGVHD), initially characterized by mouth ulcers and intermittent skin rashes, which his doctor noted was stable and generally well-controlled by medications. (01557_CONNARD_0000000320-322, 282, 277, 273, 257, 255, 250). His energy level was good, and he enjoyed riding his motorcycle regularly. (01557_CONNARD_0000000292).

Toward the end of 2008, his cGVHD progressed with thickening of the skin and difficulty bending at the waist. The cGVHD of the skin significantly improved with photopheresis (01557_CONNARD_0000000152, 135, 116, 123, 108).[8] Mr. Connard also had low antibody levels (hypogammaglobulinemia), commonly seen in cGVHD, and associated with frequent infections. Prior to his terminal hospital admission in 2010, Mr. Connard had Scedosporium (a fungal organism capable of invading tissues) pneumonia, Nocardiosis, and influenza. (01557_CONNARD_0000000097). He was given monthly intravenous infusions of antibodies from blood donors (gamma globulin) and prophylactic antibiotics to prevent infectious complications.

Mr. Connard developed an Achilles tendon rupture of unclear etiology (01557_CONNARD_0000000097), squamous cell carcinoma of the left cheek and scalp

[7] Mr. Connard's immediate post-transplant course was complicated by febrile neutropenia (fever due to having a low white blood cell count requiring intravenous antibiotics), grade III acute GVHD of the gut and skin (see above), mastoiditis (infection of the air cells in the temporal bone behind the ear), renal insufficiency (kidney failure), pleural effusions (fluid around the lungs causing difficulty breathing), hyperglycemia (high blood sugar) from steroid-induced diabetes mellitus, and urinary tract infection.

[8] Photopheresis is a treatment where blood is taken from the patient, white blood cells are then separated, treated with medication and ultraviolet light, and then placed back into the patient.

(01557_CONNARD_0000000113-114), and difficulty swallowing, which improved after an esophageal dilation (01557_CONNARD_0000000108).

On 05/06/2010, Mr. Connard presented to the Bone Marrow Transplant outpatient clinic with symptoms of pneumonia. He had dyspnea (labored breathing), fever, and hypoxia (low blood oxygen level). Chest radiograph and CT scan showed multiple pulmonary opacities consistent with pneumonia. He required admission to the Medical Intensive Care Unit for management of hypoxic respiratory failure, including intubation and mechanical ventilation. Mr. Connard received antibiotics, and he underwent bronchoscopy (after antibiotics were already started). All cultures obtained from his lungs were sterile (without bacterial growth), and the infectious serologies were negative. A specific infectious cause of the pneumonia was not determined, but he clinically improved on antibiotics and was extubated. This is not unusual, since he had already started antibiotics.

At the time of this admission, Mr. Connard had an elevation of a cardiac enzyme troponin, which is consistent with heart muscle damage (cell death). The doctors suspected that Mr. Connard had experienced a non-ST segment elevation myocardial infarction. (01557_CONNARD_0000000212). Ultrasound of the heart (echocardiogram) demonstrated reduced left ventricular systolic function with an ejection fraction of 35% (normally, greater than 50%), moderate pulmonary hypertension, and diastolic dysfunction (01557_CONNARD_0000000031-32). He underwent a coronary artery angiogram, which revealed only mild, non-obstructive coronary artery disease without infarct-related coronary artery disease (01557_CONNARD_0000000043, 212). The cardiac troponin elevation was likely related to demand ischemia, an imbalance between the oxygen supply and the increased workload of the heart. Myocardial infarction due to demand ischemia is now called type 2 myocardial infarction.

On 05/11/2010, intravenous Amphotericin was changed to oral voriconazole. Repeat echocardiogram on 05/13/2010 (the day before his death) showed recovery of the left ventricular function with mild pulmonary hypertension and a trivial pericardial effusion (01557_CONNARD_0000000033-34). There was no mention of an arrhythmia during the echocardiogram. The last ECG in the record before starting voriconazole showed QTc interval 483 milliseconds (grade 2 QT prolongation), which is not typically associated with the risk of ventricular arrhythmia and sudden death.

I have reviewed the progress note on the day before his death (01557_CONNARD_0000000040-41) and labs on the day of his death. Potassium, magnesium, and calcium were all normal. Brain natriuretic peptide (BNP) had decreased to 505, indicating an improvement in his cardiac function. Oxygen saturation also was normal. He continued voriconazole prophylactically. He was hypotensive (but asymptomatic). All medications for myocardial infarction including anticoagulation and congestive heart failure were discontinued (nitrate, hydralazine, and metoprolol). His blood pressure also had improved by that time (01557_CONNARD_0000000048-49). He felt better the morning of 05/14/2010, and he was eager to go home.

He had a cardiopulmonary arrest on 05/14/2010 while in the CT scan for follow up chest imaging prior to the planned discharge. There are only two notes in the provided record from this event: the attending bone marrow transplant physician (01557_CONNARD_0000000050) and the anesthesiology resident performing an emergency intubation (01557_CONNARD_0000000233). The only details of the event are in the anesthesiology note. Two different terms are listed for this event in this hand-written note: PEA and asystole. In one place, the term PEA is crossed out and replaced by asystole. However, the term PEA is included twice in the event table with CPR listed below both times. Furthermore, the attending physician's note indicates he had a pulseless cardiac arrest (PEA). PEA is an abbreviation for pulseless electrical activity, indicating normal sinus rhythm (normal cardiac electrical activity) but without palpable pulses. Asystole is an electrocardiographic term indicating no electrical activity in the heart (flat line). The record of the event does not include any mention of attempts at electrical cardioversion or administration of drugs used in asystole (atropine, epinephrine), causing me to question the diagnosis of asystole. Cardioversion and epinephrine would be appropriate therapies for ventricular arrhythmias, such as ventricular tachycardia and ventricular fibrillation, that can lead to sudden death such as in this case. Atropine is the recommended medical treatment of asystole. However, the attending physician's hospital note contained the following quote: "Mr. Connard had cardiac arrest at ER CT scan radiology unit while preparing to have a CT scan of the chest. He was intubated and started on CPR. No pulse (PEA) despite 30 minutes of continuous CPR under anesthesia" (01557_CONNARD_0000000233).

The death certificate lists acute myeloid leukemia as cause of death and heart failure as a contributing cause. He had neither AML nor heart failure at the time of death. He was cremated. His wife, a nurse, declined post-mortem examination, indicating in her testimony that he had already been through enough (page 130 of Mrs. Connard's deposition). There is no final hospital discharge note from 05/14/2010 in the provided record; the only discharge note was the transfer from the Medical Intensive Care Unit to the Bone Marrow Transplant unit.

VI. Analysis

A. It is highly unlikely that Mr. Connard's limited exposure to water at Camp Lejeune caused his AML.

Neutrophils and monocytes (types of white blood cells), red blood cells, and platelets must be produced continuously in the bone marrow throughout a lifetime. Humans are born with a finite number of hematopoietic stem cells (HSC) that provide the continuous source of blood cell formation. Given the critical role of HSC throughout life, HSC are protected from DNA damaging events that lead to cellular apoptosis (cell death) or neoplastic transformation. For example, the bone surrounding the marrow spaces provides some degree of protection from the DNA damaging effects of ambient ionizing radiation. There are "safe" levels of radiation. Likewise, DNA damaging chemicals must first reach the HSC compartment in the bone marrow to cause harm. The liver metabolizes many substances. Nonetheless, some of these metabolites can also cause DNA damage, which is true for benzene. However, the kidneys eliminate these substances and

their metabolites. If these foreign substances do reach the bone marrow, HSC have at least two mechanisms that protect against DNA damage. HSC express drug efflux pumps such as P-glycoprotein on their cell surface that can eliminate harmful chemicals from reaching the DNA in the HSC nucleus. Furthermore, there are DNA repair enzymes that can fix damage caused by chemicals such as alkylating agents. Therefore, like ionizing radiation, there is a threshold amount of exposure to DNA damaging chemicals that is required to overcome these protective mechanisms and cause DNA damage followed by either cellular apoptosis or leukemic transformation.

Secondary AML (not de novo, or idiopathic) is associated with exposure to cytotoxic chemotherapy, ionizing radiation, and environmental toxins. AML cases due to DNA-damaging chemotherapy agents (such as alkylating agents) typically occur within 10 years of exposure. The risk of AML following such exposure has been directly associated with the dose as well (for example, see Curtis RE, et al. N Engl J Med. 1992; 326: 1745-51; Jonsdottir G, et al. Eur. J. Haematol. 2021; 107: 275-282). Secondary AML cases often have adverse cytogenetic features like monosomy 7 and are refractory to chemotherapy. However, these clinical and pathologic findings are not specific for therapy-related AML (tAML). In the Danish registry study, 40% of therapy-related AML patients had adverse risk karyotypes (like monosomy 7) but so did 18% of de novo AML patients (Granfeldt-Ostgard LS, et al. J Clin Oncol. 2015; 33: 3641).

As Dr. Goodman explains in her general causation report, there is little data to support a causal connection between leukemia and TCE or PCE. In the analysis of the New Jersey drinking water contamination, there was no increased risk of AML at any TCE exposure level (Cohn P, et al. Environ Health Perspectives 1994; 102: 556-561). The same is true for all leukemia subtypes and PCE exposure.

Dr. Goodman agrees that benzene can cause AML, but she concludes that sufficient exposures are required. The details of the high-quality Chinese workers study and the Pliofilm study discussed in Dr. Goodman's report are critical to understand in relation to Mr. Connard's AML. In those studies, a statistically significant increase in AML was demonstrated in workers who were exposed to at least 40-75 ppm-years of cumulative benzene exposure, and the latency between exposure and development of leukemia was generally within 10 years (see Dr. Goodman's 02/07/2025 report at 87-88).

In Mr. Connard's case, he was at Camp Lejeune for less than three years, and, as Dr. Bailey estimates in her risk assessment report, his cumulative exposure to benzene was at most 0.00033 ppm-years, which is orders of magnitude below the thresholds of 40-75 ppm-years indicated in the Chinese workers and Pliofilm studies. As Dr. Bailey explains, Mr. Connard's estimated lifetime increase of risk was 0.0001%, which is equivalent to 1 in 1,000,000 exposed people. Given the annual incidence of AML in the year 2000 was 3.9 cases per 100,000, Mr. Connard's increased AML risk was significantly below the expected.

Regarding latency, Mr. Connard developed AML just shy of 20 years (19.75, to be exact) after leaving Camp Lejeune, which is nearly twice the ten-year latency periods demonstrated in the Chinese workers and Pliofilm studies. Although it is conceivable that Mr. Connard could have had an undiagnosed myelodysplastic syndrome prior to progression to AML, documentation of his

blood counts prior to his initial diagnosis is not available. Moreover, the indication listed for his diagnostic bone marrow biopsy in 2001 was "new onset pancytopenia", implying that his blood counts were known to be normal prior to this event.

I am aware that the comparison of Marines and Navy personnel stationed at Camp Lejeune and Camp Pendleton between 1975 and 1985 does demonstrate a statistically significant increase in the incidence of AML among those at Camp Lejeune (Bove FJ, et al. Environ. Health Perspective 2024; 132: 107008-1). However, the analysis does not include important covariates for the risk of leukemia, including tobacco use and family history. Even more importantly, as Dr. Goodman discusses in her report, the study has a high likelihood of exposure misclassification due to lack of individual exposure information.

The cancer diagnoses in the Bove analysis were collected over a 20-year period between 1996 and 2017. Therefore, there was a minimum of 10 years between the end of the exposure and collection of the cancer diagnosis. Since myeloid neoplasms generally occur within 10 years of exposure to benzene or cytotoxic chemotherapy, the observed results at least raise the possibility that the detected differences were more likely related to events in the lives of these people following their time at these two military camps. For example, since 5% of AML cases are therapy-related (Granfeldt-Ostgard LS, et al. J Clin Oncol. 2015; 33: 3641), an unmeasured imbalance in the use of chemotherapy and radiation over time to treat common solid tumors (e.g., smoking-related cancers such as lung cancer and esophageal cancer) in these two populations could very well have affected the observed difference in the incidence of AML and/or MDS.

B. Mr. Connard's death was not caused by his AML.

The use of timed sequential chemotherapy has fallen out of favor and is no longer used as initial AML therapy. This treatment approach was based on pre-clinical models, but unfortunately was associated with significant morbidity and mortality and did not appear to improve overall survival of AML patients. However, remarkably Mr. Connard experienced a durable complete remission following allo HSCT, despite having primary refractory disease. One of the most encouraging reports supporting the application of allo HSCT for primary refractory AML at the time of Mr. Connard's presentation suggested relapse-free survival and overall survival at 3 years of 31% and 30%, respectively (Fung HC, et al. Biol Blood Marrow Transplant 2003; 9: 766). However, in those patients with adverse risk karyotype, like monosomy 7, only 3 of 21 patients were still alive 2-3 years post allo HSCT.

The diagnosis of AML results in a significant impact on a person's quality of life. Mr. Connard's initial chemotherapy was followed by allo HSCT, all of which was marked by extended hospitalizations and life-threatening complications. It does appear, however, that Mr. Connard enjoyed an improved quality of life until the last two years of his life, when he developed complications of his chronic GVHD. He lost weight. He could not ride his motorcycle due to weakness. Nevertheless, according to his wife's testimony, he insisted on working until the end.

The underlying pathologic event leading to Mr. Connard's last hospitalization in May 2010 was multi-focal pneumonia and respiratory failure, which was treated successfully with antibiotics. At the time of presentation, there was global decrease in cardiac function without any regional wall motion abnormalities, arguing against a typical "heart attack" or myocardial infarction. In support of this hypothesis, the coronary arteriogram did not find any significant coronary artery blockages. The troponin leak was likely due to demand ischemia in the setting of hypoxia and infection. Cardiac muscle damage due to a mismatch of oxygen supply to the heart muscle and the demands of the increased cardiac activity as a pump is now termed "type 2 myocardial infarction". Type 1 myocardial infarction is due to occlusive disease in the coronary arteries. Mr. Connard had no evidence of significant coronary artery disease during his last hospital admission by cardiac catheterization. The transient decrease in cardiac function observed by echocardiogram was likely related to the infection, now called Takotsubo cardiomyopathy.

Based on Dr. Akpek's deposition, the cardiopulmonary arrest on 05/14/2010 was completely unexpected. He states, a patient would not be sent for an elective procedure, if he/she were clinically unstable. Although Dr. Akpek suspected a new infection, there were no clinical signs of sepsis at the time of the cardiopulmonary arrest; there was no documentation of fever or rigors. Mr. Connard's immune system was clearly compromised by the cGVHD. However, he was not neutropenic at the time of his death, and he was receiving sulfamethoxazole/trimethoprim, making Stenotrophomonas bacteremia and sepsis less likely. A ventricular arrhythmia could cause sudden death by preventing the effective delivery of blood (and oxygen) to the vital organs such as the brain. Voriconazole may cause prolongation of the QT interval on an electrocardiogram, predisposing to ventricular tachyarrhythmia such as torsades de pointes. His QT interval was already prolonged before starting the voriconazole. However, I could not find any record of an ECG after voriconazole had started (or during the cardiopulmonary arrest). Furthermore, if there was evidence of a ventricular tachyarrhythmia at the time of a cardiopulmonary arrest, the resuscitation team would have ordered epinephrine and electrical cardioversion, neither of which were given. The recent cardiac catheterization virtually excluded the possibility of a sudden occlusion of a coronary artery.

I believe there are two potential causes of Mr. Connard's very sudden and unexpected death that are not considered in the medical record or Dr. Akpek's deposition: massive pulmonary embolism or tension pneumothorax. I strongly favor the former. Pulseless electrical activity (PEA) may be due to low blood pressure due to volume loss (hemorrhagic shock), dehydration, hypoxia, and heart attack. All of these are unlikely given the patient's condition as documented in the progress note on that day and the recent results of the coronary angiogram. However, massive pulmonary embolism is a potential cause of PEA, especially in this patient who likely had a poor functional status as he recovered from pneumonia and insertion of central venous catheters that can lead to venous trauma and thrombosis (blood clot). A large blood clot (thrombosis) blocking both the main pulmonary artery and/or the right and left pulmonary arteries is often a terminal event. Tension pneumothorax results from sudden rupture of the lung, with increasing accumulation of air between the lung and the chest wall. Given the underlying lung disease related to cGVHD, recent multifocal pneumonia, and recent intubation with mechanical ventilation, Mr. Connard would be at risk of a pneumothorax. As the pressure in the chest cavity increases, the lungs

collapse, and blood return to the heart is compromised. Although this process causes PEA, the patient would very likely report chest pain and progressive shortness of breath before the pressure in the chest cavity reached catastrophic levels compromising hemodynamic stability. Since there is no suggestion of these symptoms in the medical record, I doubt a tension pneumothorax was the cause of his death. I do not believe his death was directly related to AML, as he had achieved a complete remission of AML following his allo HCST procedure almost a decade before his death on 05/14/2010. His AML did not relapse following that remission. Although the treatment for his AML may have contributed to his premature death a decade later, it was not the proximate cause of his death.

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

In my review of Dr. Gondek's expert report, I first note a factual error regarding Mr. Connard's family history; he did have a family history of cancer. His mother was a smoker, and she died from complications of lung cancer. Although this does not suggest any familial cancer predisposition syndrome, it does suggest the risk associated with secondhand smoke, which has not been considered in this case. Mr. Connard himself was not a smoker, but he was likely exposed to tobacco smoke at home and during his time at Camp Lejeune. Since there is no suggestion of a familial cancer predisposition syndrome (for example, defects in DNA repair enzymes), we have no reason to suspect that Mr. Connard may have been more sensitive to DNA damaging agents.

I also disagree with Dr. Gondek's implication that the presence of monosomy 7 indicates "exposure to genotoxic therapies and environmental exposures to DNA damaging agents such as benzene." As discussed above, monosomy 7 can be seen in patients with therapy-related AML, as well as de novo (or idiopathic) AML.

Also contrary to Dr. Gondek's opinion, it is highly unlikely that TCE and PCE contributed to Mr. Connard's AML. As the Cohn study of the New Jersey water contamination demonstrates, the evidence does not support a causal association between TCE or PCE exposure and AML.

Finally, although Dr. Gondek cites the manuscripts by Glass and colleagues, he fails to note an important observation made by Dr. Glass regarding latency between benzene exposure and AML diagnosis. "[Dr. Glass and colleagues] showed that the risk of leukemia is associated with exposure within 15 years of diagnosis, the association with exposure prior to this period is weak." (second paragraph on page 85 of Glass D, et al. Ann. N. Y. Acad. Sci. 2006; 1076: 80-89). This further supports my opinion that Mr. Connard's development of AML 19.75 years after his departure from Camp Lejeune indicates that his AML was not caused by any chemicals in the water at Camp Lejeune.

VIII. Conclusions

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

1. In light of (1) the lack of a causal connection between AML and TCE or PCE; (2) Mr. Connard's less than three-year exposure to small amounts of benzene, increasing his lifetime risk of AML by a maximum of 0.0001% (1 in 1,000,000 exposed people); and (3) his development of AML nearly 20 years after his exposure to the water at Camp Lejeune, it is highly unlikely that Mr. Connard's limited exposure to water at Camp Lejeune caused his AML.

2. Mr. Connard's death, although possibly related to the complications associated with his AML treatment, was not directly caused by his AML, which had been in remission for nearly a decade prior to his death.

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

Publications (Peer-reviewed Manuscripts):

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examining enriched leukemic blasts rather than mononuclear cells from acute myeloid leukemia patients. Biomarker Research volume 11, Article number: 31 (2023).

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139. Jacob S Appelbaum, Andrew H Wei, Sumithra J Mandrekar, Ing S Tiong, Chong Chyn Chua, Tse-Chieh Teh, Chun Yew Fong, Stephen B Ting, Daniela Weber, Axel Benner, Hannah Hill, Maral Saadati, Jun Yin, Richard M Stone, Guillermo Garcia-Manero, Harry P Erba, Geoffrey L Uy , Guido Marcucci, Richard A Larson, Abin Thomas, Sylvie D Freeman, Nuria Marquez Almuina, Konstanze Döhner, Ian Thomas, Nigel H Russel, Hartmut Döhner, Megan Othus, Elihu H Estey, Roland B Walter. Clinical evaluation of complete remission (CR) with partial hematologic recovery (CRh) in acute myeloid leukemia: a report of 7235 patients from seven cohorts. Leukemia 2024 Feb;38(2):389-392

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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.

7. The International Consensus Classification of Myeloid and Lymphoid Neoplasms. Arber DA, Borowitz MJ, Cook JR, de Level L, Goodlad JR, Hasserjian RP, King RL, Kvasnicka HM, Orazi A, eds. Acute Myeloid

Leukemia with t(9;11)(p21.3;q23.3)/*KMT2A*::*MLLT3* and Other *KMT2A* Rearrangements. Tam W, **Erba HP**, Liu Y-C. Wolters Kluwer 2025: 202-207.

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]