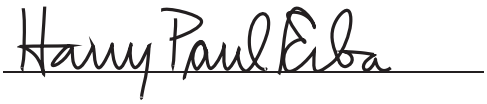
Exhibit 477

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

Bruce Hill v. United States
7:23-cv-00028
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: Bruce Hill

Case: *Bruce Hill v. United States*, Civil Action No: 7:23-cv-28-D-KS (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 hematologist and leukemia specialist on whether Mr. Bruce Hill's chronic lymphocytic leukemia (CLL) was, in fact, caused by his exposure to the water at the Camp Lejeune military base in North Carolina. I was also asked to comment on Dr. Dean Felsher's February 7, 2025, report.

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

1. It is highly unlikely that Mr. Hill's CLL was caused by his distant, intermittent exposure to the water at Camp Lejeune. Not only is there insufficient evidence of an association between CLL and the contaminants alleged in the Camp Lejeune water, but Mr. Hill's demographic profile has widely accepted features associated with a higher risk of developing CLL: adult age, male gender, and obesity. Despite these associated demographic features, there are no environmental factors or genetic events that are known to cause CLL.
2. Mr. Hill has other known risk factors for chronic kidney disease, including cardiovascular risk factors of hypertension, hyperlipidemia, obesity, glucose intolerance, and positive family history for arterial occlusive disease (myocardial infarction). Mr. Hill also has other findings of arterial vascular disease, including hypertensive retinopathy and coronary artery disease (90% occlusion of the posterior descending artery of the heart).
3. There is no reference to hepatic steatosis in Mr. Hill's medical records.

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 CLL patients.

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, 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 CLL for over 20 years at the University of Michigan and the University of Alabama at Birmingham. Although I now spend much of my clinical time treating patients with acute leukemia, I am currently treating a CLL patient at the Duke Blood Cancer Institute with the combination of obinutumomab and venetoclax. As Chair of the SWOG Leukemia Committee I have contributed to the clinical research studies leading to the approval of the current treatments for CLL. 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 received $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 a 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

[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.

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 acute or chronic based on the rate of disease progression, the type of cancer cells that accumulate, and treatment methodology. Acute leukemias are cancers that rapidly expand in the bone marrow and require immediate chemotherapy (within days of initial presentation of the patient). With some exceptions, most adult patients with acute leukemia will require hematopoietic stem cell transplant (also known as bone marrow transplant or blood and marrow transplant) after achieving an initial complete remission as the only potentially curative option. The two major types of acute leukemia are acute myeloid leukemia and acute lymphoblastic leukemia.

Chronic leukemias progress more slowly. In chronic leukemias, patients have a greater number of mature blood cells. The two major types of chronic leukemia are chronic myeloid leukemia (CML) and chronic lymphocytic leukemia (CLL).

A. CLL as a form of Non-Hodgkin Lymphoma

CLL is characterized by an accumulation of mature B lymphocytes in the blood, bone marrow, lymph nodes, and spleen. These lymphocytes are not functional. CLL results in suppression of the immune system. These patients have reduced ability to make antibodies and to respond to vaccinations. CLL and small lymphocytic lymphoma (SLL) are biologically equivalent diseases. If the diagnosis is made by detecting the neoplastic B lymphocytes in the blood or the bone marrow, the cancer is called CLL. On the other hand, if the diagnosis is made by biopsy of an organ of the immune system, such as lymph nodes, spleen, or other organs, the cancer is considered a subtype of non-Hodgkin lymphoma, currently called small lymphocytic lymphoma. Over the years, pathologists have realized that this is an oversimplification, since the same cancer cells can be found in lymph nodes, blood, and bone marrow, but the diagnosis was based on the first tissue to be biopsied. Oncologists treating these patients realized that the prognosis and treatment of CLL and SLL are similar. Therefore, CLL has been re-classified as a subtype of non-Hodgkin lymphoma.

The World Health Organization now classifies this subtype of non-Hodgkin lymphoma as CLL/SLL. The name CLL has been used for many decades. However, the name of this specific subtype of non-Hodgkin lymphoma has changed several times over the last 100 years.

Most recently, the Revised European American Lymphoma (REAL) classification and the World Health Organization (WHO) diagnostic criteria have considered CLL and SLL to be two presentations of the very same cancer. SLL is characterized by lymph node involvement with less than 5,000 neoplastic B lymphocytes per microliter of blood. CLL is defined as having over 5,000 neoplastic B cells per microliter of blood with or without other organ involvement. Monoclonal B lymphocytosis (MBL) of uncertain significance is diagnosed by the presence of less than 5,000 CD5 positive, CD23 positive monotypic B cells per microliter of blood. MBL is NOT considered cancer, but MBL patients may develop CLL/SLL many years later. MBL has been identified in 3.5% of people over 40 years of age [Rawstron AC, et al. Blood 2002; 100: 635-639].

Given the evolution of lymphoma diagnosis and classification over the last 50 years, older epidemiologic and animal toxicology reports can be difficult to apply in the current era. Furthermore, the etiology of all non-Hodgkin lymphoma is not necessarily the same. Antigenic stimulation has been hypothesized to underly several subtypes of lymphoma.

B. Epidemiology and risk factors associated with the development of CLL

The annual incidence of CLL in the United States is 6.75 and 3.65 cases per 100,000 men and women, respectively. Although the incidence of CLL and AML are similar, the prevalence of CLL is higher due to the longer life expectancy of these patients. CLL accounts for 25-35% of all leukemia cases. CLL is a disease of advancing age; the median age at diagnosis of CLL is 70 years. According to the SEER data, less than 2% of patients are diagnosed prior to age 45 years. The incidence of CLL increases with advancing age: 7.4% ages 45-54 years, 22.3% ages 55-64 years, 32.3% ages 65-74 years, 24.6% ages 75-84 years, and 11.6% > age 84 years (https://seer.cancer.gov/statfacts/html/clyl.html). CLL is less common in Asian countries, and the incidence remains low even after immigration to the Western countries. The incidence of CLL in African Americans and Asian Pacific Islander Americans is lower than in Caucasian Americans. Nonetheless, CLL can be found among any racial group. The annual incidence per 100,000 persons is 1.6 for Asian, 2.6 for Hispanics, 4.5 for non-Hispanic Blacks, and 7.8 for non-Hispanic Whites. There is also a familial tendency with a higher-than-expected frequency of CLL among first degree relatives of CLL patients. These, and other, observations suggest that genetic factors are likely to predispose to CLL [Swerdlow SH, et al. WHO Classifications of Tumours of Haematopoietic and Lymphoid Tissues, 2008; 4th edition, page 180]. Therefore, advanced age, gender, ethnicity, and family history of CLL are the most widely accepted risk factors associated with the development of CLL. However, the cause of CLL is unknown, and a specific causative agent or genetic event has not been identified. CLL occurs in adults of either gender, any ethnicity, and with or without a family history of CLL.

Environmental exposures are clearly associated with cancer risk. However, the American Cancer Society only recognizes radon exposure as a potential risk factor for CLL. Agent Orange and pesticides have been suggested as risk factors for development of CLL, but these associations have not been confirmed (see www.cancer.org/cancer/types/chronic-lymphocytic-leukemia/causes-

risks-prevention/risk-factors). Volatile organic chemicals are not listed as possible risk factors for the development of CLL on the American Cancer Society website.

The InterLymph Non-Hodgkin Lymphoma Subtypes Project evaluated the incidence of specific non-Hodgkin lymphoma subtypes according to self-reported medical history, family history, occupation, and lifestyle [Morton LM, et al. J Natl Cancer Inst Monogr. 2014; 48: 130-144]. "Risks differed statistically significantly among lymphoma subtypes for medical history factors (autoimmune diseases, hepatitis C virus seropositivity, eczema, and blood transfusion), family history of leukemia and multiple myeloma, alcohol consumption, cigarette smoking, and certain occupations, whereas generally homogeneous risks among subtypes were observed for family history of lymphoma, recreational sun exposure, hay fever, allergy, and socioeconomic status." This observation again illustrates that the subtypes of non-Hodgkin lymphoma are distinct pathologic entities and cannot be necessarily considered as one. Any self-reported family history of leukemia was associated with a 2.41 relative risk of development of CLL; the confidence interval was statistically significant, 1.85 – 3.14. Although CLL could have accounted for most of this effect, any family history of leukemia was associated with a higher risk of CLL.

Obesity has been associated with an increased risk of cancer in general. Obesity is defined as a body mass index greater than or equal to 30 kg/m^2. J.J. Castillo and colleagues performed a meta-analysis of studies evaluating the relation between obesity and the risk of leukemia and the associated mortality [Castillo JJ, et al. Leuk Res. 2012; 36: 868-875]. They first identified 1,778 manuscripts dealing with obesity and leukemia. They narrowed their meta-analysis analysis to 16 prospective cohort studies on the incidence of leukemia associated with obesity. Ten of these studies reported specifically on CLL. A statistically significant increased risk was only observed in obese men (not just overweight, and not in women). The incidence increased by 1.3% for every 1 kg/m^2 increase in the body mass index. The linear relation between the incidence of CLL and body mass index supports a direct relation. Marshall Lichtman, president of the American Society of Hematology in 1989, concluded in his peer-reviewed investigation of obesity and hematologic malignancies, that there is a significant association between obesity and the risk of chronic lymphocytic leukemia [see pages 1093 and 1097 of Lichtman, M. The Oncologist 2010; 15: 1083-1101].

CLL is preceded by an expansion of clonally related B cells with an immunophenotype identical to CLL cells (see section on CLL diagnosis below for definition), termed monoclonal B cell lymphocytosis (MBL). MBL is arbitrarily defined as less than 5000 monoclonal B cells per microliter of blood (values above 5000 per microliter define CLL). People with higher levels of MBL are at higher risk of developing CLL. Landgren and colleagues conducted a prospective cohort study based on 77,469 healthy adults enrolled in the population-based, U.S. Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial. Forty-five participants were subsequently diagnosed with CLL during the period of longitudinal observation. Using highly sensitive flow cytometry, 44 of these 45 CLL patients (98%) had MBL prior to the diagnosis of CLL. MBL was detected up to 6.4 years prior to CLL diagnosis in these individuals. Therefore, virtually all CLL patients have MBL for years prior to diagnosis. The annual risk of progression of MBL to CLL has been estimated to be 1-2% (see review by Shanafelt TD, et al. Leukemia 2010; 24: 512-520).

The pathogenesis of CLL is unknown. However, antigen interactions mediated by the B-cell receptor immunoglobulin are critical for the survival and proliferation of CLL malignant cells. In the largest analysis to date of over 29,000 samples from patients with CLL (see Agathangelidis A, et al. Blood 2021; 137: 1366), there was limited use of the available immunoglobulin V, D, and J segments in the CLL samples compared to normal B lymphocytes. For example, MBL are more likely to have immunoglobulin gene rearrangements with the IgHV-3 sequences. Since the V, D and J segments of the immunoglobulin molecule determine antigen binding, this stereotypy suggests the hypothesis that CLL is initiated by expansion of B lymphocytes in response to a limited number of commonly occurring antigens in the body or in the environment. Taken together, these observations suggest that CLL is initially due to autoimmunization (reaction against common endogenous antigens in the patient's own body) or a commonly encountered antigen in the environment followed by other genetic events leading to cancer. The subsequent genetic events leading to cancer have not been elucidated.

C. Diagnosis and Prognosis of CLL

Most patients with CLL are asymptomatic at the time of initial diagnosis. The diagnosis is made after discovery of an elevated white blood cell (WBC) count, and specifically, an elevated number of small, mature blood lymphocytes. The diagnosis of CLL is commonly established by flow cytometric analysis of the cells in any tissue involved by the small, mature lymphocytes characteristic of CLL. Flow cytometry detects the presence or absence of specific cell markers. These CD ("clusters of differentiation") markers provide a specific signature for each subtype of non-Hodgkin lymphoma. This signature is called an immunophenotype. The characteristic immunophenotype of CLL cells is positive for CD5 and CD23 on monotypic mature B cells with expression of CD19, CD20, and low-level expression of either kappa or lambda light chain surface immunoglobulin. The immunophenotype is distinct from other subtypes of non-Hodgkin lymphomas that may also be present in the blood, marrow, and lymph nodes, such as lymphoplasmacytic lymphoma, marginal zone lymphoma, mantle cell lymphoma, lymphoblastic lymphoma, and others.

The prognosis of CLL varies according to stage. There are five stages in the Rai staging system, commonly used in the United States. Stage 0 refers to patients with only an elevated peripheral blood WBC count with over 5000 CLL cells per microliter; this is called lymphocytosis. Stage I includes patients with lymphocytosis and enlarged lymph nodes (lymphadenopathy), stage II includes patients with lymphocytosis and enlargement of the spleen and/or liver (splenomegaly and/or hepatomegaly), stage III is lymphocytosis with anemia, and stage IV is lymphocytosis with thrombocytopenia. Stage 0 is considered low risk, stages I and II are intermediate risk, and stages III and IV are high risk. The stage informs the decision to initiate therapy (see below).

Once the diagnosis has been established, other studies are performed to provide further prognostic information and to guide initial treatment decisions. Recurrent alterations in the chromosomes in CLL cells have been recognized and are prognostically important. Deletion of the long arm of chromosome 13 [del(13q)] is the most common chromosomal change and associated with a more favorable prognosis. On the other hand, deletion of the long arm of chromosome 11 [del(11q)] or the short arm of chromosome 17 [del(17p)] are associated with a poor prognosis. Three copies of

chromosome 12 (trisomy 12, or +12) is associated with an intermediate prognosis. These changes are detected by either fluorescence in situ hybridization (FISH) or metaphases chromosome analysis (karyotype). Mutations in specific genes also impact prognosis. Mutations of the *TP53* gene are associated with a worse outcome.

The expression levels of two proteins by the neoplastic cells, CD38 and ZAP-70, have been associated with the prognosis of CLL patients. The presence of CD38 and/or ZAP-70 expression has been associated with higher WBC counts, lower platelet counts, lower hemoglobin, higher Rai stage disease, and worse overall survival [Hus I, et al. Annals of Oncol. 2006; 17(4): P683-690]. The expression levels of these two proteins were difficult to standardize, and both eventually fell out of favor as prognostic markers in CLL. The expression of ZAP-70 and/or CD38 were found to correlate with CLL cells carrying an unmutated immunoglobulin gene (see next paragraph).

CLL has been shown to be a neoplasm affecting either pre-germinal center B cells or post germinal center B cells. Shortly before birth, our B cells are programmed to recognize and respond to foreign antigens (proteins, sugars, or lipids not found in our body). However, when a B cell enters a lymphoid organ (e.g. lymph node) and engages a foreign antigen in a germinal center, DNA changes (somatic mutations) occur in the antibody-producing gene of the pre-germinal center B cell. The resulting B cells, that produce an antibody with the greatest affinity for a foreign antigen, are selectively expanded in number. This normal process results in an immune system capable of selectively and efficiently eliminating any foreign antigen. We can distinguish these pre-germinal B cells from post-germinal B cells by sequencing the DNA in the immunoglobulin gene (the gene that makes the antibody). CLL derived from pre-germinal center (unmutated) B cells have a worse prognosis compared with the post-germinal center (mutated) B cells.

The Rai stage, cytogenetic abnormalities in the CLL cells, presence or absence of *TP53* mutations in the CLL cells, and the mutational status of the immunoglobulin gene in the CLL cells are now included in prognostic models.

D. Treatment of CLL

Many patients with CLL often do NOT require treatment at the time of diagnosis. Early treatment of asymptomatic CLL patients has not been shown to improve survival in the past. Patients with Rai stage 0 disease (lymphocytosis alone) are typically not treated, unless there is a rapid rise in the lymphocyte count or constitutional symptoms. Patients with Rai stage I and II disease (adenopathy, splenomegaly) may be treated, but typically only if the lymphoid organ enlargement is painful, cosmetically unacceptable to the patient (e.g. enlarged lymph nodes in the neck), obstructing the intestines or the airway, or obstructing ducts draining urine from the kidneys or bile from the liver. Treatment is recommended for patients with anemia and thrombocytopenia (Rai stage III and IV) due to bone marrow failure. A significant minority of CLL patients may never require therapy before dying of unrelated causes. These patients typically will have either Rai stage 0 or stage I disease at diagnosis. The prognostic factors discussed in the previous section are associated with the risk of progression to higher stages of the disease over time. The SWOG Leukemia Committee S1905 study is actively recruiting asymptomatic CLL patients with high-

risk features and evaluating earlier intervention with modern, time-limited therapy (venetoclax and obinatuzumab, see below).

Historically, CLL was treated with single agent, oral chemotherapy drugs (DNA alkylating agents such as chlorambucil and cyclophosphamide) and corticosteroids (prednisone). Most responses were only partial responses and were not durable. Other chemotherapy agents became available over time including the intravenously administered purine analogs (fludarabine, cladribine, and pentostatin) and bendamustine. These agents were more effective but caused more suppression of normal blood counts as well as the immune system, leading to more frequent infectious complications. Monoclonal antibodies directed against CD20 on the surface of CLL cells were developed as well (e.g., rituximab, obinutuzumab). These agents cause antibody-dependent cell-mediated toxicity, by engaging the immune system to attack the neoplastic CLL cells. Rituximab and obinutuzumab frequently can cause infusion reactions, especially with the first administration. Patients may experience fever, shaking chills, low blood pressure, shortness of breath, low blood oxygen levels, wheezing, hives, and back pain. Infusion reactions are common and can be successfully managed in most cases by slowing the infusion and administering corticosteroids and antihistamines. Most patients can continue to receive these monoclonal antibodies even in the event of a serious reaction with the first dose.

Combination regimens became the standard of care for initial therapy of CLL for at least two decades (1995 through 2015). The most frequently used immunochemotherapeutic regimens were fludarabine and rituximab (FR); pentostatin, cyclophosphamide, and rituximab (PCR); fludarabine, cyclophosphamide, and rituximab (FCR); bendamustine and rituximab (BR); and oral chlorambucil with obinutuzumab. The selection of the specific regimen depended on the fitness of the patient to receive aggressive immunochemotherapy regimens. The latter was evaluated in older CLL patients with comorbid illnesses (who would not tolerate more aggressive chemotherapies such as fludarabine regimens). The combination of chlorambucil with obinutuzumab resulted in higher rates of complete remission and superior progression free survival compared with either chlorambucil alone or chlorambucil with the first generation anti-CD20 monoclonal antibody, rituximab [see Goede V, et al. N Engl J Med. 2014; 370(12): 1101-1110].

Most recently, cytotoxic chemotherapy drugs have been replaced by oral, targeted therapies. The two most popular regimens for initial therapy are the oral inhibitors of the Bruton tyrosine kinase (BTK) inhibitors and the BCL2 inhibitor venetoclax with the anti-CD20 monoclonal antibody, obinutuzumab. Ibrutinib, the first BTK inhibitor to be approved by the FDA, demonstrated superiority to single agent chlorambucil as initial therapy for CLL. The overall response rate has higher (86% versus 35%), progression free survival was better (84% lower risk of progression or death), and the overall survival was longer (98% versus 85% at 2 years) with ibrutinib versus chlorambucil, respectively [see Burger JA, et al. N Engl J Med 2015; 373: 2425]. But BTK inhibitors are associated with significant risks including atrial fibrillation (heart rhythm abnormality), excessive bleeding, and high blood pressure. CLL patients usually take the oral BTK inhibitors indefinitely in the absence of unacceptable toxicity, progression, or loss of response. There are now several FDA-approved, second generation BTK inhibitors for CLL including acalabrutinib, zanubrutinib, and pirtobrutinib. The second generation BTK inhibitors

may have less risk of the common toxicities observed with ibrutinib, such as atrial dysrhythmia and bleeding tendency. The second generation BTK inhibitors may still be effective in patients developing resistance to ibrutinib.

More recently, the ALPINE trial compared the BTK inhibitors, ibrutinib and zanubrutinib, in patients with relapsed CLL (see Hillman P, et al. J Clin Oncol 2022; 4: 1035-1045 and Brown J, et al. Blood 2024; 144: 2706-2717). Most of the patients (76% and 80% in the two arms) had previously received immunochemotherapy regimens such as FR, FCR, and BR. The median survival was similar in both arms of the study; however, progression free survival was longer with zanubrutinib. At 3 years, 80% of patients in both arms were still alive. This study only has a median follow-up of 42.5 months (range 0.1 to 60 months). Fewer patients developed atrial fibrillation while taking zanubrutinib compared with ibrutinib.

The combination of venetoclax and obinutuzumab is a time-limited regimen for initial therapy, limited to only one year duration and then discontinued. This regimen leads to an overall response of 85% and complete remission rate in 50% of CLL patients with other comorbid illnesses. The progression free survival and response rates were superior with venetoclax and obinutuzumab compared with the chemotherapy agent chlorambucil with obinutuzumab [Fischer K, et al. N Engl J Med 2019; 380: 2225]. The risks associated with venetoclax therapy include tumor lysis syndrome (kidney failure and cardiac arrhythmias due to rapid destruction of the malignant cells) and low WBC count. Patients begin obinutuzumab prior to venetoclax, and the dose of venetoclax is escalated slowly over a month, to minimize the risk of tumor lysis syndrome. Venetoclax can also be used for patients with relapsed/refractory CLL.

The BTK inhibitors and the BCL2 inhibitor venetoclax have improved the overall survival and progression free survival of CLL patients requiring initial therapy compared with immunochemotherapy regimens [see Shanafelt TD, et al. Blood 2022; 140(2): 11 and Woyach JA et al. N Engl J Med 2018; 379: 2517]. Allogeneic hematopoietic stem cell transplantation is very rarely considered now for CLL patients. Nevertheless, these regimens are not curative. Other forms of cellular immunotherapy are in development including chimeric antigen receptor T cells (CAR-T) and bispecific T cell engagers (BiTE).

IV. Summary of Exposure

According to the operative Third Amended Short Form Complaint (D.E. 21, Aug. 16, 2024), Mr. Hill claims to have been exposed to the water at Camp Lejeune between May 1983 and June 1985.

V. Mr. Hill's Relevant Medical History

Mr. Hill was initially evaluated by an oncologist (Dr. Lynch) on 08/31/2004 for suspected CLL (initial consultation note, 00028_HILL_VBA_0000007509). His primary care provider requested the consultation for evaluation of a progressively increasing WBC count. In 03/19/2001, the WBC count was 12,100/microliter with 68% lymphocytes. The WBC count was 19,000 with 93% lymphocytes on 08/18/2023 and WBC 29,800 with 85% lymphocyte on 06/29/2004. On the day

of the consultation, the WBC count was 39,600 with 95% lymphocytes, hemoglobin 15.1 gram/dL, and platelet count 329,000/microliter. The physical examination noted cervical and supraclavicular adenopathy. There was no hepatosplenomegaly. A CT scan of the chest, abdomen, and pelvis on 08/16/2004 demonstrated thoracic, axillary, abdominal, and pelvic lymphadenopathy. The spleen was normal. There was also prostatic enlargement and colonic diverticulosis. I cannot find the diagnostic flow cytometry report at this time in the provided records. However, this initial consultation note indicates that the PCP had sent the peripheral blood to the University of Florida Shands for flow cytometry. According to the PCP note, the flow cytometry was interpreted as "B cell chronic lymphocytic leukemia involving 60% of leukocytes and no overt evidence of large cell transformation". At the time of diagnosis, he had Rai stage I disease with lymphocytosis and adenopathy only.

I reviewed the flow cytometric findings from the bone marrow biopsy on 02/24/2010 (00028_HILL_VBA_0000002871-77), and I agree with the diagnosis of CLL based on the characteristic immunophenotype of the cells. Mr. Hill had already received FR and PCR immunochemotherapy at the time of this bone marrow biopsy. The bone marrow is hypercellular (90% cellularity) with near-total replacement by chronic lymphocytic leukemia infiltration. There were only 20-25% prolymphocytes (i.e. no evidence of prolymphocytic transformation). There is storage iron. The flow cytometry detects a monoclonal B cell population with moderate surface lambda light chain, CD5, and CD23 expression; there was dim expression of CD20. CD38 was expressed, but ZAP-70 was absent. The cytogenetic analysis demonstrated a normal diploid male karyotype in 20 metaphases; there were several non-clonal abnormalities (not significant). I cannot tell if the karyotype was performed with a B cell mitogen. I cannot find any pathology reports of a CLL FISH panel, TP53 mutation status, or IgHV mutation status. However, in progress notes from Dr. Schmidt (e.g. on 01/17/2024, 00028_HILL_VHA_0000000067-69 and on 07/10/2024, 00028_HILL_VHA_0000000405), she notes "FISH negative; TP53 wildtype; IgHV unmutated".

Mr. Hill had evidence of CLL prior to his referral to oncology in 2004. According to the compensation and pension note by Dr. Sheryl Anthos on 08/12/2016 (00028_HILL_VHA_0000000126), his WBC count was normal on 09/30/1998 but with relative lymphocytosis (55% lymphocytes). Mr. Hill had been evaluated for a lump in the left cervical region and the proximal right thigh at the Gainesville VA Urgent Care on 03/19/2001 and again by his PCP at the time of routine health maintenance examination on 09/28/2001 (00028_HILL_VHA_0000000132-133). Mr. Hill wanted the mass removed. He was evaluated by a general surgeon on 11/07/2001. Mr. Hill provided a history of the lumps increasing and decreasing in size over time. He had a prior lipoma removed from his forehead in 1991 according to the initial oncology consultation note. The surgeon felt these two masses were also lipomas (benign fatty tumors) and recommended again resection. Dr. Anthos hypothesized (and I concur) that these masses were likely enlarged lymph nodes in the cervical and inguinal regions. Lymph node involvement by CLL can wax and wane over time without intervention. In retrospect, the finding of lymphocytosis and these palpable masses could have led to an earlier diagnosis of CLL, while Mr. Hill was still on active duty. However, earlier diagnosis would not have changed the prognosis or the initial recommendation from oncology for an observational protocol.

He had progressive disease and started initial immunochemotherapy on 05/31/2005. He was admitted from 05/31 through 06/05/2025 for cycle #1 of fludarabine and rituximab (Discharge summary, 00028_HILL_VBA_0000008025). He received four cycles of FR but with treatment delays due to therapy-related myelosuppression (low blood counts). He responded to therapy with a resolution of the peripheral blood lymphocytosis. He remained clinically stable for 2.5 years, but then the disease began to progress again.

He ultimately required treatment again by late 2008. He was evaluated in Urgent Care on 12/11/2009 for evaluation of increasing adenopathy and leg edema (00028_HILL_VBA_0000005894). He was admitted on 01/26/2009 for cycle #1 of the second immunochemotherapy regimen with pentostatin, cyclophosphamide, and rituximab (See progress note by Dr. Bradley Fletcher, 00028_HILL_VBA_0000005961). At the time of admission, he had lower extremity and scrotal edema due to lymphedema (lymphatic obstruction) from the increasing inguinal, pelvic, abdominal, and thoracic adenopathy. On 01/26/2009, the WBC count was 76,710, hemoglobin 12.3 gram/dL, and platelet count 152,000. By the start of cycle #3 PCR on 03/24/2009 (00028_HILL_VBA_0000005935), the edema had resolved, the adenopathy was smaller, and the WBC count had decreased into the normal range. He started the fourth and last cycle of PCR on 04/21/2009 (00028_HILL_VBA_0000005927). He then received 36 Gy involved field radiation therapy to the axillae. An echocardiogram on 02/18/2009 was performed to exclude cardiac dysfunction as a cause of the edema; the cardiac function was normal (00028_HILL_VBA_0000005952).

The response following PCR was brief, with recurrent leg and scrotal edema as well as increased adenopathy by January 2010. The spleen was enlarged by CT scan (17 cm craniocaudal dimension). A bone marrow biopsy was performed on 02/24/2010 demonstrating extensive involvement of the bone marrow by CLL. He was admitted from 02/22 through 02/26/2010 to receive the first cycle of his third immunochemotherapy regimen with bendamustine and rituximab (Discharge note, 00028_HILL_VBA_0000002154). He had a response but tolerated this less well and only received one additional cycle in April 2010.

He again had rapidly recurrent disease in late 2010. He was referred to University of Florida Shands for consideration of an allogeneic hematopoietic stem cell transplantation (allo HSCT). He was evaluated by Dr. John Wingard on 01/05/2011 (00028_HILL_MEDRECS_0000000052-55). Dr. Wingard recommended further cytoreductive therapy prior to allo HSCT. He was admitted on 01/13/2011 for PCR immunochemotherapy (00028_HILL_VBA_0000002726-2734). However, after admission and consultation with colleagues, the decision was made to begin the fourth immunochemotherapy regimen with bendamustine, mitoxantrone, and rituximab (BMR). He received a total of 4 cycles with response. However, the patient decided at that time not to pursue allo HSCT for social reasons (his wife was being treated for breast cancer, and she ultimately died of the disease).

Following BMR he had unexplained absolute neutropenia. A bone marrow biopsy and aspirate were repeated on 10/24/2011; he has had only two bone marrow biopsies for evaluation (00028_HILL_VBA_0000008733). The bone marrow was 30-40% cellular (normal cellularity) with 5-10% involvement by CLL. There was a suggestion of intramedullary destruction of

neutrophils with increased tissue macrophage with intracellular neutrophilic phagocytosis. Interestingly, on this day, the absolute neutrophil count (ANC) was again normal (2,500/microliter). The WBC was 4,150, hematocrit 41.8%, and platelet count 250,000. The prolonged neutropenia was attributed to an idiosyncratic reaction to the rituximab.

He again had progressive adenopathy with lower extremity and penile/scrotal edema. He also developed progressive thrombocytopenia as well. He was admitted from 06/18 through 06/22/2012 (00028_HILL_VBA_0000002147) for the fifth immunochemotherapy regimen with fludarabine, cyclophosphamide, and rituximab (FCR). He could only tolerate two cycles of FCR due to prolonged neutropenia following the second cycle (the decision to discontinue FCR can be found at 00028_HILL_VBA_0000003268). Bactrim prophylaxis was discontinued at this time due to concern of its potential role in persistent neutropenia. It took four months for the blood counts to recover according to the progress note by Dr. Saruna Sliesoraitis (00028_HILL_VBA_0000006552).

PET/CT scan on 03/20/201 demonstrated "massive" hypermetabolic lymphadenopathy throughout the chest, abdomen and pelvis as well as splenomegaly. He was then treated with lenalidomide (Revlimid) and rituximab (Rituxan). Lenalidomide is an oral immunomodulatory agent approved by the FDA for multiple myeloma and myelodysplastic syndrome. Lenalidomide has limited activity in forms of non-Hodgkin lymphoma. Mr. Hill started cycle #1 on 05/22/2013. He was admitted from 06/06 through 06/13/2013 for evaluation and treatment of increase in back pain. The discharge summary lists muscle strain as the cause of the back pain (00028_HILL_VBA_0000002139). However, the CT scan performed during this admission showed increased adenopathy and splenomegaly compared with the prior CT scan on 03/20/2013. Dr. Sliesoraitis hypothesized in her progress note on 06/07/2013 (00028_HILL_VBA_0000001761) that the pain and CT findings were due to a flare of the disease, which had been described in the literature—I agree. He was treated with a steroid pulse (dexamethasone) with improvement in the pain. The lenalidomide was held until 06/19/2013. He was again admitted from 06/24 through 06/26/2013 for evaluation of fever one day following rituximab infusion in the setting of grade 3 neutropenia (00028_HILL_VBA_0000002887). He received cycles on 06/20, 07/15, 08/15, 09/12, 10/21 (delayed due to neutropenia and restarted at a lower dose). The lenalidomide and rituximab regimen was discontinued on 01/14/2014 due to pruritus without rash.

On 04/22/2014 he was evaluated in the oncology clinic for early satiety and increased abdominal fullness. A CT scan on 05/08/2014 demonstrated bulky conglomerate adenopathy in the upper abdomen, retroperitoneum, mesentery, and pelvis. The largest mass at the aorto-iliac bifurcation measured 15.4 cm x 18.1 cm. The spleen had increased to 19.4 cm and the liver to 19.5 cm. He started ibrutinib oral daily therapy on 05/13/2014. He was evaluated two months after starting ibrutinib on 07/08/2014 by Dr. Sliesoraitis and Dr. Jennifer Roeser Duff (00028_HILL_VBA_0000008292). He was tolerating ibrutinib. The spleen and inguinal lymph nodes were smaller. The WBC count was 4,490, absolute lymphocyte count (ALC) 2,180, hemoglobin 13.8 gram/dL, and platelet count 190,000. The liver function tests were all within normal limits.

Mr. Hill saw Dr. Maxim Norkin, V.A. Oncology Clinic, for the last time on 03/12/2019 (00028_HILL_VBA_0000001918). Dr. Norkin felt that Mr. Hill was tolerating ibrutinib and was responding well. The constitutional symptoms (B symptoms) had resolved, and there was no hepatosplenomegaly or adenopathy by physical examination. On 03/05/20219, his WBC count was 3,730, hemoglobin 13.4 gram/dL, and platelet count 177,000. There was no mention of hepatic toxicity in her progress note.

He was evaluated by Mr. Stephen McCready, P.A., in the V.A. Oncology Clinic on 07/17/2019, four years after starting ibrutinib (00028_HILL_VBA_0000006632). His liver enzymes were normal on 03/12/2015 (00028_HILL_VBA_0000006473) and again on 05/15/2015 (00028_HILL_VBA_0000006456). However, subsequent laboratory evaluation was notable for abnormal liver enzymes. The PCP suggested that the liver injury may be due to ibrutinib. On this day there was no adenopathy or splenomegaly noted by physical examination. On 07/15/2019 the blood counts were normal; WBC count was 4,590, ALC 1,520, ANC 1,980, hemoglobin 14.1 gram/dL, and platelet count 210,000. However, the liver enzymes were elevated; ALT was 83, AST 51, and alkaline phosphatase 173 (all grade 1 elevations not requiring dose adjustments). Mr. McCready recommended a dose reduction, but Mr. Hill preferred to discontinue the ibrutinib.

He was referred to General Surgery for a possible gall bladder polyp found by ultrasound in August 2019 (00028_HILL_VBA_0000006608). However, a repeat abdominal ultrasound detected a mobile, echogenic focus in the gall bladder suggestive of cholelithiasis (gall stones); there was no gall bladder polyp.

The ibrutinib was not restarted. However, he was admitted from 12/28/2020 through 01/02/2021 for evaluation of abdominal pain, fever, chills, and abnormal liver function tests including increased bilirubin now (Emergency Department note, 00028_HILL_VBA_0000006233). Laboratory evaluation on admission was remarkable for AST 365 (normal <45), ALT 201 (normal < 40), alkaline phosphatase 141 (normal < 125), and total bilirubin 1.9 (normal < 1.3). His WBC count was 6,320, ANC 5,110, ALC 580 (low), hemoglobin 14.9 and platelet count 178,000. An abdominal ultrasound demonstrated mild gall bladder wall thickening with cholelithiasis, but without biliary ductal dilation. The spleen was “unremarkable”. CT scan of the abdomen and pelvis demonstrated periportal and portal hepatic edema, gall bladder wall edema, cholelithiasis, umbilical hernia, and prostatomegaly. There was no mention of adenopathy or splenomegaly in the impression. The bilirubin continued to increase after admission. He underwent an endoscopic retrograde cholangiopancreatography (ERCP) on 12/30/2020 (00028_HILL_VBA_0000009224). There were multiple duodenal ulcers. A sphincterotomy was performed. There were no gall stones in the common bile duct. A surgical consultant felt the abdominal pain was related to the peptic ulcer disease (HILL_VBA_6049). According to the discharge summary (00028_HILL_VBA_0000002881-87) Mr. Hill had cholecystitis and ulcer disease. He was discharged on ciprofloxacin, metronidazole, and omeprazole.

Following discharge, he again presented to the Emergency Department on 01/06/2020 for evaluation of peri-umbilical pain, nausea, and chills. The WBC count was 8,040, ANC 6,390 (high normal range), ALC 810 (low), hemoglobin 13.9, and platelet count 200,000. The liver function panel was again abnormal (AST 225, ALT 125, alkaline phosphatase 135, and bilirubin 1.4). A

CT scan of the abdomen and pelvis did not demonstrate any acute abnormality. In fact, there were NO gall stones visualized on the scan. The spleen had decreased from 19 cm to 13 cm. The mesenteric and retroperitoneal lymph nodes were now all less than 1 cm.

Mr. Hill was evaluated by Mr. Stephen McCready and Dr. Jessica Schmit on 02/13/2020 in the V.A. Oncology Clinic (00028_HILL_VBA_0000006582). He had no adenopathy or splenomegaly by physical examination. On 02/10/2020 his WBC count was 3,730, ANC 1,680, ALC1,790, hemoglobin 14.5, and platelet count 205,000. The liver function panel was completely normal. The note confirms that Mr. Hill self-discontinued ibrutinib in July 2019 due to possible hepatic injury. He was seen in the Emergency Room again on 02/22/2021 for abdominal pain. However, at this time the liver function panel was normal, and the CT scan only demonstrated gall bladder wall thickening without cholelithiasis.

He was started on the oral BTK inhibitor zanubrutinib in July 2023 as documented in the progress note of Dr. Jessica Schmit on 01/17/2024 (00028_HILL_VHA_0000000067-69). His CLL treatment was restarted at that time due to "significant progression of the adenopathy and splenomegaly" by CT scans in March 2023 and 06/22/2023. On 01/17/2024 he only endorsed "mild fatigue", but he was otherwise "doing very well". His WBC count was 4,480, ANC 1690, ALC 1,830, hemoglobin 13.8 gram/dL, and platelet count 145,000. Repeat CT scan on the same day demonstrated "mild residual splenomegaly and significant reduction in the size of the [lymph nodes] in the mesentery" by Dr. Schmit's personal review of the images, which she interpreted as a "great response". She recommended continuing Zanubrutinib (the radiologist's interpretation of this CT scan of the chest, abdomen, and pelvis can be found at 00028_HILL_VHA_0000000120-123). There had been "marked decrease size in the thoracic, abdominal, and pelvic lymphadenopathy". The largest lymph node measured 2 cm (decreased from 4.8 cm). There was no mention of hepatic steatosis.

The dose of zanubrutinib was decreased by 75% (from 160 mg twice daily to 80 mg once daily) on 04/10/2024. CT scan on 06/04/2024 demonstrated continued improvement in the lymphadenopathy in the abdomen and pelvis (impression of this CT scan can be found at 00028_HILL_VHA_0000000407).

He was admitted from 06/30 through 07/06/2024 for evaluation and treatment of Salmonella colitis and bacteremia (00028_HILL_VHA_0000000346). Zanubrutinib was held on admission. He was treated with IV antibiotics. On 07/04/2024, his WBC count was 4,100, ANC 2,150, ALC 810, hemoglobin 11.0 gram/dL and platelet count 155,000 (00028_HILL_VHA_0000000392). CT scan demonstrated thickening of the descending colon, sigmoid colon, and rectum consistent with reactive inflammation. There was also trace pelvic ascites and mesenteric edema. The lymph nodes were increased in size compared with a prior CT scan on 06/04/2024. The spleen measured 14.1 cm, and the liver was enlarged. It isn't clear if the increased size of the spleen and lymph nodes was due to the colitis or the CLL.

Following discharge, he saw Dr. Schmit on 07/10/2024. He was still very weak following discharge. According to her progress note (00028_HILL_VHA_0000000405), "[Mr. Hill] has been wanting to come off of chemotherapy for a long time and at this time he requests a treatment

break". Therefore, Mr. Hill did not restart zanubrutinib following discharge from the hospital. He remains off CLL therapy at the time of this review.

Mr. Hill presented on 10/08/2024 for evaluation of shortness of breath and increased abdominal fullness. He was not able to take a deep breath due to the abdominal distention. On 10/09/2024, his WBC count was 3,600, ALC 1,840, hemoglobin 11.2 gram/dL, and platelet count 129,000. CT scans demonstrated increased size of the liver and spleen and "similar abdominal adenopathy". A mass in the interatrial septum was identified by this CT scan. Evaluation of this intracardiac mass has occurred, but he has not been evaluated by Dr. Schmit since the admission in October 2024. Most recently on 01/08/2025, his WBC count was 4100, ALC 1720, hemoglobin 12.9 gram/dL, and platelet count 116,000 (00028_HILL_VHA_0000001076). It appears that the progression of his disease in the abdomen is no longer reflected by a simultaneous increase in the peripheral blood WBC count or ALC.

He has hypogammaglobulinemia due to both CLL as well as the immunochemotherapy regimens and the BTK inhibitors. Hypogammaglobulinemia increases the risk of bacterial infections, especially sinusitis, pharyngitis, bronchitis, and pneumonia. He has been receiving gamma globulin (antibodies from blood donors) infusions every 4-8 weeks for close over a decade. Despite this prophylactic therapy, he has had multiple visits to the Emergency Department and Urgent Care for evaluation of symptoms of upper respiratory tract infections. He has been prescribed oral antibiotics for sinusitis and pharyngitis. Most recently, he was evaluated in the Emergency Department on 09/08/2023 for rhinorrhea and sinus congestion (00028_HILL_VHA_0000001028) and in an Urgent Care on 09/20/2024 for cough and nasal congestion. On 09/20/2024, his WBC count was 4,100, ANC 1,300, ALC 1,600 (normal), hemoglobin 12.5 gram/dL, and platelet count 169,000 (00028_HILL_VHA_0000001000). Both times he was treated symptomatically.

VI. Mr. Hill's Other Medical Issues

Abnormal colonoscopy

- Colonoscopy 04/11/2012 (00028_HILL_VBA_0000002863-64): Biopsy of ascending colon polyp positive for submucosal lymphoid aggregate consistent with chronic lymphocytic leukemia.
- Colonoscopy 10/02/2017 (00028_HILL_VBA_0000009227): Normal, no biopsies.
- Colonoscopy 11/07/2022 (00028_HILL_VBA_0000009219): 6 mm polyp in the transverse colon and diverticulosis. Biopsy of polyp diagnostic of tubular adenoma (00028_HILL_VBA_0000008724)

Arthropod assault reactions (bug bites)

- Dermatology consultation 11/12/2014 (00028_HILL_VBA_0000006537): Open lesions on the arms and legs. Pathology consistent with arthropod assault reaction (bug bites). Treated with topical steroids. These developed after starting ibrutinib, but exaggerated reactions to bug bites are often seen in CLL patients unrelated to therapy.

Atrial myxoma

- CT chest, abdomen, pelvis for evaluation of abdominal fullness, difficulty with deep inspiration 10/08/2024 (00028_HILL_VHA_0000001072-74): Increase in hepatosplenomegaly, similar abdominal adenopathy, increase in size of left atrial mass. No pulmonary embolism.
- Initial cardiothoracic surgery consultation 10/09/2024 (00028_HILL_VHA_0000001312): Increased size of intracardiac mass noted on CT chest, abdomen, and pelvis for evaluation of increasing dyspnea and difficulty taking deep breath. Consultant recommends cardiac MRI and admission for evaluation of dyspnea and abdominal distention.
- Transthoracic echocardiogram 10/09/2024 (00028_HILL_VBA_0000000864-865): No evidence of an interatrial septal mass
- Cardiac MRI 10/09/2024 (00028_HILL_VHA_0000001066-69): 1.6 x 1.4 cm mass of the interatrial septum with thrombus at periphery. Splenomegaly and myocardial edema/inflammation also noted.
- Discharge summary 10/09 – 10/10/2024 (00028_HILL_VHA_0000001109-1112)
- Cardiac catheterization 12/02/2024 (00028_HILL_VHA_0000001151-53): 90% stenosis of the posterior descending artery.
- Cardiothoracic telephone note 12/04/2024 (00028_HILL_VHA_0000001149): Mr. Hill was offered mass resection and one vessel (PDA) bypass. Mr. Hill declined, elected continued observation, and continued apixaban (Eliquis) for prevention of thromboembolic complications of the atrial myxoma.

Bell's palsy (Cranial nerve VII dysfunction)

- Neurology consultation 06/16/2007 (00028_HILL_VBA_0000008950): Diagnosis of right Bell's palsy, treated initially with acyclovir and prednisone.

Benign paroxysmal positional vertigo (BPPV)

- Emergency department visit 10/03/2020 (00028_HILL_VBA_0000006256): Evaluation of nausea, emesis, dizziness. Head CT negative.

Benign prostatic hypertrophy with lower urinary tract symptoms

- CT scan on 08/16/2004 demonstrated enlarged prostate.
- Urine cytology for hematuria 12/13/2012 (00028_HILL_VBA_0000008746): Negative for malignancy
- Ultrasound 11/27/2024 (00028_HILL_VHA_0000001064): Prostatomegaly, prostate volume 135 cc, kidneys normal, spleen 16.9 cm (enlarged)
- Urology Clinic progress note 11/14/2024 (00028_HILL_VHA_0000001188): Current medical therapy = alfuzosin and finasteride

Cataracts, ocular hypertension, dry eyes. Current therapy = timolol and brimonidine ophthalmic

Circumcision

Chronic kidney disease, stage 2. Related to hypertension, hyperlipidemia, and pre-diabetes

Chronic sinusitis / allergic sinusitis

- Nasopharyngolaryngoscopy note 06/19/2017 (00028_HILL_VBA_0000003448): no pathologic findings

de Quervain's radial styloid tenosynovitis

- Rheumatology note 02/23/2015 (00028_HILL_VBA_0000006490-92): Steroid injections on 01/12/2015 and 02/23/2015
- Operative note 01/21/2016 (00028_HILL_VBA_0000007552): Release of first dorsal compartment of the left wrist.

Genital herpes infection

- Operative note 06/24/2014 (00028_HILL_VBA_0000008301): Excision of penile ulceration
- Pathology report (00028_HILL_VBA_0000008290): Herpetic ulceration with inflammation

Giant cell tumor of tendon

- Operative note 02/06/2014 (00028_HILL_VBA_0000009214): Resection of mass from left fourth finger
- Pathology report (00028_HILL_VBA_0000008731): Giant cell tumor of the tendon

Hemorrhoids

- Urgent Care visit 05/06/2009 (00028_HILL_VBA_0000005923): Evaluation of rectal pain

Hyperlipidemia. Current medical therapy = rosuvastatin

Hypertension. Current medical therapy = carvedilol, losartan

Hypertensive retinopathy.

Hypothyroidism. Current medical therapy = levothyroxine

Low back pain / degenerative arthritis of the spine

- Discharge summary 06/06 – 06/13/2013 (00028_HILL_VBA_0000002139): MRI of the spine demonstrated multilevel degenerative disease of the spine with both canal and neuroforaminal stenoses.

Pyogenic granuloma

- Pathology report of right great toe biopsy 03/02/2015 (00028_HILL_VBA_0000002858): Pyogenic granuloma.

Rectal abscess drainage

VI. Analysis

A. It is highly unlikely that Mr. Hill's distant and intermittent exposure to water at Camp Lejeune caused his CLL.

Although the formal diagnosis of CLL was not made in Mr. Hill until 2004, there was evidence of the disease earlier in his life. In 1998, 13 years after leaving Camp Lejeune, there was a relative lymphocytosis. In 2001, he developed cervical and inguinal masses which in retrospect were likely due to CLL. Given the natural history of CLL with evolution from a monoclonal B cell lymphocytosis in all patients, the disease process could have already been present during his time at Camp Lejeune. He was 30 years old at the time he first started living at Camp Lejeune. Nevertheless, the environmental and/or genetic events that initiate the monoclonal B cell expansion and then progression to CLL are unknown. The limited use of the available V segment genes in patients with CLL suggests the hypothesis that the disease initiates due to an abnormal expansion of B lymphocytes in response to an auto-antigen or a very common environmental antigen in susceptible people.

Mr. Hill was nearly 20 years younger than the median age of patients at the time of diagnosis with CLL. Nevertheless, 7.4% of CLL patients in the SEER registry are between ages 45 and 54 years at the time of diagnosis. In the ECOG1912 clinical trial of previously untreated CLL patients less than age 70 years, 60% of the patients were less than age 60 years. The median age was 56 years (Shanafelt TD et al. N Engl J Med. 2019; 381(5): 432). CLL is less common in people of African descent. However, in the Unites States SEER registry data the annual incidence in non-Hispanic Whites is less than twice as high as non-Hispanic Blacks. The African-American population represents a genetically diverse population. His body mass index has been documented in the record as greater than 30 kg/m2 at the time of his diagnosis and thereafter.

Bove and colleagues (see Bove FJ, et al. Environmental Health 2014; 13: 10) evaluated the mortality of Marines and Navy personnel who began service between 1975 and 1985 and were exposed to contaminated drinking water at Camp Lejeune in a retrospective cohort study. The mortality data were collected from 1979 to 2008 from death registries and the cause of death from the National Death Index. The control cohort were Marines and Navy personnel stationed at Camp Pendleton in California during the same time. This cohort study does not distinguish between death due to the multiple subtypes of acute and chronic leukemia. A shared generic name (such as leukemia) does not indicate a shared pathophysiologic mechanism. You cannot tell any specific risk for any single leukemia subtype based on a risk estimated for leukemia as a group.

In such a retrospective cohort study, it is important to conduct a multivariate analysis that includes all other potential covariates that may impact the results. Even setting aside the issue of estimating risk for all leukemia subtypes combined, a study should still adjust for potential confounders. The models would need to include age, gender, ethnicity, obesity, and family history of leukemia. These are known demographic features that affect the incidence of CLL. The Bove

analysis includes the first three, but not obesity or family history. Furthermore, since CLL has a long natural history with or without therapy, the incidence of CLL, rather than mortality related to CLL, would provide a better assessment of any potential risk for this disease.

The standardized mortality rate for all leukemia was significantly lower in both the Camp Lejeune and Camp Pendleton populations compared with the general population [0.74 (95% CI 0.57, 0.95) and 0.78 (95% CI 0.60, 0.99), respectively]. There was not a statistically significant difference in leukemia-related mortality in the Camp Lejeune and Camp Pendleton population. Furthermore, the relation between cumulative exposure and leukemia-related mortality was not linear; the calculated hazard ratios were lower at the higher exposures to TCE, benzene and TVOC than the lower exposures.

	Low exposure	Medium exposure	High exposure	Cumulative exposure	Log_{10} cumulative exposure
			c. Leukemias (N=66)		
TCE	2.00 (1.00, 4.00) N=16	1.54 (0.71, 3.36) N=11	1.81 (0.85, 3.85) N=13	.00002 (−0.00004, 0.00008) p=.46	.0801 (−0.0093, 0.1695) p=.08
Benzene	2.54 (1.27, 5.08) N=17	1.46 (0.66, 3.20) N=11	1.69 (0.77, 3.67) N=12	.00168 (−0.00158, 0.00494) p=.31	.1276 (0.0020, 0.2532) p=.05
TVOC	2.50 (1.24, 5.03) N=19	1.33 (0.56, 3.14) N=9	2.33 (1.08, 5.03) N=15	.00001 (−0.00003, 0.00005) p=.44	.0950 (0.0032, 0.1868) p=.04

Bove and colleagues (see Bove FJ, et al. Environmental Health Perspective 2024; 132: 107008-1-15) also evaluated the incidence of cancers with longer follow up in the Camp Lejeune and Camp Pendleton cohorts (https://doi.org/10.1289/EHP14966). In this evaluation, they included diagnoses obtained from cancer registries, a more reliable source of data than death registries, between 1996 and 2017. The Marine and Navy personnel were on base between 1975 and 1985. There was NO association between exposure to the contaminated water supply at Camp Lejeune between 1975 and 1985 and the subsequent development of all leukemia subtypes COMBINED between 1996 and 2017 (first line of table below). This analysis distinguishes between the various subtypes of leukemia. There was NO association between contaminated water supply at Camp Lejeune with the subsequent development of CLL [adjusted HR 1.02 (0.79, 1.32)].

	Camp Lejeune			Camp Pendleton
Cancer outcome	Cases (*n*)	Unadjusted HR (95% CI)	Adjusted HR (95% CI) CIR	Cases (*n*)
Leukemias	314	1.06 (0.91, 1.24)	1.07 (0.91, 1.25) 1.4	319
Lymphoid cancers	979	1.03 (0.95, 1.13)	1.02 (0.94, 1.12) 1.2	1,018
Hodgkin lymphoma	108	1.01 (0.78, 1.31)	1.01 (0.77, 1.31) 1.7	114
Non-Hodgkin lymphoma	550	1.00 (0.89, 1.13)	1.01 (0.90, 1.14) 1.3	588
Mantle Cell	27	1.21 (0.70, 2.09)	1.26 (0.73, 2.19) 3.0	24
Follicular	130	1.03 (0.81, 1.31)	1.07 (0.84, 1.36) 1.6	135
Diffuse large B-cell	160	0.88 (0.72, 1.09)	0.89 (0.72, 1.10) 1.5	194
Burkitt	15	1.33 (0.62, 2.84)	1.53 (0.71, 3.30) 4.6	12
Marginal zone B-cell	43	1.41 (0.89, 2.21)	1.45 (0.92, 2.28) 2.5	33
Multiple myeloma	185	1.22 (0.99, 1.51)	1.13 (0.91, 1.40) 1.5	163
Acute lymphocytic leukemia	23	0.97 (0.55, 1.70)	0.94 (0.53, 1.67) 3.2	25
Chronic lymphocytic leukemia	114	1.01 (0.78, 1.30)	1.02 (0.79, 1.32) 1.7	122
Myeloid cancers (including polycythemia vera, myelodysplastic and myeloproliferative syndromes)	239	1.21 (1.00, 1.45)	1.24 (1.03, 1.49) 1.4	213
Myeloid cancers (including myelodysplastic and myeloproliferative syndromes)	186	1.19 (0.96, 1.46)	1.19 (0.97, 1.47) 1.5	169
Acute myeloid leukemia[a]	104	1.36 (1.02, 1.81)	1.38 (1.03, 1.85) 1.8	82
Chronic myeloid leukemia	39	0.75 (0.50, 1.12)	0.74 (0.49, 1.12) 2.3	56
Myelodysplastic and myeloproliferative syndromes	49	1.66 (1.07, 2.60)	1.68 (1.07, 2.62) 2.4	32
Polycythemia vera	53	1.29 (0.87, 1.93)	1.41 (0.94, 2.11) 2.2	44

I agree with physicians, Dr. Schmit and Dr. Fletcher, that Mr. Hill has had a very prolonged treatment journey. However, the prognosis of a disease is not only related to the disease biology but also to the efficacy and tolerability of the therapeutic options. There are multiple examples of how treatment advances have changed the prognosis of people with cancer. For example, acute promyelocytic leukemia (APL) was first recognized by Leif Hillestad in 1957 and characterized by a "very rapid fatal course of only a few weeks' duration. Chemotherapy improved outcomes, but with arsenic trioxide and all trans-retinoic acid, APL is now recognized as the most curable form of acute myeloid leukemia (see Thomas X. Oncol Ther. 2019; 7 :33-65).

I am concerned about his decision to stop therapy with the BTK inhibitors. The FDA approved prescribing information for ibrutinib recommends ibrutinib dose interruption for grade 3 non-hematologic toxicity. In terms of hepatic enzymes, grade 3 elevation would by five times the upper limits of normal. Ibrutinib can be restarted at the full dose once the hepatic enzyme elevations return to only grade 1 (less than 3 times upper limit of normal). Mr. Hill never had ALT or AST elevations greater than grade 1 while on ibrutinib. In fact, he did develop grade 3 hepatic toxicity when he was off ibrutinib, likely related to cholecystitis and passage of a gall stone (in my opinion). I would have tried to keep him on ibrutinib. Mr. Hill stopped zanubrutinib after his hospitalization for Salmonella colitis and bacteremia. He reported extreme fatigue to Dr. Schmit when she saw him a few days following the hospital discharge. The fatigue at that time was related to the severe infection, and not to cumulative toxicity of zanubrutinib. Given his recent presentation on 10/08/2024 with increased abdominal distention and increased hepatosplenomegaly by CT scan, I would strongly advise him to restart zanubrutinib.

I believe the greatest risks to his mortality now are the refractory nature of his CLL and the risk of infection due to hypogammaglobulinemia and CLL. Eighty percent of subjects treated with ibrutinib or zanubrutinib in the ALPINE trial were still alive at 3 years. He was first treated with a BTK inhibitor over a decade ago. However, the five-year follow up of the ALPINE study was just recently published, so we do not yet have estimates for the survival rates after 10-20 years of BTK therapy in CLL patients previously treated with immunochemotherapy. Nonetheless, he has demonstrated excellent response to both ibrutinib and zanubrutinib without evidence of acquired resistance at this time. He can be treated again with a BTK inhibitor, as Dr. Schmit is planned according to her deposition. He could also receive venetoclax based therapy with a second generation anti CD20 monoclonal antibody obinutuzumab. Bispecific T cell engagers (BiTE) and chimeric antigen receptor T (CAR-T) cells are also available for progressive disease. Given his treatment with fludarabine and cyclophosphamide he is at risk of secondary, treatment-related acute myeloid leukemia and myelodysplastic syndrome.

B. It is highly unlikely that Mr. Hill's kidney disease was caused by exposure to Camp Lejeune water or any treatment related to his CLL

Mr. Hill claims stage 2 chronic kidney disease as related to the alleged exposure to the water supply at Camp Lejeune (00028_HILL_DPPF_0000000006; Third Amend. Short-Form Compl., D.E. 21, pg. 3;). However, he has other known risk factors for chronic kidney disease, including

cardiovascular risk factors of hypertension, hyperlipidemia, obesity, glucose intolerance, and positive family history for arterial occlusive disease (myocardial infarction). In fact, Mr. Hill has other findings of arterial vascular disease, including hypertensive retinopathy and coronary artery disease (90% occlusion of the posterior descending artery of the heart).

C. There is no reference to hepatic steatosis in Mr. Hill's medical records

Mr. Hill also claims hepatic steatosis as related to his alleged exposure to the water supply at Camp Lejeune (00028_HILL_DPPF_0000000006; Third Amend. Short-Form Compl., D.E. 21, pg. 3). However, I cannot find any mention of hepatic steatosis in the records available in this case, including a recently updated problem list. He has had liver enlargement, likely related to infiltration by the CLL. He has had abnormal liver function tests, which I believe are more likely related to intermittent passage of gall stones and to infiltration of the liver by CLL rather than to ibrutinib.

D. Mr. Hill's Work Life Expectancy and Future Treatment

Additionally, Mr. Hill's CLL did not shorten his work life expectancy. Mr. Hill has an ECOG Performance Status Scale of 1, a score indicating the ability to perform activities of daily living and restriction from strenuous activity. Thus, Mr. Hill does not have any functional work limitations related to his CLL. However, Mr. Hill's reason for separating from the Florida Department of Corrections as a pastoral counselor due to a compromised immune system and high risk of illness exposure in 2012 was valid. Mr. Hill's lowered immunity, secondary to his CLL, prohibited him from working outdoors or in highly populated areas. Notwithstanding, Mr. Hill was and is capable of gainful employment except in the following contraindicated environments: outdoors and highly populated areas, especially in small, enclosed spaces. Mr. Hill could work in a low-population office or in an individual office alone.

Regarding a treatment protocol to monitor Mr. Hill's CLL moving forward, I would recommend the following:

- Oncologist Follow-Up: once every three months.
- Infectious disease follow-up: 2 visits per year.
- Bone Marrow Biopsy*: 1 – 2 times total. *Needed if there is a significant change in blood counts that is not explained by the activity of leukemia. These changes include unexplained neutropenia, unexplained anemia, unexplained thrombocytopenia, and the appearance of peripheral blood blast cells.
- Blood Analysis: every 2 months.
- PET Scan*: TBD. *If there is significant change in LDH or the development of constitutional symptoms, such as fever, drenching night sweats, and unexplained weight loss (greater than 10% of body weight).
- CT Scan Abdomen: 2 - 4 times per year.
- Compression Stockings: Replace every 2 - 3 months.
- Cane: Replace every 3 - 5 years.

- Antibiotics: Cipro or Augmentin as needed.
- Zanubrutinib (Brukinsa) 80 mg or Venetoclax (Venclexta) 100 mg: 1 tablet per day.
- Obinutuzumab (Gazyva) IV Therapy*: TBD. * If Venetoclax is prescribed.
- Skilled Nursing Facility Admission: 1 -2 Admissions Total; Each Admission 2 Weeks To 1 Month Total.

VII. Comments on Treating Physician Depositions

I disagree with some of the statements made by the plaintiff and physicians in their depositions. Mr. Bruce Hill stated in his deposition on 04/22/2024 that he could not go to the gym for the prior two months, because his blood counts were too low (Hill Dep., 181:7-19). However, on 01/15/2024 his blood counts were all adequate with a normal hemoglobin, ANC, and ALC. On 04/08/2024, his WBC count was 4,940, ANC 1,990, ALC 2,040, hemoglobin 13.5 gram/dL, hematocrit 41.2%, and platelet count 174,000. He was not anemic. His disease was also under excellent control. He did have mild fatigue per Dr. Schmit's progress note, potentially related to treatment with zanubrutinib or his other medical problems. However, he had been on zanubrutinib since July 2023, so I am not certain why the inability to exercise would be new. Even the most recent blood counts on 01/08/2025 demonstrate only a mild anemia; his hemoglobin is 12.9 gram/dL (00028_HILL_VHA_0000001076).

Dr. Fletcher mentions several times in his deposition that the disease progressed to stage III. (Fletcher dep., 49-53, 143, 158). Rai stage III indicates a hemoglobin less than 11.0 gram/dL. Mr. Hill has never had this degree of anemia, except when he developed Salmonella bacteremia and colitis.

Dr. Fletcher recounts his prior research experience in a Department of Pharmacology, studying chemically induced tumors in mice (Fletcher dep., 129-134). He admits that all chemicals are not toxic, but he states "[the VOC in the Camp Lejeune water supply] would not be in the news, if they did not cause harm". (Fletcher dep., 133:8-21). On the other hand, he specifically recognizes jet fuel as a potential carcinogen since it contains benzene and toluene. (Fletcher dep., 134:1-3). Although Dr. Felsher discounts the exposure to the jet fuel as a risk factor in Mr. Hill's case, we are not certain what protective measures were taken by the service men during the activity of re-fueling jets at that time. On the other hand, I completely agree with one of Dr. Fletcher's statements regarding chemotherapy. He acknowledges that "Almost all chemotherapy causes a **dose dependent** problem." (Fletcher Dep., 156:4-5). However, many of the epidemiologic studies have not clearly established a relation between leukemia and the dose of the VOC and/or the duration of exposure.

Dr. Maxim Norkin states that radiation exposure and cigarette smoking are risk factors for CLL (Norkin Dep., 27:6-13). There is no definite data confirming these associations.

Dr. Jessica Schmit felt that Mr. Hill was compliant with therapy during the time she cared for him. (Schmit Dep., 61:6-12). She does note, like many patients, he does not like to take medications. (Schmit Dep., 50:12-42). She also notes that he demonstrates his autonomy by engaging in "shared decision making" with his medical providers. (Schmit Dep., 61:6-12). Some of these decisions

may negatively impact his outcome. For example, the decision to discontinue ibrutinib and zanubrutinib were both at his request, as opposed to the physician's plan. His oncologists have at times recommended closer follow-up visits when concerned that his disease was progressing, but Mr. Hill has instead requested more time until his next evaluation. In fact, Dr. Schmit noted in her deposition that she would want to follow Mr. Hill closely and with radiographic imaging (CT scan) after discontinuation of zanubrutinib in July 2024. (Schmit Dep., 56:19-24). Despite his presentation on 10/08/2024 (already three months following his last visit with Dr. Schmit) for evaluation of dyspnea due to increasing abdominal distention, and a CT scan demonstrating an increase in the size of the liver and spleen, he has not been re-evaluated by Dr. Schmit.

VIII. Response and Rebuttal to Dr. Felsher's Report

Dr. Felsher contends it is generally accepted for "purposes of evaluating potential risks for lympho-hematopoietic cancers, that these cancers can be grouped together" (see page 11 of Dr. Felsher's report). This practice may allow for a greater number of subjects and events to be analyzed, increasing the statistical power of the analysis. However, grouping may obscure an actual effect in a smaller subset of the population. The practice also presupposes that these factors are known to play a pathogenic role in every subtype of the disease. This is clearly not always true. For example, Dr. Felsher cites the familial predisposition syndromes for non-Hodgkin lymphoma such as ataxia telangiectasia and Wiskott-Aldrich Syndrome (see page 10 of Dr. Felsher's report). However, these congenital immunodeficiencies have been mostly associated with high grade B cell lymphoma, especially in children, and often associated with the Epstein-Barr virus, but not low-grade indolent lymphoma or CLL. Autoimmune disorders such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) are associated with non-Hodgkin lymphoma, but again these autoimmune diseases are associated with more aggressive B cell lymphoma such as large B cell lymphoma, not CLL (see review by S Yadlapati and P Efthimiou. BioMed Research International 2016). One study from the Swedish Cancer Registry (Baecklund E, et al. Arthritis and Rheumatism 2003; 48: 1543-50) found "an increased incidence of one specific lymphoma subtype, [diffuse large B cell lymphoma], in RA patients, as well as a possible association with RA disease activity." Sjogren's syndrome has been associated with mucosal-associated lymphoid tissue (MALT) lymphoma, but not CLL.

Finally, immunosuppressive therapy following organ transplantation or for the treatment of autoimmune diseases increases the risk of both aggressive lymphoma and myeloid neoplasms. However, clinical studies have not associated the development of CLL with prior exposure to immunosuppressive therapies. Animal model studies are cited by the general causation experts, demonstrating the negative effects of VOC on immune cells and immune function. I agree with the evidence that these chemical agents cause DNA damage and immune defects in animal models. Nonetheless, there is no clinical data that other DNA damaging agents (like chemotherapy) or any immunosuppressive therapy is associated with CLL.

Finally, Dr. Felsher discounts Mr. Hill's exposure to jet fuel during his service (see page 21 of Dr. Felsher's report). In his deposition, Mr. Hill answers in the affirmative that he "wore gloves, masks, or anything?" And added he wore his helmet and a life vest. (Hill Dep., 124:12-19).

However, we cannot be certain as to his actual exposure to the fumes of the jet fuel during the entire time he was on deck.

IX. Conclusion

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

1. Mr. Hill has at least two demographics features associated with a greater risk for the development of CLL: male gender, and obesity. Although relatively young at the time of diagnosis, it is not unusual to present after age 50 years.
2. There is insufficient data in the literature to implicate volatile organic chemicals, such as those found in the water supply at Camp Lejeune, in the development of CLL. Despite the epidemiological available data, CLL has no known cause.
3. Mr. Hill has other known risk factors for chronic kidney disease, including cardiovascular risk factors of hypertension, hyperlipidemia, obesity, glucose intolerance, and positive family history for arterial occlusive disease (myocardial infarction). Mr. Hill also has other findings of arterial vascular disease, including hypertensive retinopathy and coronary artery disease (90% occlusion of the posterior descending artery of the heart).
4. There is no reference to hepatic steatosis in Mr. Hill's medical records.

Therefore, I believe it is highly unlikely that any potential exposure to the organic chemicals in the water supply at Camp Lejeune caused his CLL, kidney disease, or hepatic steatosis.

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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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]