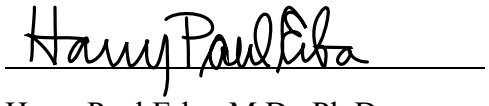
# Exhibit 476

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

***Joseph Gleesing v. United States***
***7:23-cv-01486-D***
***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:** Joseph Gleesing

**Case:** *Joseph Gleesing v. United States*, No: 7:23-cv-01486-D (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. Joseph Gleesing'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. Damian Laber's opinions in his February 7, 2025, report.

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

1. It is highly unlikely that Mr. Gleesing's CLL was caused by his 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. Gleesing's demographic profile exhibits several widely accepted features associated with a higher risk of developing CLL including age, gender, and obesity. Despite these associated demographic features, there are no environmental factors or genetic events that are known to cause CLL.
2. It is unlikely that Mr. Gleesing's CLL diagnosis will result in his premature death.

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

# II. Qualifications

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

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

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

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

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

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

## III. An Overview of Leukemia

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

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

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

Myeloid and lymphocytic leukemia are also classified as either 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. 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. 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 (European vs African/Asian ancestry), 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.

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/m2. 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/m2 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 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 "clusters of differentiation" (CD) 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 white blood cell 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 white blood cell 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 (as in the case of Mr. Gleesing). 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 of cytotoxic chemotherapy and monoclonal antibodies was the standard of care for initial therapy of CLL for at least two decades (1995 through 2015). The most used immunochemotherapeutic regimens were 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 combination of chlorambucil and obinutuzumab 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].

Several studies have investigated the combinations of more targeted therapies with immunochemotherapy, for example, the Bruton tyrosine kinase inhibitor ibrutinib (see below) with FCR [Davids MS, et al. Lancet Haematol. 2019; 6: e419-e428]. In the phase II multicenter clinical study of iFCR, 85 previously untreated CLL patients less than or equal to age 65 years were treated with up to six cycles of the FCR regimen with concomitant ibrutinib. The ibrutinib was continued as maintenance therapy following completion of the immunochemotherapy regimen. However, ibrutinib could then be discontinued after two years of maintenance, if there was no evidence of CLL in a bone marrow sample by flow cytometry (undetectable measurable residual disease, or uMRD). The rate of uMRD was greater than in historical controls treated with FCR. Furthermore, the benefit of this regimen was observed both in patients with or without IgHV somatic hypermutation. The major toxicity was related to myelosuppression (decreased blood counts), atrial fibrillation, and infection.

The use of cytotoxic chemotherapy has fallen out of favor, mostly related to the early and late toxicities associated with the chemotherapy agents, as well as the availability now of other more convenient and effective regimens. Most recently, cytotoxic chemotherapy drugs have completely been replaced by oral, targeted therapies. The two most popular regimens for initial therapy are the oral inhibitors of the Bruton tyrosine kinase (BTK) and the BCL2 (B-Cell Lymphoma 2) 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.

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 white blood cell 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.

More recently, the ALPINE trial compared the BTK inhibitors, ibrutinib and zanubrutinib, in patients with relapsed CLL [see Hillmen 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 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

Mr. Gleesing's exposure history to the water at Camp Lejeune is summarized on pages 14-16 of the Plaintiff Specific Causation Report prepared by Dr. Damian Laber. It is also summarized in the Expert Report of Dr. Judy S. LaKind. Mr. Gleesing served as an active-duty member of the United States Marine Corps from 09/01/1977 through 08/31/1981. Except for short periods of leave, Mr. Gleesing was stationed at Camp Lejeune from 01/25/1979 through 08/10/1981.

## V. Mr. Gleesing's Relevant Medical History

Mr. Gleesing was first seen by an oncologist, Dr. Mohammad Rafi at the West Michigan Cancer Center, on 09/17/2015. (01486_GLEESING_0000000996).[2] On 8/31/2015, during laboratory

[2] West Michigan Cancer Center was originally a collaboration of Bronson Healthcare Group and Borgess Medical Center until Bronson formed its own cancer center, at which time Mr. Gleesing continued his treatment at Bronson's cancer center. (Usman Dep. Tr., p 34-35).

evaluation for routine health maintenance, a complete blood count demonstrated mild leukocytosis. His WBC count was 15,700, hemoglobin 15.9 gram/dL, and platelet count 192,000. Peripheral blood flow cytometry was performed on 09/03/2015. The analysis detected 55% monoclonal B lymphocytes. The immunophenotype was consistent with chronic lymphocytic leukemia; there was expression of CD5, CD19, CD20 (dim), CD23, and dim surface kappa light chain. Only 10% of the cells expressed CD38 (CD38 negative). During this initial visit, he told Dr. Rafi he was "a veteran who was stationed at Camp LeJeune… and he is in touch with the VA for his present diagnosis." Mr. Gleesing related a history of lung cancer in his family, including his father, paternal aunt and paternal uncle, all of whom smoked cigarettes. His grandparents also had colon, prostate, and breast cancer. There is no family history of any leukemia. Mr. Gleesing never had tobacco use disorder; he denies drinking significant amounts of alcohol. Mr. Gleesing did not have any constitutional symptoms. On the day of this initial visit, he weighed 210.6 pounds, and his height was 68 inches; his body mass index is calculated to be 32.0 kg/m2 (consistent with obesity). Dr. Rafi did not find any palpable lymphadenopathy or hepatosplenomegaly. CT scans on 10/28/2015 did not detect any intra-abdominal lymphadenopathy. The spleen was at the upper end of normal in size. Dr. Rafi felt Mr. Gleesing had Rai stage 0 disease, and he recommended a watch and wait observational protocol.

Other diagnostic studies performed after diagnosis include CLL FISH panel. (01486_GLEESING_0000000940-941). There was deletion of chromosome 13q in 65.5% of the peripheral blood nucleated cells; there were no other abnormalities on the panel (including probes for ATM, TP53, chromosome 12 centromere, chromosome 6q, and IgH::BCL1). IgHV sequence analysis of peripheral blood sample on 03/21/2017 demonstrated an unmutated IgHV3-30 gene sequence.

Mr. Gleesing was evaluated by Dr. Qing Li at the University of Michigan for a second opinion on 11/25/2015. He remained asymptomatic from the CLL. There was no adenopathy or hepatosplenomegaly on physical examination. His WBC count was 14,400, hemoglobin 15.6 gram/dL, platelet count 184,000, absolute neutrophil count 6,400, and absolute lymphocyte count 7,200. The flow cytometry was repeated and confirmed the initial findings. Dr. Li agreed with the recommendation for an observational protocol. Mr. Gleesing returned to the care of Dr. Rafi at the West Michigan Cancer Center, and alternated visits with oncologists at the Ann Arbor V.A. Hospital.

Mr. Gleesing continued an observational protocol. On 02/22/2018, Mr. Gleesing was evaluated by Dr. Jing Christine Ye at the Ann Arbor V.A. Hospital (01486_GLEESING_0000000593-598). He reported pain in the right lower quadrant of the abdomen (resolved by the time of this visit) and an intentional 8-pound weight loss (after trip to Italy). His WBC count had increased to 222,800. Dr. Ye recommended continued observation. However, when he was re-evaluated by Dr. Rafi on 03/05/2018, he reported night sweats and weight loss (01486_GLEESING_0000001012-1015). He had palpable splenomegaly and palpable bilateral occipital and left axillary lymph nodes. His WBC count was 182,600. He had a mild grade 1 anemia (hemoglobin 12.4 gram/dL) and mild grade 1 thrombocytopenia (platelet count 134,000). Dr. Rafi felt his WBC count doubling time was short, and he recommended initiating therapy with

FCR. However, one week later (03/13/2018), Mr. Gleesing returned to see Dr. Rafi with new, tender submental lymph nodes, and his WBC count had increased to 208,000 (01486_GLEESING_0000001016-1019). Mr. Gleesing provided signed informed consent for a phase II clinical trial combining the BTK inhibitor ibrutinib with FCR chemotherapy (01486_GLEESING_0000003979-4000).

Bone marrow biopsy and aspirate were performed on 03/21/2018 (01486_GLEESING_0000000942-947). The bone marrow was hypercellular for age (80-90% cellularity) with chronic lymphocytic leukemia involving approximately 90% of the marrow cellularity. ZAP-70 expression by B cells was borderline negative. The cytogenetic analysis demonstrated a normal male karyotype in all 20 metaphases. The IgVH3-30 gene was unmutated (0.0% mutated). TP53 mutation was not detected. CT scan on 03/20/2018 demonstrated splenomegaly (spleen 21.3 cm, increased from prior) as well as mildly enlarged cervical, supraclavicular, axillary, mediastinal, and retroperitoneal lymph nodes. Incidental notes are made of colonic diverticulosis, enlarged prostate, and thickened bladder wall consistent with bladder outlet obstruction (01486_GLEESING_0000001023).

He started cycle #1 ibrutinib by mouth daily, fludarabine 25 mg/m2 IV daily on days 1-5, cyclophosphamide 250 mg/m2 IV daily on days 1-5, and rituximab (iFCR) on 04/10/2018. He started cycle #2 iFCR on 05/07/2018. Cycle #3 was delayed due to grade 2 thrombocytopenia (01486_GLEESING_0000001054-1058). Dr. Rafi considered a 50% dose reduction of the fludarabine, but the study's principal investigator may have suggested continuing the same doses, since the office notes indicate that Mr. Gleesing continued to receive the full dose of each medication. He started cycle #3 on 06/11/2018 (01486_GLEESING_0000001059-1063), cycle #4 on 07/16/2018 (01486_GLEESING_0000001064-1068), cycle #5 on 08/13/2018 (01486_GLEESING_0000001069-1073), and cycle #6 on 09/10/2018 (01486_GLEESING_0000001074-1078). There were no further delays. He did not require transfusion support. Mr. Gleesing experienced the following symptoms during the FCR chemotherapy: fatigue, arthralgias, headache (head MRI negative), rash, mucositis (tenderness and sores in the mouth and throat), oral candidiasis, mild hyperbilirubinemia, grade 2 thrombocytopenia, calf hematoma (see below), and rash. He reports worsening peripheral neuropathy in a hand-written note on 06/12/2024 (01486_GLEESING_0000006869-6870), potentially related to fludarabine but may also be due to pre-diabetes. Hypogammaglobulinemia is related to both the CLL and therapy. He has started gamma globulin infusions; the first infusion occurred on 06/28/2024. (01486_GLEESING_BMH_0000001864).

Bone marrow biopsy and aspirate were performed on 07/02/2018 (after three cycles of iFCR). The bone marrow sample was a limited specimen, but there were no definite features of CLL (01486_GLEESING_0000000948-950). The MRD flow cytometry of the bone marrow aspirate was negative for CLL/SLL (01486_GLEESING_0000000957). CT scan on 11/06/2018 did not demonstrate any adenopathy or splenomegaly. There had been no change compared with the CT scan on 07/05/2018 (01486_GLEESING_0000001087). He had repeat bone marrow biopsy and aspiration on 11/07/20218 (following completion of the iFCR) (01486_GLEESING_0000000959-961). The bone marrow was normocellular for age (40% cellularity) with evidence of trilineage

hematopoietic maturation and few nonspecific non-necrotizing granulomas. There was no morphologic, immunohistochemical, or flow cytometric evidence of involvement by chronic lymphocytic leukemia. The iron stores were markedly decreased. There was mild thrombocytopenia of the peripheral blood (platelet count 124,000). MRD analysis of the marrow aspirate sample by flow cytometry was negative for CLL/SLL (01486_GLEESING_0000000957).

He had hemorrhagic complications related to ibrutinib. He did experience a hemorrhage in the right calf likely related to ibrutinib on 07/18/2018. He had hematuria while on ibrutinib alone; cystoscopy on 02/11/2019 was normal. The hematuria was attributed to the enlarged prostate but could have been exacerbated by the ibrutinib as well (01486_GLEESING_0000001090). He developed a right thigh hematoma with minimal activity. He had also taken aspirin and turmeric around this time. The ibrutinib was held to allow healing (01486_GLEESING_0000001118-1123), and Dr. Usman advised him to stop turmeric, aspirin, and other non-steroidal anti-inflammatory drugs.

Peripheral blood MRD detection by flow cytometry was negative for CLL cells on 07/02/2018, 11/07/2018, 03/14/2019, 09/26/2019, and 04/22/2020 (01486_GLEESING_0000000951). On 10/19/2020, he completed two years for ibrutinib maintenance, and he remained in MRD negative complete remission by peripheral blood MRD flow cytometry. He was instructed to discontinue the ibrutinib per protocol, which he did (01486_GLEESING_0000001136-1141). He has remained in remission. The peripheral blood MRD flow cytometry was negative for CLL cells on 11/09/2021, 05/09/2022, 11/11/2022, 06/06/2023, 12/07/2023, and 06/12/2024 (01486_GLEESING_BMH_0000002219).

# VI. Other medical problems unrelated to CLL but included in the medical records provided

1. Coronary Artery Disease
   a. 04/11/2013: Cardiac catheterization shows 70% diffuse stenosis of the mid right coronary artery, requiring a stent 4/11/2013 (01486_GLEESING_0000000009-11).
   b. 05/10/2016: A second coronary artery stent was placed (01486_GLEESING_0000000012-15).
2. Dilated ascending aorta / ascending aortic aneurysm
   a. 07/05/2018: CT scan of chest and abdomen ascending aorta slightly dilated, 4.3 by 4.3 cm at level of the right pulmonary artery (01486_GLEESING_BMH_0000002239).
   b. 09/22/2020: transthoracic echocardiogram demonstrates thickening of the aortic valve, consistent with sclerosis, mildly to moderately dilated ascending aorta, right ventricle moderately to severely dilated. (01486_GLEESING_0000006622-6623).
   c. 10/31/2020: CT scan of chest and abdomen ascending aorta 4.4 cm. (01486_GLEESING_0000006628).
   d. In January 2024, the ascending aorta remains dilated, measuring 4.5 cm.

(01486_GLEESING_0000006617).

3. Hypertension 9/22/2009 (01486_GLEESING_BMH_0000002369).
4. Hyperlipidemia (01486_GLEESING_0000006364).
5. Prediabetes (01486_GLEESING_0000006364).
6. Hepatic steatosis (fatty liver) 9/28/2015 (01486_GLEESING_0000000977).
7. Sleep apnea 04/11/2016 (01486_GLEESING_0000002448-2449).
8. Hand surgery (01486_GLEESING_0000006366).
9. Umbilical hernia repair 10/23/2015 )01486_GLEESING_0000002636).
10. Intraductal papillary mucinous neoplasm (IPMN) 11/11/2022 (01486_GLEESING_BMH_0000001304, 1308).
11. Pancreatic divisum 11/11/2022 (01486_GLEESING_BMH_0000001311).
12. Hypogammaglobulinemia 3/7/2022 (01486_GLEESING_BMH_0000000887).
13. Upper airway cough syndrome (01486_GLEESING_0000006364).
14. Benign prostatic hyperplasia (01486_GLEESING_0000006364).
    a. Prostate enlargement 9/28/2015 [977] 10/19/2020 (01486_GLEESING_BMH_0000002114-2115).
    b. Bladder wall thickening 11/28/2018 (01486_GLEESING_BMH_0000002097).
    c. Elevated PSA 10/19/2020 (01486_GLEESING_BMH_0000002114-2115).

# VII. Analysis

## A. It is highly unlikely that Mr. Gleesing's exposure to water at Camp Lejeune caused his CLL.

As stated above, it is my opinion that it is highly unlikely that Mr. Gleesing's exposure to water at Camp Lejeune caused his CLL.

There is no known cause of CLL. There is insufficient evidence linking the specific contaminants at Camp Lejeune to CLL. Furthermore, Mr. Gleesing has at least three known demographic features associated with a higher risk of CLL compared with people without these features. Men are 1.8 times as likely to be diagnosed with CLL compared with women (6.75 and 3.65 cases per 100,000 men and women, respectively). He is Caucasian, although without a thorough genealogy we cannot state if he has African or Asian ancestry. Based on the reported height and weight (BMI >= 30 kg/m2) around the time of his CLL diagnosis, he was obese: BMI 30 kg/m2 on 11/25/2015 (01486_GLEESING_0000000889) and 32.0 kg/m2 by my calculation at the time of diagnosis. Mr. Gleesing was 56 years old at the time of his initial diagnosis. The median age at diagnosis is 70 years in the Surveillance, Epidemiology, and End Results (SEER) registry. However, 31.5% of CLL patients are younger than 65 years at diagnosis (https://seer.cancer.gov/statfacts/html/clyl.html).

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 |

Like many patients, Mr. Gleesing did not require therapy at the time of diagnosis. Although del(13q) is associated with a more favorable prognosis, CLL patients with unmutated IgHV gene typically progress more rapidly and require therapy. Mr. Gleesing did have progressive lymphocytosis with a WBC count greater than 200,000 in early 2017. Mr. Gleesing also had progressed from Rai stage 0 at diagnosis to Rai stage II CLL when he initiated therapy. He had symptoms that were annoying to him including night sweats and tender adenopathy. It was reasonable to begin therapy at that time. He achieved a remarkable complete remission that is negative by sensitive flow cytometry. He has been able to discontinue all therapy. He has been off ibrutinib for 4.5 years at this point without evidence of relapse. This is even more striking given the fact that responses to FCR are less durable in CLL patients with unmutated IgHV gene.

Hypogammaglobulinemia is related to both CLL and therapy. Dr. Usman commented on recent recurrent URI without hospitalizations in his progress note dated 06/12/2024 (01486_GLEESING_BMH_0000002194-2200), and he recommended starting intravenous immunoglobulin (IVIG) replacement therapy at that time. Peripheral neuropathy could also be due to fludarabine, but neuropathy usually presents during therapy, and Mr. Gleesing did not report neuropathy at that time. The pre-diabetes may also be contributing to his symptoms of peripheral neuropathy. Furthermore, I have not been able to review the neurology consultation to confirm that Mr. Gleesing's symptoms are due to peripheral neuropathy, and that other causes have been excluded.

Given Mr. Gleesing's deep and durable response to iFCR to date, and the absence of any follow up data regarding long term outcomes with this investigational therapy, it is very unlikely that this diagnosis will result in his premature death. Furthermore, if he does relapse, there are other effective treatments available including second and third generation BTK inhibitors, the BCL2 inhibitor venetoclax, obinutuzumab, BiTE and CAR-T cell therapies.

## VIII. Response and Rebuttal to Dr. Laber's Expert Report

I disagree with some of the assertions made by Dr. Laber in his report (dated Feb. 7, 2025).

As stated above, it is very difficult to conclude, as Dr. Laber does, that Mr. Gleesing's CLL diagnosis will lead to his early death. Mr. Gleesing has other known medical conditions that pose a risk to his life expectancy including known coronary artery disease. His IPMN puts him at an increased risk of pancreatic cancer (01486_GLEESING_BMH_0000002193). Dr. Usman also expressed concern for the rapid increase in the ascending aortic aneurysm over the prior year in his progress note on 12/07/2023 (01486_GLEESING_BMH_0000002187).

Dr. Laber and general causation experts cite the New Jersey study of exposure to volatile organic compounds (VOC) in drinking water and the incidence of leukemia and non-Hodgkin lymphoma [Cohn P, et al. Environ Health Perspect. 1994; 102: 556-561]. There are several methodological issues with this population-based study that limit the applicability of the conclusions to this case. First, the authors could not adjust their models for all known risks associated with a higher incidence of CLL. The authors were able to comment on the effect of age and gender, but the available data precluded an analysis based on other known risks for CLL including obesity and family history of leukemia. Although self-reported ethnicity was collected in the New Jersey State Cancer Registry, the potential effect of ethnicity was not described in the report. It is conceivable that the regions of higher volatile organic chemical exposures also had higher prevalence of other demographic characteristics associated with CLL. A multivariate analysis including all known risks for CLL would be required to conclude that the VOC exposure was the cause of CLL in these people. Since the reported relative risks are so close to unity, an imbalance in other known risk factors could potentially affect the conclusions. The incident cases of primary leukemia and lymphoma were collected between 1979 and 1987, but the first time that the VOC levels were measured was in 1984-1985. It may be reasonable to assume that the water supply was contaminated prior to the first rounds of mandatory water testing; however, additional cases were cases not collected after 1987. If the exposure to VOC contamination of the New Jersey water supply was responsible for the reported higher incidence of CLL in this cohort, these people likely already had MBL, given the natural history of MBL and progression to CLL reviewed by Shanafelt and colleagues [Shanafelt TD, et al. Leukemia 2010; 24: 512-520]. Given the limited timing of the collection of leukemia cases from the New Jersey State Cancer Registry, this analysis does not allow us to conclude that an exposure to VOC may be a cause of CLL decades later, as in this case. Although the authors point out that the geographic regions evaluated in this study did not "exhibit major population influx" during the 1970-1980 period, there was no attempt to evaluate each reported case of cancer with time of residency in one of the New Jersey counties.

With these methodological weaknesses we can now evaluate the results in the analysis by Cohn et al. CLL would have been included in the low-grade NHL group. The relative risk of developing low-grade lymphoma was numerically higher with trichloroethylene (TCE) exposure, but not statistically increased. The relative risk was numerically lower with the higher TCE exposure compared to lower. The lack of a relation between exposure dose and risk of low-grade lymphoma further argues against a causal relation. The relative risk of CLL was numerically, but not statistically, higher even at the higher level of TCE exposure. There was no relation between CLL and perchloroethylene (PCE). In fact, the relative risk of CLL was numerically (but not statistically) lower with exposure to PCE than without.

Despite other reports in the literature similar to that of Cohn et al. (1996) and given methodological issues common to many population-based epidemiologic studies, there remains no known cause of CLL. The American Cancer Society does not recognize any definite environmental agent as causative of CLL.

Dr. McCabe addresses the evidence citing the effects of volatile organic chemicals on the immune system. Immune dysfunction is hypothesized to be a contributing factor for the development of neoplasia (cancer) in general due to decrease in immune surveillance. However, not all cancers are associated with deficiencies in our immune system. For example, congenital immunodeficiency states are not associated with CLL. Immunosuppressive therapy with drugs such as the calcineurin inhibitors, methotrexate, and azathioprine is associated with subsequent development of skin cancers and myeloid neoplasms, but again not CLL. There is no clinical data to suggest that immune injury of any kind is a cause of CLL. Furthermore, Mr. Gleesing had no evidence of an auto-immune disorder or immunodeficiency syndrome prior to the diagnosis of CLL. The pathogenesis of CLL is unknown, but the current favored hypothesis suggests an exaggerated response to autoantigens or common environmental antigens.

## IX. Conclusions

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

1. Mr. Gleesing has at least three factors associated with a greater risk for the development of CLL: male gender, European ancestry, and obesity. There is insufficient data in the literature to implicate VOCs, 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. It is my opinion therefore that Mr. Gleesing's CLL is highly unlikely to be related to or caused by his exposure to any contaminants in the water at Camp Lejeune 35 years earlier.

2. Mr. Gleesing achieved an MRD negative complete remission of his CLL that has been durable for 4.5 years without any therapy, and there are effective therapy options if the CLL does recur. In light of this and his other unrelated medical problems, it is unlikely that his life expectancy has been shortened by the diagnosis of CLL.

# 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):**

1. Varley JM, MacGregor HC, **Erba HP**. Satellite DNA is transcribed on lampbrush chromosomes. Nature 1980; 283: 686-8.

2. Varley JM, McGregor HC, Nardi I, Andrews C, **Erba HP**. Cytological evidence of transcription of highly repeated DNA sequences during the lampbrush stage Triturus cristatus carnifex. Chromosoma 1980; 80: 289-307.

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16. O'Brien S, Berman E, Ghalla K, **Erba HP**, Wetzler M et al. Chronic Myelogenous Leukemia. J Natl Compr Canc Netw 2005; 3: 732-555.

17. Collins CD, DePestel DD, Stuntebeck ER, **Erba HP**, Stevenson JG. Cost Analysis of a Ferbrile Neutropenia Algorithm with Respect to Empirical Antifungal Therapy in Adult Hematology/Oncology Patients: PIN5. Value in Health 2005; 8: 306.

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134. Anjali S. Advani, Anna Moseley, Kristen M. O'Dwyer, Brent L. Wood, Jae Park, Matthew Wieduwilt, Deepa Jeyakumar, George Yaghmour, Ehab L. Atallah, Aaron T. Gerds, Susan M. O'Brien, Jane L. Liesveld, Megan Othus, Mark Litzow, Richard M. Stone, Elad Sharon, **Harry P. Erba**. Dasatinib/prednisone induction followed by blinatumomab/dasatinib in Ph+ acute lymphoblastic leukemia. Blood Adv (2023) 7 (7): 1279–1285.

135. Era L. Pogosova-Agadjanyan, Xing Hua, Megan Othus, Frederick R. Appelbaum, Thomas R. Chauncey, **Harry P. Erba**, Matthew P. Fitzgibbon, Isaac C. Jenkins, Min Fang, Stanley C. Lee, Anna Moseley, Jasmine Naru, Jerald P. Radich, Jenny L. Smith, Brooke E. Willborg, Cheryl L. Willman, Feinan Wu, Soheil Meshinchi & Derek L. Stirewalt. Verification of prognostic expression biomarkers is improved by

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

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

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**Chapters in books**

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

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

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

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

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

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

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

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

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

Daiichi Sankyo Pharmaceutical
Kura Oncology
Servier
Sumitomo Pharma

**Honors and Awards**

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

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

Intramural Committee and Administrative Service

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

Extramural Committee, Organizational, and Volunteer Service

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

Scientific Steering Committees and DSMB Positions

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

**Memberships in Professional Societies**

Active Member, American Society of Hematology

Active Member, American Society of Clinical Oncology

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

**Teaching Activities**

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

Stanford University:

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| 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:

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| 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:

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|---|---|
| 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]