Exhibit 455

Karen Marie Amsler v. United States of America

U.S. District Court for Eastern District of NC, Southern Division

Case No. 7:23-cv-00284

Specific Causation Expert Report of Lukasz P. Gondek, MD, PhD

Lukasz Gondek, MD, PhD
1340 Smith Avenue, Suite 200
Baltimore, Maryland 21209

February 7, 2025

Pat Telan
Bell Legal Group
291 Ridge St.
Georgetown, SC 29440

Dear Mr. Telan,

I am writing this report in my capacity as a scientist and practicing hematologist concerning the case involving Mrs. Karen Amsler, a 64-year-old female who was diagnosed with Acute Lymphoblastic Leukemia (ALL) at the age of 60 years and subsequently treated with several cycles of chemotherapy followed by allogeneic hematopoietic cell transplantation (alloHCT). Her diagnosis and treatment were complicated by leukemic retinopathy, worsening bone loss, deep vein thrombosis requiring long-term anticoagulation, atrial fibrillation requiring antiarrhythmic medication, and dry eyes due to chronic GVHD.

In an effort to provide a comprehensive analysis, I have examined the relevant medical data and documentation, including pathology reports and clinical documentation, as listed below:

- May River Dermatology, Bluffton, SC
- Medical University of South Carolina Health, Charleston, SC
- Low Country Cancer Care Hematology/Oncology, Okatie, SC
- Optim Orthopedics, Savannah, GA
- Charleston ENT and Allergy
- Memorial Health University Medical Center-Hollings Cancer Center, Charleston, SC
- Memorial Health University Physicians-Women's Care, Savannah, GA

The findings and conclusions detailed in the following report are based solely on the information made available to me at the time of the review.

Please be aware that the views expressed herein are provided with the understanding that I am acting in the role of a medical expert. I am not a direct caretaker of the patient. Consequently, the details of the report should be regarded as professional recommendations and observations that reflect my interpretation of the materials reviewed. I hold all opinions to a reasonable degree of medical certainty.

Expert Qualifications

I am a physician-scientist with a Ph.D. in cancer genomics and post-doctoral training in cancer biology and genetics of blood cancers. In addition to being a practicing hematologist/oncologist, I also lead a research laboratory studying cancer genomics, cancer biology, and the role of DNA mutations in cancer development and progression. I obtained my M.D. degree from the Medical University of Silesia in 2003, and my Ph.D. from the University of Warsaw. Following my graduation from medical school, I undertook a research fellowship at the Cleveland Clinic and studied the pathogenesis and genomics of leukemia. Following 4 years in the laboratory, I completed Internal Medicine Residency training at the Cleveland Clinic and Hematology Fellowship at Johns Hopkins. I joined the faculty of Johns Hopkins University in 2014. For the last decade, my academic career has focused on cancer genomics and the role of DNA aberrations in cancer development and progression. I have published over 60 original research manuscripts, 10 review articles and contributed to 4 book chapters as well as review articles and editorials. My research is frequently cited, with over 4000 citations in scientific literature. I also participate extensively in peer-review activities, serving as a reviewer for many cancer journals as well as study sections for both private and governmental funding organizations, including The European Hematology Association (EHA) and the National Institute of Health. I also served as a Scientific Session Chair for the American Society of Hematology and a Steering Committee Member for the Break Through Cancer initiative.

In addition to my research and clinical activities, I am actively involved in the education of future generations of physicians and scientists. At Johns Hopkins University, I serve as a clinical educator in the Oncology Department. I am a preceptor to internal medicine residents and hematology and oncology fellows. In addition to serving as a teacher in inpatient leukemia service, I am also an organizer of the weekly Leukemia Tumor Board for faculty and fellows. As an expert in leukemia biology and treatment, I have been leading the discussion on a particularly challenging leukemia case, where my specialized knowledge in molecular pathogenesis and advanced therapeutic strategies is critical to guiding prognosis and optimal patient management. I have been an active participant in medical student education on the topics of oncology, regenerative medicine, and cancer stem cells. Furthermore, I have served as an advisor for several postdoctoral fellows and undergraduate students. I have been the mentor for 12 postdoctoral fellows, internal medicine residents, as well as medical and graduate students. Several of my mentees successfully competed for prestigious awards such as the Molina-Grasmick Scholar Award and the American Society of Hematology HONORS Award.

My work has been recognized and supported by governmental and private funding agencies, including the National Institute of Health, Aplastic Anemia, and MDS International Foundation (AA&MDSIF), Edward P. Evan Foundation, Hopkins-Allegheny Health Network (AHN), and the Break Through Cancer Initiative.

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Case Synopsis

Mrs. Karen Amsler is a 64-year-old female with a past medical history of osteoporosis, migraines, rosacea and multiple liver cysts requiring laparoscopic fenestration. Mrs. Amsler had been in her usual state of health until September 12, 2020, when she presented to Memorial Health University Medical Center Emergency Department in Savannah, GA with acute onset central vision loss in her right eye. Her complete blood count at that time demonstrated marked leukocytosis with total white blood cell count of 608,000/ul (normal range 4,500-11,000/ul) with 64% leukemic blasts, anemia with hemoglobin of 8.7 g/dl and thrombocytopenia with platelet count of 138,000/ul. Computed tomography (CT) of the head showed no acute intracranial process. Magnetic resonance imaging (MRI) of the brain showed no acute process but diffuse abnormal marrow signal consistent with marrow infiltrative process was noted. She was also found to have deep vein thrombosis (DVT) in left lower extremity. Ophthalmologic exam revealed leukemic retinopathy.

Bone marrow biopsy performed on September 13, 2020, confirmed the diagnosis of B cell acute lymphoblastic leukemia (B-ALL). Cytogenetics studies revealed KMT2A (MLL) gene rearrangement and the absence of BCR/ABL rearrangement. Metaphase karyotype demonstrated t(4:11) (q21;q23). Lumbar puncture demonstrated no leukemia in cerebrospinal fluid. She began her combination chemotherapy, Hyper-CVAD cycle A1 (cyclophosphamide, vincristine doxorubicin and dexamethasone) started with intrathecal methotrexate. She was also treated for DVT, initially with a blood thinner, Lovenox, followed by Xarelto. She then received Hyper-CVAD cycle B1 (methotrexate, cytarabine) on October 10, 2020.

On October 27, 2020, she was seen by Dr. Praneeth Baratam at the Medical University of South Carolina for a consultation regarding possible allogeneic hematopoietic cell transplantation (alloHCT). Bone marrow biopsy performed at the time of the visit demonstrated complete remission with no flow cytometric evidence of residual leukemia.

Mrs. Amsler received cycles A2, B2 which were complicated by atrial fibrillation with rapid ventricular response, which was rhythm controlled with Flecainide. She then completed cycle A2 and additional 4 intrathecal treatments. Repeat bone marrow biopsy on December 29, 2020, confirmed complete remission with negative measurable residual disease (MRD) by multiparametric flow cytometry (sensitivity 1 in 10000 cells). She then underwent matched unrelated (10/10 match) alloHCT with peripheral blood stem cells and fludarabine, melphalan conditioning regimen on BMT CTN 1703 clinical trial and was randomized to tacrolimus, methotrexate graft vs. host (GVH) prophylaxis on January 14, 2021. Her early post-alloHCT course was complicated by likely methotrexate-induced transaminitis that subsequently resolved.

A repeat bone marrow biopsy on April 26, 2021 (day +102 post alloHCT) demonstrated ongoing remission with 100% donor chimerism. On day +116 she was noted to have a worsening rash that coincided with tapering off tacrolimus. The rash was consistent with skin GVH grade 2 treated with triamcinolone and clobetasol and increased tacrolimus dose which resulted in clinical improvement. Bone marrow biopsy on July 12, 2021 (6 months post-alloHCT) showed no evidence of ALL and 100% donor chimerism. Tacrolimus was slowly tapered off and discontinued at 9 months post-alloHCT with no recurrence of skin GVH.

Her bone marrow biopsy at 1-year post-alloHCT demonstrated ongoing remission. She had been experiencing fatigue (Dep p. 119) and dry eyes post-alloHCT which is a known complication after alloHCT and one of the symptoms of chronic GVHD (Dep p.121).

With regard to her medical history, Mrs. Amsler's father had a heart condition that resulted in a stroke and a cerebral bleed after the fall and prostate cancer. Her mother had Parkinson's disease and atrial fibrillation. Neither of her parents were diagnosed or treated for any kind of leukemia (Dep. p. 32-36). Ms. Amsler's maternal grandmother had colon cancer and breast cancer. Her paternal grandfather may have had esophageal cancer (Dep. p. 37-38). She has 3 siblings, 2 sisters and 1 brother, none of whom were diagnosed with any type of cancer (Dep. p. 26-28) and do not have any serious medical conditions. Her brother has hypertension and her sister has osteoporosis. Mrs. Amsler has no children. She has 6 siblings, all of whom are healthy (Dep. p. 29-31). None of the family members suffered from hematologic malignancy. Mrs. Amsler never smoked and only drank alcohol socially (Dep. p. 135).

Concerning environmental exposure, during periods from approximately October 1965 to June 5, 1967, when Mrs. Amsler was ages five to seven years old, Mrs. Amsler was exposed to contaminated water containing trichloroethylene (TCE) and benzene at Camp Lejeune. Mrs. Amsler testified that she moved to North Carolina in October of 1965 when her father was relocated there by the Navy. (Dep. p. 90:19-91:3; 94:19-23). Her family initially resided off-base from October 1965 until May or June of 1966 (Dep. p. 96:21-25); however, she occasionally visited the base during this time for activities such swimming and shopping. (Dep. p. 95:13-98:5). From May 25, 1966 until June 5, 1967, a period of 12 months 2 weeks, base housing records denote that she and her family lived at 2517 Saint Mary's Drive, located in the Paradise Point area of Camp Lejeune. (See CLJA_CLHousing-0000006052). Further records and Mrs. Amsler's testimony confirm that she attended school, the first grade, on Stone Street on base for the 1966-67 school year. (Amsler Dep. p. 97:4-6; see also 00284_AMSLER_0000007966),

Methods

As a clinician-scientist specializing not only in the treatment of hematologic cancers, including but not limited to ALL, but also studying the molecular underpinning of hematologic cancers, my approach to determining specific causation integrates clinical expertise, research experience, and evidence-based methodologies. The assessment begins with a comprehensive review of the patient's medical history, the detailed assessment including molecular underpinning of patient's cancer, pre-existing conditions, possible genetic predispositions, and any prior exposures that may contribute to the disease. I also perform an analysis of the individual's occupational, environmental, and lifestyle exposures, focusing on potential links to carcinogenic substances.

To establish causation, I rely upon a differential etiology analysis, a systematic method to evaluate all plausible causes of the disease. This process seeks to determine causation to a level of "at least as likely as not," and involves a consideration of potential confounders given the unique circumstances of the individual claimant involved. I utilize my years of education, training and experience to determine which factors can be ruled out as potential causes, but I want to make it clear that the term "rule out" does not mean that I can eliminate the confounder as a causative factor to a degree of 100%, but that I do so to a reasonable degree of scientific/medical probability. To the extent that I am able to say that a single factor is the most likely cause of Mrs. Amsler's leukemia, my causation opinion may be phrased in a "more likely than not" manner. If there are competing causes for which I am unable to determine which is the most likely cause of Mrs. Amsler's leukemia, my causation opinion will be phrased in an "at least as likely as not" manner. I use my expertise in hematologic malignancies to interpret clinical findings, including the molecular and cytogenetic profiles of the patient's cancer. These profiles may reveal specific biomarkers or mutational signatures associated with exposure to carcinogens, providing a direct mechanistic link between the exposure and disease development.

Additionally, I incorporate insights from epidemiological studies and toxicological data, which demonstrate population-level associations between specific exposures and hematologic malignancies (please see my general causation report which is incorporated in its entirety by reference in this report). This evidence is then contextualized within the patient's individual exposure history, dose-response relationship, and latency period. By synthesizing these elements, I can provide a scientific opinion on whether the exposure to TCE, PCE and benzene are at least as likely as not to be the cause of the patient's condition. This comprehensive approach reflects my experience as a clinician treating hematologic cancers and a scientist advancing our understanding of their pathogenesis.

Molecular and cytogenetic profile of plaintiff's B-ALL

Mrs. Amsler was diagnosed with B-ALL with a certain type of genetic rearrangement denoted as (4;11)(q21;q23)**.** In this type of genetic change also known as chromosomal translocation, parts of two chromosomes—chromosome 4 and chromosome 11—break and exchange segments. This creates a fusion of two genes: MLL or KMT2A (on chromosome 11) and AF4 (on chromosome 4). This genetic rearrangement leads to the production of an abnormal protein that disrupts the normal regulation of genes involved in blood cell development. As a

result, immature white blood cells (called lymphoblasts) start growing uncontrollably. Research has established a strong association between 11q23 translocations and exposure to cytotoxic agents, including certain topoisomerase II inhibitors such as anthracyclines or etoposide, which are frequently used chemotherapeutic agents in solid and hematologic malignancies, alike (Felix, Kolaris, and Osheroff 2006; Cowell and Austin 2012; Ezoe 2012; Pendleton et al. 2014). While it is possible that an 11q23 rearrangement may occur spontaneously, research has shown that this specific type of rearrangement is more likely to be associated with exposures to cytotoxic agents such as benzene, TCE and PCE. In Mrs. Amsler's case, the molecular results further support the notion that her B-ALL was indeed caused by exposure to cytotoxic agents. Importantly, metabolites of benzene were demonstrated to act as topoisomerase II inhibitors (Chen and Eastmond 1995; Lindsey et al. 2004) and 11q23 rearrangements were seen in white blood cells obtained from people exposed to benzene. (Zhang et al. 2007; Vaughan et al. 2005).

Hereditary factors

ALL is predominantly a non-hereditary disease; however, certain hereditary factors can contribute to its development in some cases. Genetic predisposition to ALL is usually recognized in the context of syndromic disorders or familial leukemia predisposition syndromes. These syndromes are usually suspected when multiple relatives are affected by ALL or other hematologic malignancies or solid tumors. Inherited mutation in several genes were reported to be associated with ALL and PAX5, ETV6**,** TP53, IKZF1 and others (Greaves 2018). 11q23-rearranged ALL is not generally considered hereditary.

Mrs. Amsler's father had prostate cancer, and her maternal grandmother had colon cancer and breast cancer, and her paternal grandfather may have had esophageal cancer (Dep p. 37-38). She has 3 siblings, 2 sisters and 1 brother, none of whom were diagnosed with any type of cancer (Dep p. 26-28) and do not have any serious medical condition. Her brother has hypertension and her sister has osteoporosis. Mrs. Amsler has no children. She has 6 siblings, all of whom are healthy (Dep p. 29-31). None of the family members suffered from malignancy.

Given the fact that some family members were diagnosed with solid tumors, I did consider hereditary syndrome as a possible etiology of her ALL. However, none of Mrs. Amsler's relatives have been diagnosed with hematological malignancy. Also given the molecular profile of her ALL, namely 11q23 rearrangement, it is highly unlikely that her cancer was a consequence of hereditary predisposition. Based upon my education, training and experience, it is my professional opinion, to a reasonable degree of medical/scientific certainty that hereditary factors can be ruled out as a cause of Mrs. Amsler's ALL.

Social and medical factors

The detailed review of her medical records did not identify any underlying medical conditions, or history of radiation or chemotherapy prior to his diagnosis of ALL that could have potentially contributed to Mrs. Amsler's cancer. She was not a smoker and he was not obese (BMI ~23 based on available medical records).

Occupational exposure to potential carcinogens

Mrs. Amsler is a microbiologist by training. Her first employment was at the American Medical Laboratories in Fairfax, Virginia from approximately 1984 until 1986 (Dep. p. 45) where she worked as a medical technologist, performing hematology blood work, urinalysis, and coagulation studies (Dep. p. 59). She then worked at Bancroft Medical Laboratories in Wilmington, Delaware from approximately 1986-1987 (Dep. p. 46) where she was a general microbiologist and performing general chemistry and hematology tests (Dep. p. 59-60). She then worked part-time as a medical technologist for Christiana Care, originally Medical Center of Delaware in Newark, Delaware until approximately 1998 (Dep. p. 46) in their microbiology lab performing bacterial and fungal cultures as well as virology tests (Dep. p. 60) and concurrently a full-time position at the Children's Hospital of Philadelphia in their virology lab (Dep. p. 47). She transferred to DuPont Pharmaceuticals where she was employed from 1998 to 2001 (Dep. p. 49-50). Her work focused on preclinical antimicrobial drug discovery (Dep p. 62). Mrs. Amsler then took a position at Enanta Pharmaceuticals in Watertown, Massachusetts from 2002 to 2003 (Dep. p. 50) where she was also involved in new antimicrobials testing in vitro and in vivo in small animal models (Dep. p. 62). She then accepted the position at the Johnson & Johnson companies in Raritan, New Jersey, Ortho-McNeil. She worked there from approximately 2003-2019. She started as a scientist and then transferred to the Research and Development and was supporting sales, attending conferences and doing the microbiology support for an antibiotic levofloxacin (Dep. p. 61-62). In March 2020, she found a job at St. Joseph's Hospital in Savannah, GA as a medical technologist in the clinical lab but stopped working in September when she was diagnosed with leukemia (Dep. p. 52). Given the nature of her work as a microbiologist and contact with a variety of bacteria and viruses she was unable to perform her duties given her immunocompromised status due to antileukemia therapy and alloHCT (Dep. p. 67-68).

Mrs. Amsler testified that she worked around biological hazardous material including tissue and bodily fluids from humans and animals, and from bacterial cultures while working as a medical technologist and research and development from 1986-2020 (Dep. p. 80-82). Of note, biological hazards include bacteria, viruses, parasites and fungi that can pose a threat to human health when they are inhaled, eaten or come in contact with skin. They can cause illnesses such as bacterial, fungal and viral infection. However, I saw no evidence that Mrs. Amsler was exposed to genotoxic chemicals or other substances in her workplace that would be considered causative of her ALL. Additionally, Mrs. Amsler testified that while working as a microbiologist, she would have been using personal protective equipment, thereby minimizing, if not completely negating any true exposure to biological hazardous materials. (Dep. p. 69 line 20-p.70 line 4; p. 81 lines 14-22; and p. 82 line 7-p. 83 line 2).

In my review of the depositions of Mrs. Amsler, Jennifer Yanucci MD and Praneeth Baratam MD, each were questioned about Mrs. Amsler's workplace exposures. Mrs. Amsler was asked several questions about her job duties at Johnson and Johnson in the 2003 to 2019 timeframe. Mrs. Amsler testified generally that she transitioned from her job as a scientist into the research and development side of the company, which involved "supporting the sales side, microbiologist, and attending conferences and things. Supporting the – doing the microbiology support for Levaquin, levofloxacin" (Dep. p.51 line 7 – p.52 line 2).

Subsequently, Dr. Yanucci and Dr. Baratam, two of Mrs. Amsler's treating oncologists were questioned about the possibility that Mrs. Amsler's workplace exposures may have contributed to her ALL. Dr. Baratam, in particular, did not believe that her workplace exposure to Levaquin was causative of her ALL. (Dep. of Dr. Baratam p.48 line 20 – p. 49 line 21). While Dr. Yanucci testified that she did not get very specific with Mrs. Amsler in terms of her microbiology duties at Johnson and Johnson, she did not believe that her work there had an "impact" on her development of ALL. (Dep.of Dr. Yanucci p. 90 lines 12-18).

Since the association between type II topoisomerase inhibitors and secondary leukemias, particularly with 11q23 rearrangement, has been well-established, there may be a question whether levofloxacin (Levaquin), the inhibitor of bacterial type II topoisomerase, may have been causative of Mrs. Amsler's ALL. Levaquin is a fluoroquinolone antibiotic that acts as a potent inhibitor of bacterial type II topoisomerases, specifically DNA gyrase and topoisomerase IV, which are essential for bacterial DNA replication and transcription. At therapeutic concentrations, levofloxacin exhibits selective inhibition of bacterial enzymes without significant activity against human topoisomerase II. This selectivity is attributed to structural differences between bacterial and human topoisomerases, which minimizes off-target effects on human cells. Supporting studies confirm this differential activity, indicating the safety of levofloxacin in targeting bacterial infections without compromising human topoisomerase function.(Fief et al. 2019) Also, there is no scientific evidence that exposure to Levaquin causes leukemia generally, or ALL specifically. Finally, as per above, Mrs. Amsler has testified that she would have been using personal protective equipment during her work, thereby minimizing, if not completely negating any true exposure to biological hazardous materials. (Dep. p. 69 line 20 – p. 70 line 4; p. 81 lines 14-22; and p.82 line 7- p.83 line 2).

Based upon the above, it is my professional opinion, to a reasonable degree of medical probability, that any workplace exposures, including Levaquin while working as a microbiologist at Johnson and Johnson would not have caused or contributed to Mrs. Amsler's ALL.

Based upon my education, training and experience, it is my professional opinion, to a reasonable degree of medical/scientific certainty that any job exposures outside of Camp Lejeune can be ruled out as a cause of Mrs. Amsler's ALL

While there were questions asked of the above treating physicians about whether Mrs. Amsler's history of alcohol use could have caused her ALL, neither Dr. Yanucci nor Dr. Baratam opined that alcohol use was a risk factor for ALL, and I agree with them on this point. That is, it is my professional opinion, to a reasonable degree of medical probability, based upon my education, training and experience as an oncologist and a scientist who researches blood cancers, that alcohol use is not a risk factor for ALL.

Environmental exposure to benzene and TCE

The relationship between the above contaminants and leukemia was discussed in detail in my general causation report, which is incorporated by reference into this report. Benzene is a potent carcinogen, and its ability to cause cancer is closely associated with either direct damage

to DNA or indirectly through a weakening of the immune system (Guo 2022). Benzene is metabolized in the bone marrow; thus, people exposed to benzene are particularly at risk for blood cancers. Because of these mechanisms, and particularly because benzene is a tumor initiator, it is at least as likely as not that the levels of benzene in the water at Camp Lejeune are biologically significant and can lead to cancer, including leukemia. Given this overwhelming body of scientific evidence, the International Agency for Research on Cancer (IARC) has classified benzene as a Group 1 carcinogen (IARC's highest classification), indicating that there is sufficient evidence of its carcinogenicity in humans. The IARC classified benzene as a human carcinogen in 1979. In 2012, the Working Group confirmed the previous findings of sufficient evidence of carcinogenicity in humans and experimental animals and, for the first time, presented strong evidence of multiple genotoxic effects based on a review of extensive mechanistic data. In humans, the Working Group concluded that benzene causes AML (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans 2012). It is a generally accepted practice in my field to rely on IARC classifications where available because IARC provides a robust and authoritative analysis of peer-reviewed scientific literature regarding potential carcinogens. In addition, I have independently reviewed the studies and data underlying the IARC Working Group's classification of benzene and agree with IARC's classification. In a separate report on general causation, which I have also included in my materials reviewed section and which I adopt as part of this report, I presented an overwhelming scientific data that proves the causal relationship between benzene exposure and hematological malignancies including ALL.

While my report speaks to the ingestion exposures of Mrs. Amsler to the Camp Lejeune water and to the contaminants (TCE, PCE, and benzene), it is known that these contaminants all fall within the category of chemicals known as volatile organic compounds (VOC). VOC evaporates at room temperature, and as a result, anytime someone ingests VOC, they are also inhaling them. It is my professional opinion, to a reasonable degree of medical/scientific certainty, that additional exposure to the water at Camp Lejeune and its VOC, via inhalation and/or dermal contact, would serve not only to increase the individual's cumulative exposure level to said VOC's, but would also serve to increase the individual's risk for developing Leukemia as a result of the additional exposures. In my review of Dr. Steven Bird's general causation report, he referenced the inhalation and dermal exposures to the VOC that individuals who lived and/or worked on base would likely have experienced. (General Causation report of Steven Bird pages 22-26). Notably, a 10-minute shower would equate to the exposure of ingesting 2 liters of contaminated water, contributing to the significant level of exposure through daily activities while living and working on the base (Bove et al. 2014). Physiologically based pharmacokinetic modeling confirms, at least generally, that daily living exposure to VOC like TCE, through inhalation and dermal routes, can approximate exposures to the same VOC through ingestion(Weisel and Jo 1996). Thus, the cumulative exposure values presented below underestimate the true exposure. In my opinion, the additional exposure that Mrs. Amsler would have experienced via inhalation and dermal routes due to showering, ingestion and/or swimming, would therefore significantly increase both the total exposure as well as the hematopoietic cancer risk, to a reasonable degree of medical/scientific probability.

According to Dr. Maslia's report, Hadnot Point water treatment plant (HPWTP) was delivering water to the Stone Street school she attended and residences in the area of Saint

Mary's Drive, where Mrs. Amsler lived, until 1972. Mrs. Amsler initially resided off-base from October 1965 until May 1965, but they would often visit the base, dine at the base, and swim in the pool. She then moved to Paradise Point with her father where she lived from May 1965 until June 1967. Therefore, her school and home would have received HPWTP water during her entire time there. According to Dr. Maslia's analysis the range of concentration of TCE, PCE and benzene at the time Mrs. Amsler resided on-base were the following:

HPWTP (October 1965- June 1967) (Appendix A7)

a. TCE: 19-30 ug/L
b. PCE: 0 ug/L
c. Benzene: 1 ug/L

According to Dr. Reynolds' report, Mrs. Amsler's cumulative exposure to TCE was 496 ug/L-months and to benzene was 20 ug/L-months.

Regarding TCE and PCE exposure, Cohn and colleagues investigated the association between exposure to trichloroethylene (TCE) and perchloroethylene (PCE) in drinking water and the incidence of leukemia and non-Hodgkin's lymphoma (NHL) in the northern New Jersey study area. The study indicated that exposure to TCE levels exceeding 5 µg/L was associated with elevated risks of ALL among females, the relative risk (RR) for exposure to TCE >5 µg/L was 2.36 (95% CI: 1.03–5.45). The association with high PCE exposure (>5 µg/L) with NHL was also observed with RR of 2.74 (95% CI: 1.20–6.26). The highest exposure category for TCE was >5 µg/L (population-weighted average: 23.4 µg/L), with the maximum recorded level being 67 µg/L. For PCE, the highest exposure category was also >5 µg/L (population-weighted average: 7.7 µg/L), with a maximum recorded level of 14 µg/L.

Mrs. Amsler's exposure to TCE (~496 µg/L) was significantly higher than the levels in the Cohn study for TCE. TCE at 50 µg/L is well above the EPA's maximum contaminant level of 5 µg/L, which the study used as a threshold for increased health risks. Based on the study findings, individuals exposed to such high levels of TCE for two years could have a substantially elevated risk of ALL. PCE at 2 µg/L, while within lower exposure categories, may contribute to risk in combination with TCE exposure, as the two compounds share toxic metabolic pathways and potential synergistic effects. The relative risks calculated in the study would likely be higher for these exposure levels. Thus, it is highly likely that Mrs. Amsler exposure to water contaminant with TCE increased her risk of ALL.

The genotoxic effects of benzene are primarily mediated predominantly by its metabolites, produced by CYP2E1 (as described above). Most recently, studies using biomarkers of benzene toxicity demonstrated that albumin adducts (an indicator of toxicity) can be introduced with an ambient benzene concentration of much less than 1ppm and there is no "minimum threshold" to harm . Rappaport et al. 2002; 2005). Moreover, the same study reported that the human CYP2E1 system becomes saturated at a benzene exposure of 1ppm resulting in a supralinear exposure-response curve. In other words, the exposure-response relationship is steepest at the lowest exposure levels. This indicates that the actual risks of hematologic cancers after low-level benzene exposure can be substantially higher than initially predicted.

Importantly, the production of toxic metabolites is directly related to the activity of CYPE1 in humans. Rappaport and colleagues reported that the metabolism of benzene decreases with age at a rate close to 2% per year (Rappaport et al. 2002). In other words, the level of toxic metabolites is expected to be higher in younger compared to older individuals exposed to the same levels of benzene. These findings can be further supported by some epidemiological studies that demonstrated an increase in MDS/AML particularly in younger patients before age 30 years (Linet et al. 2019). Mrs. Amsler was exposed to benzene at the age of 5 years after suggesting that the effect of benzene exposure may have been more pronounced compared to older individuals exposed to the same level of benzene

In terms of benzene exposure, Mrs. Amsler's cumulative exposure was 20 ug/L (0.02 ppm) through contaminated drinking water. The significant heterogeneity in assessing cumulative benzene exposure complicates a precise and definitive assessment of the exposure-response curve. Additionally, most published studies have not evaluated continuous measures of benzene exposure. Several studies demonstrate an increased risk of AML at near-ambient benzene concentrations (Rushton et al. 2014; Glass et al. 2003; 2006; Smith 2010; Shallis et al. 2021). Some flawed conclusions of a "safe" benzene exposure threshold came from the studies that demonstrated a significant increase in AML risk when compared to the "control" population below an arbitrary exposure level. This is particularly true for older studies where the "control" population may have been dexposed to more than ambient levels of benzene. This would result in a significant underestimation of the exposure effect on the incidence of AML.

Based upon my differential etiology analysis and to a reasonable degree of medical certainty, Mrs. Amsler's exposure to levels of TCE above those found to be associated with a significantly increase risk of leukemia in the Cohn study, along with her exposure to benzene are more likely than not the cause of her ALL.

Damages Analysis

ALL itself as well as chemotherapy and alloHCT used to treat Mr. Amsler's ALL are associated with significant clinical complications that can impact short- and long-term outcomes. One of the complications of ALL, particularly with very high white blood cell count, as in Mrs. Amsler's case may lead to organ dysfunction due to leukemia infiltrates. One of such manifestations is leukemic retinopathy leading to visual changes and occasional vision loss. Mrs. Amsler did develop leukemic retinopathy and fortunately it was resolved after initiation of chemotherapy. Another complication of ALL is coagulopathy that may present either as bleeding or excessive clotting. In Mrs. Amsler's case, she developed lower extremity deep vein thrombosis provoked by ALL which required prolonged use of anticoagulation (she has been treated with rivaroxaban). Long-term use of anticoagulants such as rivaroxaban carries risks of increased bleeding, including gastrointestinal and intracranial hemorrhages that may be life-threatening.

Infections are a major concern in ALL and during treatment with HyperCVAD, owing to immunosuppression from both the leukemia and its therapy. Patients are at high risk for bacterial infections (e.g., gram-negative bacteremia), viral reactivations (e.g., herpes simplex virus and

cytomegalovirus), and invasive fungal infections such as aspergillosis. These infections not only increase morbidity but can also be fatal without prompt treatment (Tomblyn et al., 2009).

Organ toxicities are frequent during HyperCVAD therapy. Doxorubicin, a core component of the regimen, is associated with dose-dependent cardiotoxicity, manifesting as arrhythmias, myocarditis, or long-term cardiomyopathy. Mrs. Amsler did develop atrial fibrillation after her cycle B of chemotherapy requiring antiarrhythmic therapy. She is also at risk for heart failure in the future.

Endocrinopathies and musculoskeletal complications are additional concerns, particularly with long-term steroid use. Corticosteroids can lead to osteoporosis, fractures, and avascular necrosis (Velentza, Zaman, and Sävendahl 2021). Mrs. Amsler had been diagnosed with osteoporosis and the use of steroids as a part of her chemotherapy likely exacerbated her bone loss. This, she is at risk for osteoporosis-related complications including an increased risk of fractures, particularly in the hip, spine, and wrist, leading to chronic pain, deformities such as kyphosis, reduced mobility, and loss of independence; additional risks include surgical and post-surgical complications following fractures, secondary issues like pressure ulcers or venous thromboembolism due to immobility, and significant psychosocial impacts such as isolation, anxiety, and depression.

AlloHCT is a potentially curative therapy for ALL, however, it is associated with significant early and late complications that profoundly impact the quality of life and long-term survival of transplant recipients.

One of the most common early complications of alloHCT is GVHD. Acute GVHD typically occurs within the first 100 days of post-transplant and primarily affects the skin, liver, and gastrointestinal tract. The severity of acute GVHD is influenced by factors such as HLA mismatch, the type of donor, and the efficacy of prophylactic regimens. Severe cases can lead to organ dysfunction, debilitating symptoms like severe diarrhea or jaundice, and significantly increased mortality rates (Ferrara et al. 2009). Mrs. Amsler developed Grade 2 skin GVHD that fortunately resolved with topical steroids and systemic immunosuppression with tacrolimus.

Another critical early complication is the heightened risk of infections due to profound immunosuppression. Bacterial, viral, and fungal infections are particularly prevalent, with pathogens like cytomegalovirus (CMV), Aspergillus, and gram-negative bacteria commonly implicated. CMV is a virus that stays in the body for life once someone is infected. It usually doesn't cause problems unless the immune system is weak, like after HCT. When a person who has had CMV before (seropositive) gets a transplant from a donor who has never had CMV (seronegative), such as in Mrs. Amsler's case, they are at risk of the virus waking up (reactivating). This happens because the donor's immune cells, which replace the recipient's own cells, don't know how to fight CMV. The risk of reactivation is highest in the first 3 to 6 months after the transplant, while the new immune system is still getting stronger. If their immune system stays weak longer—because of complications like infections or treatment side effects—they can remain at risk for a year or more. Thus, Mrs. Amsler had to take anti CMV therapy for 3 months post-alloHCT to prevent CMV reactivation. However, CMV reactivation may still occur late after transplant particularly in patients with a weakened immune system, and lead to serious

complications. Direct complications include tissue-invasive diseases such as pneumonitis (injury to the lung), which can cause respiratory failure; gastrointestinal disease, leading to ulcers, bleeding, pain, and diarrhea; hepatitis; retinitis, which can result in vision loss; encephalitis, causing neurological deficits; and, rarely, pancreatitis. Severe infections can cause systemic inflammation and organ dysfunction, potentially leading to multiorgan failure. Overall, CMV reactivation is linked to increased morbidity and mortality, highlighting the importance of early detection and prompt antiviral therapy to prevent or mitigate these complications.

Chronic GVHD is a common late complication that develops after the first 100 days and can involve multiple organs, including the skin, liver, eyes, and lungs. It often mimics autoimmune disorders leading to chronic pain, fatigue, and substantial organ dysfunction. Chronic GVHD not only impacts the patient's quality of life but also necessitates prolonged immunosuppressive therapy, which increases the risk of secondary infections (Lee et al., 2003). Mrs. Amsler developed dry eye syndrome which is likely permanent and will require life-long treatment.

Secondary malignancies are another significant late complication, with an increased risk attributed to prior chemotherapy, radiation, and immune dysregulation. Common secondary cancers include skin cancers and hematologic malignancies, which contribute to late mortality among long-term survivors (Danylesko and Shimoni 2018). Persistent immune deficiency also predisposes patients to recurrent infections and chronic viral reactivations, such as CMV or Epstein-Barr virus, which can lead to life-threatening complications (Foord et al. 2020).

Endocrine dysfunctions, including hypothyroidism, infertility, and adrenal insufficiency, are long-term consequences of the conditioning regimens used in alloHCT. Cardiovascular complications, including hypertension, dyslipidemia, and cardiomyopathy, also emerge over time due to chronic inflammation and prior exposure to chemotherapeutic agents (Armenian and Chow 2014).

Psychological and cognitive effects further compound the challenges faced by transplant survivors. Depression, anxiety, and post-traumatic stress disorder (PTSD) are prevalent and often result from the prolonged stress of treatment and recovery. Cognitive impairments, likely due to the neurotoxic effects of therapies and the burden of chronic illness, negatively impact the patient's ability to return to normal life, including employment and social engagement.

In conclusion, while alloHCT offers a potential cure for many life-threatening conditions, the associated early and late complications necessitate monitoring and often tailored interventions to improve long-term outcomes and quality of life for recipients.

Conclusions

ALL is a rare blood cancer arising from a single hematopoietic stem cell. Certain environmental factors such as ionizing radiation, chemotherapy and genotoxic chemicals such as benzene and TCE play an important role in the pathogenesis of this disease. Some cases of acute leukemias may arise as a result of inherited predisposition syndromes and several genes have been implicated in this process. Benzene is a potent carcinogen and multiple in vitro and in vivo

data in laboratory animals and humans demonstrated the significant DNA-damaging activity of this agent, predominantly in blood cells. This may be partially because bone marrow, in addition to the liver and lungs, is an active site of benzene metabolism and certain enzymes responsible for the production of genotoxic byproducts are highly expressed in hematopoietic cells (e.g. myeloperoxidase, MPO). In addition to mechanistic studies demonstrating the mutagenic role of benzene, numerous epidemiological studies demonstrated a significant association between benzene exposure and hematologic malignancies including AML and ALL. Given this overwhelming body of evidence, benzene was classified as a Group I carcinogen by IARC and the causative agent of myeloid leukemia. Thus, the association between benzene exposure and blood cancer, including ALL, is undeniable.

Trichloroethylene (TCE) is a volatile organic compound that is known to have significant genotoxic potential and has been implicated in the pathogenesis of various malignancies, including hematologic cancers. The evidence from both *in vivo* and *in vitro* tests, in mammalian and other experimental systems demonstrated that metabolites of TCE are highly genotoxic and lead to mutations, chromosomal aberrations, micronuclei, and cell transformation. Importantly, this type of damage was detected in peripheral blood lymphocytes of humans exposed to TCE metabolites. Thus, biological evidence of carcinogenic potential of these chemicals is indisputable. Moreover, studies in humans exposed to water contaminated with TCE, similar to Camp Lejeune exposure, demonstrated significantly increased incidence of leukemia at levels of exposure as low as 0.1-5 ug/L. For comparison, Mrs. Amsler was exposed to approximately 496 ug/L of TCE.

Mrs. Amsler was exposed to the levels of TCE which have been shown to result in a significant increase in the risk of leukemia as well as above background levels of benzene through contaminated water at Camp Lejeune. Such cumulative exposure would be considered biologically significant resulting in an increased rate of DNA damage and, consequently, the elevated risk of ALL. Moreover, benzene and TCE are both known to be genotoxic and immunotoxic; thus, simultaneous exposure to these chemicals would likely result in at least an additive effect, leading to a higher risk of leukemia compared to exposure to either toxin alone.

Mrs. Amsler was diagnosed with ALL in 2020 at the age of 60 years. Even though some family members were diagnosed with solid tumors, none of her relatives has been diagnosed with hematological malignancy, thus, it is highly unlikely that her cancer was a consequence of hereditary predisposition. Moreover, molecular underpinning of her leukemia, namely 11q23 rearrangement, has been associated with exposure to genotoxic therapies and environmental exposure to DNA damaging agents such as benzene.

The detailed review of her medical records did not identify any underlying medical conditions, no obesity (BMI ~23) or history of radiation or chemotherapy prior to her diagnosis of ALL that could have potentially contributed to Mrs. Amsler's cancer. Through her employment, Mrs. Amsler was exposed to biological materials such as bacteria, viruses, parasites and fungi that can pose a threat to human health when they are inhaled, eaten or come in contact with skin. While they can cause illness such as bacterial, fungal and viral infections, they are not genotoxic and did not contribute to Mrs. Amsler's ALL.

As a result of her diagnosis and applied therapies, Mrs. Amsler has suffered numerous adverse events including transient leukemic retinopathy, worsening bone loss, deep vein thrombosis requiring long-term anticoagulation, atrial fibrillation requiring antiarrhythmic medication, and dry eyes due to chronic GVHD. As a part of her diagnostic work-up, surveillance and therapy she underwent 6 bone marrow biopsies and 4 lumbar punctures with intrathecal chemotherapy administration and a central line placement. Even though Mrs. Amsler is currently in remission, she remains at risk for disease relapse and late post-alloHCT complications (discussed in detail in the "Damages Analysis" section) including as chronic GVHD, infections, organ dysfunction, secondary malignancies, worsening osteoporosis, cardiovascular complications which can collectively impact survival and quality of life.

Applying a differential etiology analysis and following the weight of the evidence approach to investigating and interpreting the data I conclude to a reasonable degree of medical certainty that Mrs. Amsler's exposure to TCE, and benzene through contaminated water at Camp Lejeune was more likely than not causative of her ALL, and that as a result, she suffered numerous complications and is at risk for further complications outlined in this report, in the future. Based upon my education, training and experience, it takes approximately 2 years after alloHCT for the full reconstitution of the immune system. As a result, it would be advised to avoid exposure to bacterial and viral pathogens during this period. Given the nature of her work as a microbiologist it would be prudent not to work with infectious material for at least two years after her transplant.

Even though Mrs. Amsler is currently 4 years post-alloHCT her life expectancy is shorter compared to the general population. The cumulative incidence of relapse for patients with ALL 4 years post-transplantation depends on several factors including disease risk, age at transplant, and the severity of chronic GVHD. Based on the analysis of the large cohort of alloHCT recipients the cumulative incidence of relapse at 10 years post-transplantation is approximately 9% for ALL survivors (Wingard et al. 2011). This suggests that by the 4-year mark, the relapse risk is lower, with the majority of relapses occurring earlier within the first few years post-HCT but the risk of disease relapse remains elevated. Based on the same research, the life expectancy for a 4-year survivor of ALL post-HCT remains lower than that of the general population. The study reported an 85% survival probability at 10 years for 2-year survivors of ALL post-HCT. Survivors of ALL post-HCT may experience a life expectancy reduction of 10-20 years**,** depending on factors such as age at transplant, GVHD status, and post-transplant complications. For 5-year survivors without chronic GVHD or relapse, life expectancy approaches normal over time, but the relative mortality risk remains elevated compared to the general population.

All my opinions and conclusions are held to a reasonable degree of medical certainty. I reserve the right to modify or supplement this report if additional information becomes available.

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The Johns Hopkins University School of Medicine

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Case Materials Received

Legal Documents/Materials:

- Amsler Discovery Pool Profile Form
- Amsler Short Form Complaint
- Defendant United States of America's Frist Supplemental Response to Plaintiffs' Leadership Group's First Set of Interrogatories to Defendant United States of America Concerning Track 1 Discovery Pool Plaintiffs

Depositions:

- Videotaped Deposition of Karen Marie Amsler, dated 16 April 2024, with exhibits
- Videotaped & Videoconference Deposition of Dr. Frank J. Bove, vol. 1, taken 17 October 2024
- Videotaped & Videoconference Deposition of Dr. Frank J. Bove, vol. 2, taken 18 October 2024

Other:

- **Water Modeling Table:**
  - Amsler, Karen - Reconstructed HPWTP Concentrations_ATSDR_Chapter A Report_Camp Lejeune

Medical Documents/Materials:

- Amsler, Karen_Bookmarked Medical Records UPDATED 5-15-2024
- 000000_00284_AMSLER_0000010300
- 000000_00284_AMSLER_AON_0000000001
- 000000_00284_AMSLER_MEDRECS_0000000002
- 000000_00284_AMSLER_MUSC_0000000001
- 000001_00284_AMSLER_0000010492
- 000001_00284_AMSLER_MEDRECS_0000002189
- 000002_00284_AMSLER_0000010892

Exhibit 1

CURRICULUM VITAE
The Johns Hopkins University School of Medicine

Lukasz P. Gondek, M.D., Ph.D. 10/23/2024

DEMOGRAPHIC AND PERSONAL INFORMATION

Current Appointments

University

2023 – present Associate Professor of Oncology, The Johns Hopkins University School of Medicine

Hospital

2014 – present Attending Physician, The Johns Hopkins Hospital

Education and Training

Undergraduate

European School system

Doctoral/graduate

2003	MD/Medical University of Silesia, Poland	Medicine
2013	PhD/University of Warsaw, Poland	Hematology

Postdoctoral

2003-2004	Intern, Medicine, VA Hospital, Katowice, Poland
2004-2007	Postdoctoral Fellow, Hematology, Cleveland Clinic
2008-2011	Resident, Internal Medicine, Cleveland Clinic
2011-2014	Fellowship, Hematology, Johns Hopkins

Professional Experience

2007-2008	Research Associate, Cleveland Clinic
2014-2016	Instructor of Oncology, Johns Hopkins
2016-2023	Assistant Professor of Oncology, Johns Hopkins

PUBLICATIONS:

Original Research [OR]

1. Beck RC, Wlodarski M, **Gondek L**, Theil KS, Tuthill RJ, Sobeck R, Bolwell B, Maciejewski JP. Efficient identification of T-cell clones associated with graft-versus-host disease in target tissue allows for subsequent detection in peripheral blood. *Br J Haematol.* 2005;129(3):411-419. *Performed experiments.*
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42. Karantanos T, Teodorescu P, Perkins B, Christodoulou I, Esteb C, Varadhan R, Helmenstine E, Rajkhowa T, Paun BC, Bonifant C, Dalton WB, **Gondek LP**, Moliterno AR, Levis MJ, Ghiaur G, Jones RJ. The role of the atypical chemokine receptor CCRL2 in myelodysplastic syndrome and secondary acute myeloid leukemia. Sci Adv. 2022 Feb 18;8(7); *Analyzed data, wrote the manuscript*
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59. Saadatagah S, Uddin MM, Weeks LD, Niroula A, Ru M, Takahashi K, **Gondek L**, Yu B, Bick AG, Ebert BL, Platz EA, Natarajan P, Ballantyne CM. Clonal Hematopoiesis Risk Score and All-Cause and Cardiovascular Mortality in Older Adults. JAMA Netw Open. 2024 Jan 2;7(1):e2351927. doi: 10.1001/jamanetworkopen.2023.51927. *Analyzed data, wrote the manuscript*
60. Imus PH, Pasca S, Tsai HL, Aljawai Y, Cooke KR, Walston JD, Gocke CD, Varadhan R, Jones RJ, **Gondek LP**. Recipient clonal hematopoiesis in allogeneic bone marrow transplantation for lymphoid malignancies. Blood Adv. 2024 Apr 19:bloodadvances.2023011761. doi: 10.1182/bloodadvances.2023011761. *Senior author*
61. Marshall CH, **Gondek LP**, Daniels V, Lu C, Pasca S, Xie J, Markowski MC, Paller CJ, Sena LA, Denmeade SR, Luo J, Antonarakis ES. Association of PARP inhibitor treatment on the prevalence and progression of clonal hematopoiesis in patients with advanced prostate cancer. Prostate. 2024 Jul;84(10):954-958. doi: 10.1002/pros.24712. Epub 2024 Apr 20.. PMID: 38641986. *Designed the project, performed experiments, analyzed data, wrote the manuscript*
62. Hong YS, Pasca S, Shi W, Puiu D, Lake NJ, Lek M, Ru M, Grove M, Prizment A, Joshu CE, Platz EA, Guallar E, Arking DE, Gondek LP. Mitochondrial heteroplasmy improves risk prediction for myeloid neoplasms. medRxiv doi: https://doi.org/10.1101/2024.04.07.24305454. *Senior author*
63. Jitaru C, Peters MC, Aggarwal L, Bancos A, Tigu AB, Cenariu D, Selicean C, Pasca S, Moisoiu V, Rotariu P, Santa M, Iluta S, Drula R, Kegyes D, Kurtus A, Zdrenghea M, **Gondek L**, Tomuleasa C, Ghiaur G. Single low-dose decitabine as frontline therapy of acute myeloid leukaemia, with venetoclax salvage. J Cell Mol Med. 2024 Oct;28(20):e18592. doi: 10.1111/jcmm.18592. PMID: 39435884 *Designed the project, analyzed data, wrote the manuscript*

Review Articles [RA]

1. **Gondek LP**, Spivak J. Somatic mutations in polycythaemia vera and other Philadelphia chromosome negative myeloproliferative neoplasms. John Wiley & Sons, Ltd. 2012 Dec;
2. **Gondek LP**, DeZern AE., I walk the line: how to tell MDS from other bone marrow failure conditions. Curr Hematol Malig Rep. 2014 Dec;9(4):389-399
3. **Gondek LP**, DeZern AE. Assessing clonal haematopoiesis: clinical burdens and benefits of diagnosing myelodysplastic syndrome precursor states. Lancet Haematol. 2019 Dec 3.
4. DeZern AE, **Gondek LP**. Stem cell donors should be screened for CHIP. Blood Adv. Feb 25;4(4):784-788

5. Pasca S, **Gondek LP**. Clonal hematopoiesis and bone marrow failure syndromes. Best Pract Res Clin Haematol. 2021 Jun;34(2):101273.
6. **Gondek LP**. CHIP: is clonal hematopoiesis a surrogate for aging and other disease? Hematology Am Soc Hematol Educ Program. 2021 Dec 10;2021(1):384-389. doi: 10.1182/hematology.2021000270
7. **Gondek LP**, Sheehan VA, Fitzhugh CD. Clonal Hematopoiesis and the Risk of Hematologic Malignancies after Curative Therapies for Sickle Cell Disease. J Clin Med 2022 Jun 2;11(11):3160.
8. Marshall CH, **Gondek LP**, Luo J, Antonarakis ES. Clonal hematopoiesis of indeterminate potential in patients with solid tumor malignancies. Cancer Res. 2022 Aug 30:CAN-22-0985.
9. Ktena YP, Dionysiou M, **Gondek LP**, Cooke KR. The impact of epigenetic modifications on allogeneic hematopoietic stem cell transplantation. Front Immunol. 2023 May 31;14:1188853. doi: 10.3389/fimmu.2023.1188853. eCollection 2023.
10. Gibson CJ, Lindsley RC, **Gondek LP**. Clonal hematopoiesis in the setting of hematopoietic cell transplantation. Semin Hematol. 2024 Feb 1:S0037-1963(24)00012-X. doi: 10.1053/j.seminhematol.2024.01.011.

Book Chapters, Monographs [BC]

1. Maciejewski, JP, **Gondek, LP**, Selleri, C, & Risitano, AM (2010). Molecular Diagnostics in Hematology. In G.P. Rodgers, N.S. Young (Ed.), *The Bethesda Handbook of Clinical Hematology*. Philadelphia, PA. Wolters Kluwer Health/Lippincott William & Wilkins.
2. **Gondek LP**, Ghiaur G (2017) micro-RNAs: Network in Acute Leukemia. In Emadi, A, Karp, JE (Ed.), *Acute Leukemia: An Illustrated Guide to Diagnosis and Treatment*. New York: Demos
3. **Gondek LP** (2018). Epigenetic Modulators. In Emadi, A, Karp, JE (Ed). *Illustrative Oncopharmacology*.
4. Pasca S, **Gondek LP**(2023). Epigenetic Modulators. In Emadi, A, Karp, JE (Ed). *Cancer Pharmacology: An Illustrated Manual of Anticancer Drugs, Second Edition*

Editorials [ED]

1. **Gondek LP**. Hitting the bullseye with a nonlethal payload: resistance in CD123-positive malignancies. *J Clin Invest. 2019 Oct 14*.
2. **Gondek LP.** High Prevalence of Clonal Hematopoiesis in the Blood and Bone Marrow of Healthy Volunteers. *PracticeUpdate 2020*
3. **Gondek LP.** Donor Clonal Hematopoiesis and Outcomes After Transplantation. *PracticeUpdate 2021*
4. **Gondek LP.** Refining CHIP in population datasets. *Blood*. 2023 May 4;141(18):2163-2164. doi: 10.1182/blood.2023019801..

Media Release of Interviews [MR]

1. https://www.youtube.com/watch?v=pceG4k4bBxo
2. https://www.youtube.com/watch?v=4LlYYKwZXCE

FUNDING

EXTRAMURAL FUNDING

Research Extramural Funding

Current

1/15/21 – 1/31/26	The Biological Consequences of Age-related Clonal Hematopoiesis R01 HL156144-01A1 NIH/NHLBI $2,000,005 Role: PI, 30% effort
2/1/23 – 1/31/26	Identifying, Understanding, and Eradicating Measurable Residual Disease (MRD) in Patients with Acute Myeloid Leukemia (AML). Break Through Cancer $1,599,453 Role: co-PI, 5% effort
2/1/23 – 1/31/26	Targeting Clonal Hematopoiesis (CH) to Prevent Acute Myeloid Leukemia (AML). Break Through Cancer $1,877,429 Role: co-PI, 3% effort

Pending

07/01/24 – 06/30/29
Mitochondrial heteroplasmy and risk of myeloid malignancies
R01
NIH/NCI
$4,093,735.00
Role: PI, 20% effort

Previous

2006 – 2008
A Novel Approach for the Study of Genetic Predisposition in AA and PNH Using High-Density Arrays
Young Investigator Award/AA&MDS
$60,000
Role: PI, 50% effort

7/1/12 – 6/30/14
Self-renewal mechanisms in myeloid leukemias
5T32HL007525
NIH
$110,360
PI: Robert Brodsky
Role: Trainee, 80% effort

7/1/14 – 6/30/15
Hedgehog signaling in MDS progression
90056518
Aplastic Anemia & MDS International Foundation (AA&MDSIF)
$90,090
PI: Amy DeZern
Role: Project Leader, 20% effort

7/1/14 – 6/30/16
Edward P. Evan Fellowship
90056518
Aplastic Anemia & MDS International Foundation (AA&MDSIF)
$74,700
PI: Amy DeZern
Role: Project Leader, 35% effort

9/1/18 – 8/31/20
The prevalence and molecular characteristics of age-related clonal hematopoiesis in HIV-positive patients.
P30 Cancer Centers Support Grants
NIH/NCI
$133,335
Role: PI, 10% effort

7/15/18 – 5/31/21
Personalized molecular approaches to disease monitoring and maintenance therapies for Myelodysplastic Syndromes (MDS) and Acute Myeloid Leukemia (AML) patients undergoing allogeneic bone marrow transplantation.
R21 HL143096-01
NIH/NHLBI
$245,626
Role: PI, 10% effort

4/5/17 – 3/31/22
Hedgehog Signaling in the Progression of Myelodysplastic Syndromes
K08 HL136894-01
NIH/NHLBI
$856,600
Role: PI, 75% effort

INTRAMURAL FUNDING

Research Intramural Funding

Current

4/1/22 – 3/31/24
The role of clonal hematopoiesis in solid tumor malignancies: The Atherosclerosis Risk in Communities (ARIC) Study
Allegheny Health Network-Johns Hopkins Cancer Research Fund

	$200,000 Role: PI, 20% effort
Pending	None
Previous	
4/1/16 – 3/31/18	Age-related clonal hematopoiesis and the mechanism of leukemic transformation using allogeneic bone marrow transplantation model Hopkins-Allegheny Health Network (AHN) Cancer Research Fund $200,000 Role: PI, 10% effort
7/1/17 – 6/30/19	Clinician Scientist Award Johns Hopkins University, School of Medicine $80,000 Role: PI, 75% effort
6/1/19 – 5/31/20	The prevalence, molecular characteristics and clinical consequences of clonal hematopoiesis of indeterminate potential (CHIP) in HIV positive and HIV-negative men. Faculty Development Award, Johns Hopkins University Center for AIDS Research (JHU CFAR) Developmental Core. NIH/NIAID $50,000 Role: PI, 10% effort
1/1/20 – 12/31/22	The Pathogenesis, Prognosis and Treatment of Clonal Hematopoiesis and Myelodysplastic Syndromes Hematologic Malignancies and Bone Marrow Transplantation CCSG Program $150,000 Role: PI, 10% effort
1/1/20 – 12/31/22	Hematologic Malignancies and their Precursors in HIV: Applications of Advanced Molecular Techniques. Hematologic Malignancies and Bone Marrow Transplantation CCSG Program $150,000 Role: co-PI, 10% effort
1/1/21 – 12/31/22	Plasma-based minimal residual disease detection and allogeneic bone marrow transplantation outcome in patients with myeloid malignancies $50,000 Role: PI, 5% effort

CLINICAL ACTIVITIES

Clinical Focus

Care of patients with hematological malignancies on adult leukemia service.

Certification

Medical, other state/government licensure

2011 – present State of Maryland (D72150)

Boards, other specialty certification

2008 – present Advanced Cardiac Life Support
2011 – 2021 American Board of Internal Medicine
2013 – present American Board of Hematology

Clinical (Service) Responsibilities

2014 – present Hematology/Oncology - Leukemia 15% effort (8 weeks per year)
2014 – present Hematology/Oncology and Surgery – Bone marrow harvest 5% effort
2014 – present Hematology/Oncology and Pathology – Interpretation of bone marrow aspirations 5% effort

Clinical Productivity

2014 – present My targeted clinical effort assignment is 25%.

Total RVUs

FY15	FY16	FY17	FY18	FY19	FY20	FY21	FY22	FY23	FY24
1764	2104	1735	1792	1836	1628	1551	1470	1093	1829

Clinical Draw from outside local/regional area none
Membership in or examiner for specialty board none
Clinical Program Building / Leadership none

Clinical Demonstration Activities to external audience, on or off campus
2024 – present Clinical Program in Clonal Hematopoiesis and myeloid precursor states.

Development of nationally/internationally recognized clinical standard of care none

EDUCATIONAL ACTIVITIES

Educational Focus
My educational focus is on the biology, diagnostic approaches, and treatment of myeloid malignancies, in particular, Myelodysplastic Syndrome and Clonal Hematopoiesis.

Teaching

Classroom Instruction
JHMI/Regional
2015 – 2022 Instructor, medical students, Genes to Society – Hematology Course, Johns Hopkins SOM, Baltimore, MD
2024 – present Instructor, medical students, Genes to Society – Hematologic Malignancies Course, Johns Hopkins SOM, Baltimore, MD
2015 – present Instructor, medical students, Topics in Interdisciplinary Medicine - Introduction to Regenerative Medicine, Johns Hopkins SOM, Baltimore, MD
National None
International None

Clinical Instruction
JHMI/Regional
2014 – present Instructor, Internal Medicine residents and Medical Oncology fellows, Clinical skills instruction for on the Leukemia Inpatient Service, Johns Hopkins SKCCC, Johns Hopkins University, Baltimore, MD
National None
International None

CME Instruction
See Invited Talks.

Mentoring

Pre-doctoral Advisees /Mentees
2016 – 2019 Samantha Kegel; college student, Johns Hopkins; currently medical student at University of Maryland SOM
2020 – present Matthew Gao; medical student, Johns Hopkins; recipient of American Society of Hematology HONORS Award (2021)

Post-doctoral Advisees
2015 – 2018 Bonnie Lau, MD, PhD; hematology/oncology fellow, currently Assistant Professor at the Geisel School of Medicine, Dartmouth; co-authored articles OR 28, OR35
2016 – 2018 Rafael Madero-Marroquin, MD; postdoctoral fellow; currently hematology/oncology fellow at the University of Chicago; co-authored OR28, OR31, OR35, OR39
2016 – 2017 Federico De Marchi, MD; postdoctoral fellow; currently PhD student at Juntendo University Graduate School of Medicine, Tokyo, Japan; co-authored OR28, OR30, OR35
2018 – 2019 Lin Zhao, MD; visiting scholar; currently Chief Physician, Shanghai University of Traditional Chinese Medicine, China; co-authored OR31, OR39
2019 – 2022 Daniel Haldar, MD; internal medicine resident, John Hopkins; currently hematology/oncology fellow at Johns Hopkins University; recipient of Molina/Grasmick Scholar (2021); co-authored OR 56

2020 – present Sergiu Pasca, MD, PhD; postdoctoral fellow; recipient of the Romanian Society of Bone Marrow Transplantation Award; co-authored OR46, OR48, OR53, OR56, OR60, OR61, OR62, OR63, RA5
2021 – 2023 Michael Hochman, MD; postdoctoral hematology fellow, currently Assistant Professor at Department of Hematology and Medical Oncology, Emory University School of Medicine
2022 – present Jiajun Xie, MD; postdoctoral fellow. co-authored OR56, OR61.
2023 – present Anna Bereznicka, PhD; postdoctoral fellow
2024 – present Shirley Mo, MD; postdoctoral fellow

Thesis committees
None

Educational Program Building / Leadership None
Institutional Administrative Appointments None
Educational Demonstration Activities to external audiences None

RESEARCH ACTIVITIES

Research Focus
I am an Associate Professor of Oncology in the Division of Hematologic Malignancies, Leukemia Program at the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins. My career goal is to develop novel insights into the pathogenesis of myelodysplastic syndromes (MDS) and acute myeloid leukemia (AML) and translate these findings into new treatments for patients with these diseases. My research focuses on the genetic mechanisms responsible for MDS development and events leading to disease progression and leukemic transformation. We have recently utilized the allogeneic bone marrow transplantation platform to study the natural history of clonal hematopoiesis of indeterminate potential (CHIP) and the mechanism leading to clonal evolution and expansion. The experience in AML and MDS genetics will allow me to functionally characterize the genetic alteration identified in patients during disease development and progression. Since my appointment as an Assistant Professor, my laboratory research has been continuously founded by the National Institute of Health (K08, R21, R01) as well as several foundation grants. I envision my laboratory and clinical effort the centerpiece of the translational research program in MDS and clonal hematopoiesis.

Research Program Building / Leadership
2024 – present Clonal Hematopoiesis clinical and research program.

Research Demonstration Activities None
Inventions, Patents, Copyrights None
Technology Transfer Activities None

SYSTEM INNOVATION AND QUALITY IMPROVEMENT ACTIVITIES

None

ORGANIZATIONAL ACTIVITIES

Institutional Administrative Appointments
2013 – 2014 Member, Hematology/Oncology Fellowship- Tracks Committee
2016 – present Member, Medical School Admissions Committee, Johns Hopkins School of Medicine
2017 – present Member, Internal Medicine Residency Admissions Committee, Johns Hopkins School of Medicine

Editorial Activities
Editorial Board Appointments None

Journal peer review activities
2011 – present Reviewer for The British Journal of Haematology
2014 – present Reviewer for Case Reports in Hematology
2017 – present Reviewer for Journal of Clinical Investigation
2017 – present Reviewer for Biology of Blood and Marrow Transplantation
2018 – present Reviewer for Haematologica
2019 – present Reviewer for Clinical Cancer Research

2019 – present Reviewer for Blood Advances
2019 – present Reviewer for Blood
2020 – present Reviewer for American Journal of Transplantation
2021 – present Reviewer for Lancet Haematology
2021 – present Reviewer for Frontiers in Oncology
2022 – present Reviewer for Circulation
2023 – present Reviewer for Journal of Clinical Oncology
2024 – present Reviewer for American Journal of Hematology

Other peer review activities

2019 American Society of Hematology, Abstract Reviewer, Clonal Hematopoiesis: Aging and Inflammation.
2020 American Society of Hematology, Coordinating Abstract Reviewer, Clonal Hematopoiesis: Aging and Inflammation.

Advisory Committees, Review Groups/Study Sections

2020 The European Hematology Association, Grant Reviewer
2021 Stichting Kinderen Kankervrij' (Foundation Children Cancerfree) or 'KiKa' Foundation, Netherlands, Grant Reviewer
2022 Special Emphasis Panel for R21 Study Section, NIH/NHLBI, ad hoc Grant Reviewer
2023 Biology of Blood, Heart and Vasculature Study Section, NIH, ad hoc Grant Reviewer
2023 Special Emphasis Panel for R21 Study Section, NIH/NHLBI, ad hoc Grant Reviewer
2023-2024 Break Through Cancer- AML Measurable Residual Disease Steering Committee
2024 Hemostasis, Thrombosis, Blood Cells and Transfusion Study Section, NIH, ad hoc Grant Reviewer
2024 MPN Foundation, 2024 MPN Challenge Grant Reviewer

Professional Societies

2011 – present Member, American Society of Hematology
2011 – present Member, American Society of Clinical Oncology

Conference Organizer

None

Session Chair

JHMI/Regional None
National
12/2019 Session Chair, American Society of Hematology Annual Meeting, Clonal Hematopoiesis: Aging and Inflammation. Orlando, FL
12/2020 Session Chair, American Society of Hematology Annual Meeting, Clonal Hematopoiesis: Aging and Inflammation. Virtual event
12/2021 Session Chair, American Society of Hematology Annual Meeting, ASH Education Program, Clonal Hematopoiesis. Atlanta, GA
International None

Consultantships

2018 – present GLG consulting, medical consulting for GLG clients/ consultant
2020 – present VeraMedica LLC., medical/oncology consulting for legal cases/ expert witness
2021 – present Bristol Myers Squibb, MDS advisory board/ member
2022 – present Bluebird Bio, cell therapy advisory board/ member

RECOGNITION

Awards, Honors

1997 – 2003 Full academic scholarship, Medical University of Silesia

2005, 2006, 2007 Travel Award, American Society of Hematology
2014 – 2016 Edward P. Evans Fellowship, AA & MDS International Foundation
2017 – 2019 Clinician Scientist Award, Johns Hopkins School of Medicine

2019 – 2020 Faculty Development Award, Johns Hopkins University Center for AIDS Research (JHU CFAR)

Invited Talks

JHMI/Regional

5/26/15 Genetics and Society, Clinical application of genetics research, Annual Science Outreach Event, Project Bridge, Baltimore, MD.2/2016 Myelodysplastic Syndromes: From Metaphase Karyotyping to Molecular DNA Profiling, JHMI Division of Hematology, Hematology Grand Rounds, Baltimore, MD

8/17/16 Molecular Profiling in MDS, Assistant Professor Summer Lecture Series, SKCCC, Baltimore, MD

8/7/19 The Clinical Consequence of Age-related Clonal Hematopoiesis, Assistant Professor Summer Lecture Series, SKCCC, Baltimore, MD

5/27/20 Clonal Hematopoiesis: Biology and Clinical Consequences, Translational Research Conference, SKCCC, Baltimore, MD

1/15/21 Clinical Consequences of Age-related Clonal Hematopoiesis, JHH Medical Grand Rounds, Baltimore, MD

2/2/21 Age-related Clonal Hematopoiesis – Biology and Clinical Consequences, Biology of Healthy Aging Lecture Series, Baltimore, MD

5/7/21 Clonal Hematopoiesis in Cancer and Beyond, JHU SKCCC Oncology Grand Rounds, Baltimore, MD

3/29/22 Clonal Hematopoiesis in Aging and Cancer, JHU SKCCC Novel Approaches to Therapy and Prevention Course, Baltimore, MD

4/22/22 Clinical Consequences of Clonal Hematopoiesis, JHU SKCC Sibley Memorial Hospital, Grand Rounds, Washington DC.

11/02/23 Fundamentals of hematologic malignancies/stem cells/clonal evolution, JHU SKCCC Fundamentals of Cancer, Cause to Cure Course

National

12/6/19 Clinical Management of Myeloid Malignancies: The Coming of Age of Targeted Therapies, American Society of Hematology, Friday Scientific Symposia, Orlando, FL

3/2/21 Age-related clonal hematopoiesis – biology and clinical consequences. Norris Cotton Cancer Center, Dartmouth, Oncology Grand Rounds, Lebanon, NH

10/3/21 Impact of Clonal Hematopoiesis on Hematopoietic Cell Transplantation Outcome. Global Cure for SCD Virtual Conference.

11/20/21 Rationale for screening stem cell donors for clonal hematopoiesis. Association for Molecular Pathology Meeting, Philadelphia, PA.

12/12/21 ARCH: Is Clonal Hematopoiesis a Surrogate for Age and Other Disease? American Society of Hematology, ASH Education Program, Atlanta, GA

3/24/22 Clonal hematopoiesis and its implications for hematopoietic stem cell transplantation. Cellular and Molecular Therapeutics Branch and the Sickle Cell Branch at the NHLBI Scientific Meeting, Bethesda, MD

5/4/22 Clonal Hematopoiesis in Cancer and Beyond. Herbert Irving CCC, Columbia University, Hematology/Oncology Grand Rounds, New York, NY

6/10/22 Allogenic Transplant and Clonal Hematopoiesis in Sickle Cell Disease. 5th Annual Sickle Cell Disease Access to Care Summit, Fort Lauderdale, FL.

12/9/22 The Biological Consequences of Age-Related Clonal Hematopoiesis. Scientific Workshop on Hematology and Aging, American Society of Hematology, New Orleans, LA.

2/9/23 Clonal hematopoiesis and hematopoietic stem cell transplant outcomes. Division of Hematology & Oncology Cutter Lecture Series, Vanderbilt University, Nashville, TN

10/11/23 Post-curative Malignancies Risk. 16th Annual Sickle Cell in Focus Conference, NIH, Bethesda, MD.

03/13/24 Clonal hematopoiesis and the risk of myeloid neoplasms. Molecular Therapeutics Lecture, Karmanos Cancer Institute, Detroit, MI

03/19/24 Mitochondrial heteroplasmy and the risk of myeloid malignancies. Translational Hematology & Oncology Research Lecture Series, Cleveland Clinic Cancer Center, Cleveland, OH.

06/21/24 Improving outcome of patients after allogeneic hematopoietic cell transplantation, Oncology Grand Rounds, Case Western Reserve University, Cleveland, OH.

08/16/24 Novel NGS approaches to single-cell analysis and measurable residual disease detection, Food and Drug Administration, Bethesda, MD.

10/2/24 Improving outcome of patients after allogeneic hematopoietic cell transplantation, Translation Research Conference, University of Pennsylvania, Philadelphia, PA.

International

10/30/15	MDS: Clinical Application of Molecular Techniques, IV Ibero-American Symposium on Myelodysplastic Syndromes, Puerto-Vallarta, Mexico
10/04/18	MDS: Clinical Application of Molecular Techniques, V Ibero-American Symposium on Myelodysplastic Syndromes, Cancun, Mexico
10/11/18	Molecular testing in myeloid diseases – diagnostic and therapeutic implications, XXV Romanian Society of Hematology National Meeting, Sinaia, Romania
9/17/22	Donor Clonal Hematopoiesis and HSC Recipient Outcomes, Insights in Hematology, 7th Edition, Cluj, Romania.
10/28/22	Keynote Speaker, Diagnostic and clinical implications of clonal hematopoiesis, Laboratory Medicine Congress & Exhibition & KSLM 63rd Annual Meeting (LMCE 2022), Seoul, South Korea
1/4/23	Clonal Hematopoiesis, Post-New Orleans ASH 2022: Novità dal Meeting della Società Americana di Ematologia, Milan, Italy.

Exhibit 2

Fee Schedule
The VeraMedica Institute, LLC

LUCASZ GONDEK, MD, PHD.
Instructor of Oncology, The Johns Hopkins University School of Medicine.
Assistant Professor of Oncology, The Johns Hopkins University School of Medicine.
Attending Physician, The Johns Hopkins Hospital.

Baseline billable rate ……. $600.00 per hour

Travel time…….....……. $600.00 per hour

Travel Policy:

> ***Travel Policy:*** The client agrees to compensation for minimum commitment equivalent to a half day of the doctor's time when the client schedules the physician to travel more than 60 miles from home or office, for a meeting.

Deposition (Minimum 4 hours) and Trial (Minimum 8 hours) …….. $900.00 per hour

****Please note for projects requiring expedited service due to late submission of materials or announcement of deadlines that will require Expert's adjusting of schedules and staff overtime within three weeks of deadline dates will thus require a rate premium increase of 50% across the board. Thank you for your understanding.***

Cancellation/Rescheduling Policy (meeting, deposition, or testimony):

- Within 1 week of travel to a deposition or trial appearance, the client agrees to minimum commitment equivalent to a full day of the doctor's time when the client cancels or reschedules physician's time……$4,800 per calendar day
- The client assumes responsibility for travel fees, penalties, or supplemental costs resulting from change in travel plans.

Laboratory and Equipment Fees ……Advance quote prepared as needed

<u>Physician's Support Staff</u>:

Associate Scientist ……. $185 to $255 per hour

Staff MD/PhD Epidemiologist……$235 to $375 per hour

Nurse Practitioner (advance practice) …..…….…… $205 to $295 per hour

Exhibit 3

4 Year Expert Testimony List of Lucasz Gondek, MD, PhD

Date	Case Name	Case Number	Deposition or Trial
29-Jul-2024	Godfrey, Patricia v. Union Pacific Railroad Company		Deposition
7-Aug-2024	Bonar, Jason v. Norfolk Southern Railway		Deposition