Exhibit 456

Vivian Connard (for the Estate of Stephen Matthew Connard) v. United States of America

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

Case No. 7:23-cv-01557

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 Mr. Stephen Connard who passed away at the age of 50, due to medical complications after treatment of his Acute Myeloid Leukemia. Mr. Connard underwent allogeneic hematopoietic stem cells transplantation (alloHCT) which was complicated by refractory chronic graft vs. host disease (GHVD) of the skin requiring prolonged immunosuppressive therapy. As a result, he suffered recurrent life-threatening infections, cardiac and pulmonary complications that eventually led to his demise in May 2010.

In an effort to provide a comprehensive analysis, I have examined the relevant medical data and documentation, including pathology reports, clinical documentation as well as Mrs. Vivian Connard's and Dr. Aaron P. Rapoport's depositions.

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

In March of 2001, Mr. Connard presented to the Greenbaum Cancer Center with cytopenias. He had a history of seasonal allergies and well controlled hypertension. In my review of the records, I saw no history of smoking or any prior chemotherapy exposures. Mr. Connard was diagnosed with acute myeloid leukemia (AML) at the age of 40 years based on the bone marrow biopsy performed on March 26, 2001, which demonstrated hypercellular bone marrow (80-90%) with 50-60% of blasts. Cytogenetic analysis revealed deletion of chromosome 7. Mr. Connard was treated with induction chemotherapy. The time sequential therapy was mentioned in the medical records, but the exact regimen was not listed. Based on my experience and knowledge of time sequential therapy developed by my former colleague, Dr. Judith Karp, it was likely a combination of cytarabine, anthracyclines and etoposide. Unfortunately, his AML was refractory to chemotherapy. He was then reinduced on a clinical trial with bevacizumab, cytarabine and mitoxantrone but his AML was again resistant to chemotherapy. His treatment was complicated by fungal pneumonia, bevacizumab-induced hypertension and renal insufficiency.

After completing his chemotherapy regimens, Mr. Connard underwent nonmyeloablative allogeneic, matched unrelated donor, peripheral blood stem cell transplantation (alloHCT) with cyclophosphamide conditioning on July 26, 2001. In this type of therapy, hematopoietic stem cells are obtained from unrelated donors and infused to the patient. The therapeutic effect of alloHCT relies predominantly on the “new” donor’s immune system to eradicate the residual leukemia cells. His early post-alloHCT course was complicated by grade III skin and grade II gastrointestinal tract graft-vs-host disease (GVHD), Staphylococcus epidermidis bacteremia, Enterococcus urinary tract infection, malnutrition requiring intravenous nutrition, noncardiogenic pulmonary edema, bilateral pleural effusions, right subclavian vein thrombosis requiring anticoagulation with enoxaparin and prolonged pancytopenia requiring frequent red cells and platelet transfusions.

The bone marrow biopsy at 30 days post-alloHCT demonstrated mixed chimerism (residual leukemia cells). The subsequent day 60 and day 112 bone marrow biopsies showed nearly full engraftment and no evidence of AML. Mr. Connard also developed chronic GVHD of the skin and mouth treated with a prolonged courses of steroids, tacrolimus and mycophenolate mofetil, which was complicated by steroid-induced diabetes and tacrolimus-induced renal insufficiency. He also required intravenous immunoglobulin injections given profound hypogammaglobulinemia (low antibodies level).

In April of 2005 Mr. Connard developed a varicella zoster infection with residual post-herpetic neuralgia (significant pain). In May 2005, routine surveillance computed tomography (CT) of the chest demonstrated bilateral upper lobe parenchymal densities. A bronchoscopy and bronchoalveolar lavage were performed and cultures grew Scedosporium prolifican fungus. His fungal pneumonia was treated with prolonged course of voriconazole and micafungin. Of note, Mr. Connard was profoundly immunocompromised at that time due to the fact that he was still receiving immunosuppression for his chronic GVHD.

In February 2006, he was admitted to the hospital with Influenza infection and underwent 2 bronchoscopies with bronchoalveolar lavage. No other organisms were identified besides Influenza A. His symptoms improved with Tamiflu and broad-spectrum intravenous antibiotics. At that time, he also had a flare up of skin and gut GVHD because of tapering off immunosuppression. He was again treated with steroids which was complicated by worsening diabetes.

In March 2006 he developed sepsis due to coagulase negative Staphylococcus and was treated with intravenous vancomycin. In May 2006 he had another flare of skin GVHD requiring a high dose of steroids.

During 2008, his skin GVHD with scleroderma-like features continued to progress with significant skin thickening and tightness limiting his range and motion and mobility (Deposition of Dr. Rapoport p. 63 line 22). He did not respond to higher doses of steroids and systemic immunosuppression as well as rituximab, thus photopheresis was started in September 2008. He did respond to the treatment with some improvement in skin thickness.

In November 2008 he was again admitted to hospital with respiratory failure and low blood pressure. Another bronchoscopy with bronchoalveolar lavage demonstrated Nocardia infection which was treated with prolonged course of Bactrim. This complication was related to Mr. Connard's profoundly immunocompromised status due to therapy for his chronic GVHD.

His skin GHVD remained refractory to implemented therapy and continued to worsen. He was started on imatinib and hydroxychloroquine in 2009. Around the same time, he was also found to have squamous cell carcinoma on his scalp which was excised. He was also diagnosed with esophageal stricture that required dilatation.

Mr. Connard was again hospitalized in November 2009 with fever and hypotension and was treated with broad-spectrum antibiotics and Tamiflu. He clinically improved but the source of infection was not identified.

He had another flare up of skin GHVD in April 2010 and was again treated with high dose steroids and rituximab. In early May he was admitted to hospital with respiratory failure likely due to multifocal pneumonia. A CT chest scan demonstrated patchy widespread consolidation throughout the lungs. His status deteriorated despite broad-spectrum antibiotics and antifungal and he was transferred to ICU and intubated. Given elevated cardiac enzymes concerning for myocardial infarction he also underwent cardiac catheterization which showed mild diffuse non-obstructive multivessel disease and severe secondary pulmonary hypertension. His hospital course was complicated with the development of acute renal failure. His status initially improved and he was extubated. On May 14, 2010, Mr. Connard had a cardiac arrest during a CT scan of the chest and after unsuccessful resuscitation he passed away.

According to the deposition of Mrs. Connard and consistent with my review of the medical records, Mr. Connard had never smoked and drank alcohol only occasionally (Dep. p. 73).

Mr. Connard had 4 siblings: 3 brothers and 1 sister (Dep. p. 63-68). None of his siblings has any major medical problems and none has been diagnosed with cancer.

Methods

As a clinician-scientist specializing not only in the treatment of hematologic cancers including but not limited to AML, 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 of patient's cancer diagnosis including molecular profile of the cancer cells, 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 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 unequivocally eliminate the confounder as a causative factor, 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 Mr. Connard'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 Mr. Connard'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 to the carcinogen and disease development. Additionally, I incorporate insights from epidemiological studies and toxicological data, which demonstrate population-level associations between specific exposures and hematologic malignancies (I incorporate by reference my general causation report including all of the opinions contained therein and the materials considered list, submitted in this case on 12/09/24). 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 is more likely than not or at least as likely as not, 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 AML

Mr. Connard was diagnosed with AML with a certain type of genetic rearrangement denoted as deletion 7 or monosomy 7. In this type of genetic abnormality one of the two

chromosomes 7 is deleted. This genetic abnormality is associated with adverse prognosis in AML (Halik et al. 2024). This chromosomal alteration is linked to poor outcomes due to its association with genomic instability, resistance to chemotherapy and high relapse rate. It is usually considered a second hit in cancer evolution and 33% of patients also have an acquired mutation in TP53 gene (Halik et al. 2024). In the context of AML, the term second hit refers to an additional genetic alteration that occurs after an initial mutation. This second alteration can drive the progression of the disease, making it more aggressive. Monosomy 7 has been identified as a chromosomal abnormality linked to exposure to genotoxic agents, including chemotherapy and radiation (S. M. Smith et al. 2003; Niemeyer and Baumann 2008). It often emerges in therapy-related myeloid neoplasms (t-MN) and is considered a marker of genomic instability caused by prior DNA-damaging treatments including benzene. Several studies have demonstrated the presence of this abnormalities in blood cells collected from humans exposed to benzene (Luoping Zhang et al. 1998; L Zhang 1998). In Mr. Connard's case, the molecular results further support my opinion that his AML was indeed caused by exposure to cytotoxic agents including benzene, TCE and PCE.

Hereditary factors

AML is predominantly a non-hereditary disease; however, certain hereditary factors can contribute to its development in some cases. It is estimated that genetic predisposition to AML occurs in approximately 5-10% of all AML diagnoses. These hereditary forms are associated with mutations in specific genes such as CEBPA**,** RUNX1**,** GATA2**,** DDX41 and others linked to familial myelodysplastic syndrome/AML syndromes. Hereditary AML is suspected in individuals who present with a personal or family history suggestive of an inherited predisposition to hematologic malignancies or other cancers. Key clinical scenarios that raise suspicion include family history of AML, myelodysplastic syndromes (MDS), or other hematologic malignancies in multiple family members across generations and early onset of AML typically diagnosed under 40 years, in the absence of environmental or other risk factors.

None of Mr. Connard's family members was diagnosed with cancer. He had 3 younger siblings: 2 brothers and 1 sister (Dep. p. 63-68). None of his siblings has any major medical problems and none has been diagnosed with cancer. Additionally, nothing from his medical history or presentation suggests inherited predisposition to AML. According to Dr. Rapoport's testimony, Mr. Connard had no hereditary risk factors or congenital predisposition to AML (p. 28). As a result, I am able to rule out hereditary factors as a cause of Mr. Connard's AML.

Social and medical factors

The detailed review of his medical records did not identify any underlying medical conditions, or history of radiation or chemotherapy prior to his diagnosis of AML that could have potentially contributed to Mr. Connard's cancer. He was not a smoker, and he was not obese (BMI ~25 based on available medical records).

Occupational exposure to potential carcinogens

Mr. Connard was a Certified Internal Auditor for the Office of Inspector General for the House of Representatives from 1998 through 2010 when he died (Dep. p. 38). Prior to that, he worked at the Department of Transportation in Washington, DC from 1990 until 1998 (Dep. p. 42). Based on his wife's testimony, he worked mostly in the office and telecommuting and did not have any occupational exposure to carcinogens during his employment in Washington, DC. According to Dr. Rapoport's testimony, besides exposure to contaminated water at Camp Lejeune, Mr. Connard had no known occupational exposure to genotoxic agents, chemotherapy or radiation that could explain his AML (p. 28), and therefore this can be ruled out in my differential etiology analysis.

Camp Lejeune environmental exposure to benzene, TCE and PCE

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 and is listed in my materials considered. 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 I presented the scientific evidence that proves the causal relationship between benzene exposure and hematological malignancies including AML.

While my report speaks to the ingestion exposures of Mr. Connard 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 Mr. Connard 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 analysis the range of concentration of TCE, PCE and benzene at the time Mr. Connard resided on-base were the following:

1. Hadnot Point (mainside baracks) from September 1977 until May 1978 and June 1979 until July 1981
    a. TCE: 69-546 ug/L
    b. PCE: 2-23 ug/L
    c. Benzene: 3-6 ug/L

According to Dr. Reynold's report, Mr. Connard's cumulative exposure to VOC based on the ATSDR assumption was the following:

a. TCE: 1,574,286 µg/l
b. PCE: 71,032 µg/l
c. Benzene: 24,072 µg/l

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 total leukemias for females, with the relative risk (RR) for exposure to TCE >5 µg/L was 1.43 (95% CI: 1.07–1.9). 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.

Mr. Connard's exposure to TCE (1,574,286 µg/l) was significantly higher than the levels in the Cohn study that demonstrated a significantly increased risk for leukemia. TCE at 50 µg/L is well above the EPA's maximum contaminant level of 5 µg/L. Based on the study findings,

individuals exposed to low levels of TCE could have a substantially elevated risk of AML. PCE exposures at or above 2 µg/L, could also contribute to risk in combination with TCE exposure, as the two compounds share toxic metabolic pathways and potential synergistic effects. In other words, the combined impact of multiple agents can significantly amplify this risk beyond what is predicted by studying each agent in isolation. This is due to potential synergistic interactions, where one toxin enhances the mutagenic effects of another, exacerbating DNA damage and genomic instability. Consequently, studies that focus solely on single-agent exposures to benzene, TCE or PCE would underestimate the leukemia risk in populations exposed to combinations of toxins.

In terms of benzene exposure, Mr. Connard's cumulative exposure was 24,072 µg/l 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; M. T. 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 exposed to more than ambient levels of benzene. This would result in a significant underestimation of the exposure effect on the incidence of AML.

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. The evolution of cancer science, as evidenced in the above Rappaport studies and others, suggests that cancer dose-response is not necessarily linear and 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 (Rapport 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). Mr. Connard was exposed to benzene at the age of 18 suggesting that the effect of benzene exposure may have been more pronounced compared to older individuals exposed to the same level of benzene.

The additive and/or synergistic effects of multiple genotoxic agents on leukemia risk underscore the complexity of toxic exposure in real-world settings. While individual genotoxic

agents have been shown to increase leukemia risk, the combined impact of multiple agents can significantly amplify this risk beyond what is predicted by studying each agent in isolation. Thus, the study by Bove and colleagues published in 2024 is particularly relevant in this case. The authors performed the analysis of cancer incidence among Camp Lejeune Marines/Navy personnel and civilian workers, totaling 12,083 and 1,563, respectively (Bove, Greek, Gatiba, Kohler, et al. 2024). A cancer incidence study using individual-level data from US population-based cancer registries has a greater capability than a mortality study of evaluating the association between exposure and the incidence of cancer. Cancer registry data also provides more granular information about the individual cancer taking into account cancer classifications and histology. Compared with Camp Pendleton, Camp Lejeune Marines/Navy personnel had significantly higher incidence of all myeloid cancers (HR=1.24; 95% CI: 1.03, 1.49), acute myeloid leukemia (HR=1.38; 95% CI: 1.03, 1.85), myelodysplastic and myeloproliferative syndromes (HR=1.68; 95% CI: 1.07, 2.62), polycythemia vera (HR=1.41; 95% CI: 0.94, 2.11) in addition to several solid tumor cancers.

Complications related to chemotherapy and alloHCT.

AML and induction chemotherapy (cytarabine, anthracyclines) used to treat Mr. Connard's AML are associated with significant clinical complications that can impact short- and long-term outcomes. Infections, bleeding, clotting, organ damage are a major concern during treatment with induction chemotherapy. 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). It is my opinion, to a reasonable degree of medical certainty, that Mr. Connard's induction and salvage chemotherapies course resulted in numerous complications including fungal pneumonia, bevacizumab-induced hypertension and renal insufficiency.

Allo-HCT is a potentially curative therapy for AML, 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 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).

Mr. Connard's early post-alloHCT course was complicated by grade III skin and grade II gastrointestinal tract GVHD requiring prolonged courses of immunosuppression with steroid, tacrolimus, and mycophenolate mofetil. As a result of his AML and his GVHD, he was profoundly immunocompromised, and his immunocompromised state resulted in Staphylococcus epidermidis sepsis and an Enterococcus urinary tract infection. He also developed malnutrition requiring intravenous nutrition as well as pulmonary edema, bilateral pleural effusions, renal insufficiency, right subclavian vein thrombosis requiring anticoagulation with enoxaparin and prolonged pancytopenia requiring frequent red cell and platelet transfusions. It is my

professional opinion, to a reasonable degree of medical certainty, that Mr. Connard's above complications are all related to his AML and his GVHD.

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). Unfortunately, Mr. Connard suffered from refractory chronic GVHD of the skin and mouth.

Chronic skin GVHD, particularly in its scleroderma-like form, arises when donor immune cells attack the recipient's skin and connective tissues following alloHCT. The underlying mechanism involves an aberrant immune response characterized by persistent inflammation and dysregulated T-cell and B-cell activity, leading to fibroblast activation and excessive deposition of extracellular matrix proteins, such as collagen, in the skin. This results in progressive skin thickening, stiffness, and reduced elasticity, resembling systemic sclerosis. Over time, these changes can impair mobility, restrict joint range of motion, and cause significant discomfort. The sclerodermatous involvement often extends beyond the skin to include subcutaneous tissues and fascia, further exacerbating functional limitations and compromising quality of life. In severe cases, chronic skin GVHD may also predispose individuals to secondary complications such as infection, ulceration, and systemic organ involvement.

Mr. Connard's chronic skin GVHD resulted in limited range of motion and mobility negatively impacting his quality of life. Patients like Mr. Connard, who develop chronic skin GVHD, are known to have significant morbidity and disability due to joint contractures, fragile skin, poor wound healing, inadequate lymphatic drainage and skin ulcers (Uhm et al. 2014). The unfortunate reality for patients with this form of chronic skin GVHD is that their quality of life is diminished due to long-term physical and mental pain and suffering.

Mr. Connard's chronic skin GHVD was particularly difficult to treat and required multiple potent immunosuppressive therapies including mycophenolate mofetil (CellCept), sirolimus, hydroxychloroquine (Plaquenil), steroids, rituximab, imatinib and photopheresis (Dr. Rapoport's deposition, page 94).

Despite this aggressive therapy his chronic skin GHVD continued to worsen. Unfortunately, such aggressive immunosuppression resulted in several life-threatening infections. Mr. Connard suffered from Influenza pneumonia and shingles complicated by significant post-herpetic neuralgia (significant pain). He was also hospitalized multiple times with recurring pneumonia requiring intravenous broad-spectrum antibiotics. He also underwent several bronchoscopies and was diagnosed with life-threatening fungal infections with Scedosporium prolificanfungus and Nocardia. He also developed a bacterial bloodstream infection. It is my professional opinion, to a reasonable degree of medical certainty, that Mr. Connard's chronic GVHD was a complication of his alloHCT, which was required to treat his AML.

As part of his chronic skin GVHD treatment, Mr. Connard was placed on corticosteroids. While steroids represent standard of care in the treatment of GVHD, endocrinopathies and musculoskeletal complications are additional known complications of this treatment, particularly with long-term steroid use. Corticosteroids can lead to osteoporosis, fractures, and avascular necrosis (Velentza, Zaman, and Sävendahl 2021). Mr. Connard developed avascular necrosis. It is a condition where prolonged use of high-dose steroids disrupts blood supply to the bone, leading to the death of bone tissue. This results in weakened bone structure, which can collapse over time. Commonly affecting weight-bearing joints like the hips. It often progresses silently in the early stages but later causes significant pain, limited mobility, and joint dysfunction. It can lead to arthritis and may require joint replacement surgery. Additionally, Mr. Connard developed steroid-induced muscle loss and weakness (Dep. p.59) as well as diabetes. It is my professional opinion, to a reasonable degree of medical certainty, that Mr. Connard's diabetes and his musculoskeletal complications above are complications related to his GVHD, which would not have developed in the absence of his developing AML.

Secondary malignancies are another significant late complication, with an increased risk attributed to prior chemotherapy, radiation, and immunosuppression. Common secondary cancers include skin cancers and hematologic malignancies, which contribute to late mortality among long-term survivors (Danylesko and Shimoni 2018). Mr. Connard did suffer from skin cancer and required surgical excision in 2009. Reviewing Mr. Connard's medical records and deposition transcripts, I could not find other plausible causes explaining Mr. Connard's development of skin cancer other than alloHCT. Chronic skin GHVD, radiation therapy used during conditioning regimen and prolonged use of immunosuppressive therapy are well known causes of skin cancer (Mansilla-Polo et al. 2024; Gruber, Wolff, and Koelbl 2024; Nielsen et al. 2025).

The complication after allo-HCT including chronic GHVD and severely immunocompromised state eventually resulted in Mr. Connard's death in May of 2010. Mr. Connard was admitted to the hospital with respiratory failure requiring supplemental oxygen (medical records and Dr. Rapoport testimony pages 71-79) and renal failure. He required broad-spectrum antibiotics and antifungal agents, intubation and ventilatory support. Given elevated cardiac enzymes concerning for myocardial injury he also underwent cardiac catheterization which showed mild diffuse non-obstructive multivessel disease and severe secondary pulmonary hypertension. On May 14, 2010, Mr. Connard had a cardiac arrest during a CT scan of the chest and after unsuccessful resuscitation he passed away.

Prior to his diagnosis of AML, Mr. Connard had been very athletic. He used to run marathons every year before his AML diagnosis (Dep. p. 60). He was not a smoker and had no cardiovascular risk factors. Yet, in the days leading up to his death, he was diagnosed with diffuse mild cardiovascular disease and severe pulmonary hypertension. Coronary artery disease is a well-known post-alloHCT complication, particularly in a patient with chronic GVHD. Chronic GVHD is characterized by systemic inflammation and immune dysregulation, leading to vascular injury and endothelial dysfunction, which are critical contributors to the development of cardiovascular disease. Chronic GVHD-associated vascular endothelial damage leads to early atherosclerosis (damage, stiffness and narrowing of the blood vessels). Immune-mediated injury, compounded by oxidative stress, accelerates plaque deposition in coronary arteries. Radiation

therapy, used as part of conditioning, and calcineurin inhibitors (e.g., tacrolimus), frequently exacerbate endothelial dysfunction. Long-term steroid use for GVHD management increases the risk of hyperlipidemia, diabetes and insulin resistance, and central obesity, all of which are major contributors to coronary disease (Armenian and Chow 2014; Tichelli et al. 2007; Armenian and Chow 2014). Additionally, it has been shown that in patients like Mr. Connard, who received an alloHCT, active chronic GVHD was associated with 4-fold increase of cardiovascular death (Chow et al. 2014).

Shortly before his death, Mr. Connard was diagnosed with severe pulmonary hypertension. Pulmonary complications are significant contributors to morbidity and mortality following alloHCT, particularly in the context of chronic GVHD. Organizing pneumonia and bronchiolitis obliterans syndrome (BOS) hallmarks pulmonary manifestation of chronic GVHD, characterized by progressive small airway inflammation and fibrosis, leading to obstructive lung disease with symptoms such as dyspnea and cough. Additionally, pulmonary hypertension can emerge post-alloHCT due to endothelial injury, recurrent infections, lung fibrosis, thrombotic microangiopathy, or cardiac complications (O'Brien et al. 2024).

In my review of Mr. Connard's records, I did not see any history or diagnosis of pre-alloHCT cardiovascular disease or cardiac issues. It was only after his acute episode in 2010 when he was diagnosed with pulmonary hypertension and non-critical coronary artery disease. Mr. Connard's death in 2010, more than 8 years after his alloHCT, is more likely than not, and to a reasonable degree of medical certainty, related to cardiovascular and pulmonary complications of alloHCT including but not limited to heart failure, myocardial infarction, pulmonary hypertension and arrythmia. As previously mentioned, Mr. Connard required alloHCT to cure his AML that was at least as likely as not a result of having been exposed to genotoxic and immunotoxic chemicals at Camp Lejeune which.

In conclusion, while alloHCT offers a potential cure for many life-threatening conditions, including AML, early and late complications may result in significant morbidity and early mortality. In Mr. Connard's case, significant post-alloHCT complication, mainly refractory GVHD, profoundly immunocompromised status, infections as well as cardiac and pulmonary complications led to his premature death at the age of 50 years. All of these complications are directly related to his underlying AML, which as previously mentioned, was at least as likely as not, caused by Mr. Connard's exposure to Benzene, TCE and PCE at Camp Lejeune.

Conclusions

Mr. Connard was diagnosed with AML at the age of 40 years. None of Mr. Connard's relatives suffered from hematological malignancy, thus, it is highly unlikely that his cancer was a consequence of hereditary predisposition. Moreover, the molecular underpinning of his leukemia, namely monosomy 7, has been associated with exposure to genotoxic therapies and environmental exposure to DNA damaging agents such as benzene.

The detailed review of his medical records did not identify any underlying medical conditions, or history of radiation or chemotherapy prior to his diagnosis of AML that could have potentially contributed to Mr. Connard's cancer.

As a result of his diagnosis and applied therapies, Mr. Connard suffered numerous complications as listed above. His quality of life was deeply affected by chronic GVHD of the skin that significantly limited his range of motion and mobility and resulted in recurrent infections requiring frequent hospitalization and procedures such as bronchoscopies, bone marrow biopsies, several central venous access placements, and intubation. The combination of infection, cardiovascular disease and severe pulmonary hypertension eventually resulted in his death on May 14, 2010. Even though chemotherapy and alloHCT extended Mr. Connard's life, it had a profound negative impact on his quality of life.

In my experience interacting with patients with severe, chronic GVHD following alloHCT, I have witnessed the profound physical, emotional, and psychological toll this condition may cause. The burden of symptoms often leads to a chilling reflection on whether the cost the patients have to pay for the extended life was worth the benefit of prolonged life.

Applying a differential etiology analysis and following the weight of the evidence approach to investigating and interpreting the data I conclude that Mr. Connard's exposure to TCE, PCE and benzene through contaminated water at Camp Lejeune was more likely than not the cause of his AML and the complications that led to his death.

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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Case Materials Received

Legal Documents/Materials:

- Short Form Complaint
- Discovery Pool Profile Form

Depositions:

- Videotaped Deposition of Vivian Connard, taken 26 February 2024, with Exhibits
- Remote Deposition of Thor Berg taken 30 May 2024 with Exhibits
- Videotaped, In-Person, Virtual Zoom deposition of Aaron Rapoport, M.D., taken on 7 May 2024 with Exhibits

Medical Documents/Materials:

- 000000_01557_CONNARD_0000000019
- 000001_01557_CONNARD_0000000028
- 000002_01557_CONNARD_0000000029
- 000003_01557_CONNARD_0000000040
- 000004_01557_CONNARD_0000000052
- 000005_01557_CONNARD_0000000062
- 000006_01557_CONNARD_0000000076
- 000007_01557_CONNARD_0000000089
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- 000015_01557_CONNARD_0000000171
- 000016_01557_CONNARD_0000000183
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- 000000_01557_CONNARD_0000000940
- 000001_01557_CONNARD_0000000493
- 000001_01557_CONNARD_0000001124
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- 000003_01557_CONNARD_0000001506
- 000000_01557_CONNARD_0000001666

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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12. **Gondek LP**, Tiu R, O'Keefe CL, Sekeres MA, Theil KS, Maciejewski JP. Chromosomal lesions and uniparental disomy detected by SNP arrays in MDS, MDS/MPD, and MDS-derived AML. *Blood*. 2008;111(3):1534-1542.
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23. Tiu RV, **Gondek LP**, O'Keefe CL, Elson P, Huh J, Mohamedali A, Kulasekararaj A, Advani AS, Paquette R, List AF, Sekeres MA, McDevitt MA, Mufti GJ, Maciejewski JP. Prognostic impact of SNP array karyotyping in myelodysplastic

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34. Webster JA, Luznik L, Tsai HL, Imus PH, DeZern AE, Pratz KW, Levis MJ, Gojo I, Showel MM, Prince G, Bolaños-Meade J, **Gondek LP**, Ghiaur G, Dalton WB, Jain T, Fuchs EJ, Gladstone DE, Gocke CB, Ali SA, Huff CA, Borrello IM, Swinnen L, Wagner-Johnston N, Ambinder RF, Jones RJ, Smith BD. Allogeneic transplantation for Ph+ acute lymphoblastic leukemia with posttransplantation cyclophosphamide. Blood Adv. 2020 Oct 27;4(20):5078-5088. *Analyzed data*
35. Wang S, Zbib NH, Skaist A, Gui J, Madero-Marroquin R, De Marchi F, **Gondek LP**, Matsui W, Lau BW. Whole-exome sequencing identifies functional classes of gene mutations associated with bone marrow failure in pediatric Fanconi Anemia patients. Eur J Haematol. 2021 Aug;107(2):293-294. *Analyzed data.*
36. Sidhom JW, Siddarthan IJ, Lai BS, Luo A, Hambley BC, Bynum J, Duffield AS, Streiff MB, Moliterno AR, Imus P, Gocke CB, **Gondek LP**, DeZern AE, Baras AS, Kickler T, Levis MJ, Shenderov E. Deep learning for diagnosis of acute promyelocytic leukemia via recognition of genomically imprinted morphologic features. NPJ Precis Oncol. 2021 May 14;5(1):38. *Analyzed data.*
37. Karantanos T, **Gondek LP**, Varadhan R, Moliterno AR, DeZern AE, Jones RJ, Jain T. Gender-related differences in the outcomes and genomic landscape of patients with myelodysplastic syndrome/myeloproliferative neoplasm overlap syndromes. Br J Haematol. 2021 Jun;193(6):1142-1150. *Designed experiment, analyzed data, wrote the manuscript.*
38. Ambinder A, Smith M, Tsai HL, Varadhan R, DeZern A, Dalton W, Gocke C, Webster J, **Gondek L**, Gojo I, Ali SA, Huff CA, Swinnen L, Wagner-Johnston N, Showel M, Prince G, Borrello I, Bolaños-Meade J, Luznik L, Jain T, Imus P, Fuchs E, Ambinder R, Gladstone DE, Levis M, Jones R, Ghiaur G, Smith BD. Nonmyeloablative Allogeneic Transplantation with Post-Transplant Cyclophosphamide for Acute Myeloid Leukemia with IDH Mutations: A Single Center Experience. Clin Lymphoma Myeloma Leuk. 2021 Oct 9;S2152-2650(21)02071-1. *Analyzed data.*

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40. Schaefer EJ, Wang HC, Karp HQ, Meyer CA, Cejas P, Gearhart MD, Adelman ER, Fares I, Apffel A, Lim K, Xie Y, Gibson CJ, Schenone M, Murdock HM, Wang ES, **Gondek LP**, Carroll MP, Vedula RS, Winer ES, Garcia JS, Stone RM, Luskin MR, Carr SA, Long HW, Bardwell VJ, Figueroa ME, Lindsley RC. BCOR and BCORL1 mutations drive epigenetic reprogramming and oncogenic signaling by unlinking PRC1.1 from target genes. Blood Cancer Discovery. 2021 Dec 17; *Enrolled patients, wrote the manuscript*
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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*
43. Murdock HM, Kim HT, Denlinger N, Vachhani P, Hambley BC, Manning BS, Gier S, Cho C, Tsai HK, McCurdy SR, Ho VT, Koreth J, Soiffer RJ, Ritz J, Carroll MP, Vasu S, Perales MA, Wang ES, **Gondek LP**, Devine SM, Alyea EP, Lindsley RC, Gibson CJ. Impact of diagnostic genetics on remission MRD and transplantation outcomes in older AML patients. Blood. 2022 Mar 14. *Enrolled patients, analyzed data, wrote the manuscript*
44. Karantanos T, Tsai HL, **Gondek LP**, DeZern AE, Ghiaur G, Dalton WB, Gojo I, Prince GT, Webster J, Ambinder A, Smith BD, Levis MJ, Varadhan R, Jones RJ, Jain T. Genomic landscape of myelodysplastic/myeloproliferative neoplasm can predict response to hypomethylating agent therapy. Leuk Lymphoma. 2022 Apr 4:1-7. doi: 10.1080/10428194.2022.2057488. *Analyzed data, wrote the manuscript*
45. Tsai HK, Gibson CJ, Murdock HM, Davineni PK, Harris M, Wang ES, **Gondek LP**, Kim AS, Nardi V, Lindsley RC. Allelic Complexity of KMT2A Partial Tandem Duplications in Acute Myeloid Leukemia and Myelodysplastic Syndromes. Blood Adv. 2022 May 18:bloodadvances.2022007613. doi: 10.1182/bloodadvances.2022007613. *Enrolled patients, analyzed data, wrote the manuscript*
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47. Karantanos T, Teodorescu P, Arvanitis M, Perkins B, Jain T, DeZern AE, Dalton WB, Christodoulou I, Paun BC, Varadhan R, Esteb C, Rajkhowa T, Bonifant C, **Gondek LP**, Levis MJ, Yegnasubramanian S, Ghiaur G, Jones RJ. CCRL2 affects the sensitivity of myelodysplastic syndrome and secondary acute myeloid leukemia cells to azacitidine. Haematologica. 2022 Dec 15. doi: 10.3324/haematol.2022.281444. Online ahead of print. *Analyzed data, wrote the manuscript*
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49. Webster JA, Reed M, Tsai HL, Ambinder A, Jain T, Dezern AE, Levis MJ, Showel MM, Prince GT, Hourigan CS, Gladstone DE, Bolanos-Meade J, **Gondek LP**, Ghiaur G, Dalton WB, Paul S, Fuchs EJ, Gocke CB, Ali SA, Huff CA, Borrello IM, Swinnen L, Wagner-Johnston N, Ambinder RF, Luznik L, Gojo I, Smith BD, Varadhan R, Jones RJ, Imus PH. Allogeneic Blood or Marrow Transplantation with High-Dose Post-transplantation Cyclophosphamide for Acute Lymphoblastic Leukemia in Patients Aged ≥55. Transplant Cell Ther. 2022 Dec 29:S2666-6367(22)01866-8. doi: 10.1016/j.jtct.2022.12.018. Online ahead of print. *Enrolled patients, analyzed data, wrote the manuscript*
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51. Pasca S, Guo MZ, Wang S, Stokvis K, Shedeck A, Pallavajjala A, Shams C, Pallavajjala R, DeZern AE, Varadhan R, Gocke CD, Jones RJ, **Gondek LP**. Cell-free DNA measurable residual disease as a predictor of postallogeneic hematopoietic cell transplant outcomes. Blood Adv. 2023 Aug 22;7(16):4660-4670. doi: 10.1182/bloodadvances.2023010416. *Senior author*

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53. Teodorescu P, Pasca S, Choi I, Shams C, Dalton WB, **Gondek LP**, DeZern AE, Ghiaur G. An accessible patient-derived xenograft model of low-risk myelodysplastic syndromes. Haematologica. 2023 Jul 6. doi: 10.3324/haematol.2023.282967. Online ahead of print. *Perform sequencing, analyzed data, wrote the manuscript*
54. Jain T, Tsai HL, Elmariah H, Vachhani P, Karantanos T, Wall SA, **Gondek LP**, Bashey A, Keyzner A, Tamari R, Grunwald MR, Abedin S, Nadiminti KV, Iqbal M, Gerds AT, Viswabandya A, McCurdy SR, Al Malki MM, Varadhan R, Ali H, Gupta V, Jones RJ, Otoukesh S. Haploidentical donor hematopoietic cell transplantation for myelodysplastic/myeloproliferative overlap neoplasms: results from a North American collaboration. Haematologica. 2023 Jul 6. doi: 10.3324/haematol.2023.283426. Online ahead of print. *Enrolled patients, 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

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

Exhibit 3

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