Exhibit 450

Bruce Hill v. United States of America

U.S. District Court for Eastern District of NC, Southern Division
Case No. 7:23-cv-28

Specific Causation Expert Report of Dean W. Felsher, M.D., Ph.D.

Prepared by

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Contents

I. EDUCATION AND QUALIFICATIONS

I am an adult over the age of 18 and am not a party to this lawsuit. I have personal knowledge of the facts set forth in this declaration, except for those based on my professional expertise and reliance on relevant materials. I can and would competently testify to these facts if requested.

I am currently a Professor of Oncology at Stanford University, serving in both the Departments of Medicine and Pathology. I also hold the position of Associate Chief of the Division of Oncology. I have more than 25 years of experience in cancer research, carcinogenesis, and oncology. My career has focused on studying the mechanisms of cancer, specifically how oncogenes initiate and sustain tumor development. For over 25 years, I have directed the Dean Felsher Laboratory at Stanford University, which investigates these processes. I also mentor and supervise medical students, research fellows, and junior faculty in oncology, cancer biology, and translational medicine.

In my medical career, I have treated thousands of patients with cancer, including hundreds of patients with hematopoietic cancer. I have taught courses on the causes and treatments of cancer, including specialized topics in cancer biology, tumor immunology, and carcinogenesis. My work as an educator has extended to both formal classroom settings and direct mentorship of clinical and research fellows.

I received my Bachelor of Arts in Chemistry from the University of Chicago, followed by both an M.D. and a Ph. D. in Molecular Biology with a specialization in cancer immunology from the University of California, Los Angeles. I completed my residency in internal medicine at the Hospital of the University of Pennsylvania and a fellowship in hematology-oncology at the University of California, San Francisco, where I conducted post-doctoral research under the Nobel Laureate Dr. J. Michael Bishop. I am board certified in internal medicine and medical oncology, although I have not recertified, as my current role focuses on research and education rather than direct clinical care.

From 1997 to 1999, I served as a clinical instructor and Assistant Professor at the University of California, San Francisco, before joining Stanford University, where I have held various academic positions since 1999. In 2012, I became a full professor, and since then, I have held leadership roles as the founding director of Stanford's Translational Research and Applied Medicine (TRAM) Center, the Cancer Translational Nanotechnology Training Program, and the Masters of TRAM Graduate Program. I also serve as the Director of Admissions for the Medical Scientist Training Program and am one of three principal investigators for Stanford's NIH-funded Clinical and Translational Science Award (CTSA) program, which oversees clinical and translational research at the university.

My research has extensively focused on the mechanisms of cancer, including carcinogenesis and tumor microenvironment, and the development of novel cancer diagnostics and treatments. I have published over 100 peer-reviewed articles in leading scientific journals, including but not limited to:

- Smith, Martyn T et al. "The Key Characteristics of Carcinogens: Relationship to the Hallmarks of Cancer, Relevant Biomarkers, and Assays to Measure Them." *Cancer epidemiology, biomarkers & prevention: a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology* vol. 29,10 (2020): 1887-1903. doi:10.1158/1055-9965.EPI-19-1346

- Goodson, William H III et al. "Assessing the carcinogenic potential of low-dose exposures to chemical mixtures in the environment: the challenge ahead." *Carcinogenesis* vol. 36 Suppl 1,Suppl 1 (2015): S254– S296. doi: 10.1093/carcin/bgv039

- Casey, Stephanie C et al. "The effect of environmental chemicals on the tumor microenvironment." *Carcinogenesis*, vol. 36 Suppl 1,Suppl 1 (2015): S160– S183. doi:10.1093/carcin/bgv035

- Block, Keith I et al. "Designing a Broad-Spectrum Integrative Approach for Cancer Prevention and Treatment." Seminars in Cancer Biology, vol. 35 Suppl,Suppl (2015): S276–S304. doi:10.1016/j.semcancer.2015.09.007

- Casey, Stephanie C et al. "Cancer prevention and therapy through the modulation of the tumor microenvironment." *Seminars in cancer biology* vol. 35, Suppl (2015): S199– S223. doi: 10.1016/j.semcancer.2015.02.007

- Beer, Shelly et al. "Hepatotoxin-Induced Changes in the Adult Murine Liver Promote MYC-Induced Tumorigenesis." *PloS one* vol. 3,6 e2493. 18 Jun. 2008, doi: 10.1371/journal.pone.0002493

- Beer, Shelly et al. "Low-level shRNA cytotoxicity can contribute to MYC-induced hepatocellular carcinoma in adult mice." *Molecular therapy: the journal of the American Society of Gene Therapy* vol. 18,1 (2010): 161–70. doi:10.1038/mt.2009.222

- Beer, Shelly et al. "Developmental context determines latency of MYC-induced tumorigenesis." *PLoS biology* vol. 2,11 (2004): e332. doi: 10.1371/journal.pbio.0020332

- Woodard, Lauren E et al. "Impact of hydrodynamic injection and phiC31 integrase on tumor latency in a mouse model of MYC-induced hepatocellular carcinoma." *PloS One, vol. 5, no. 6, e11367, Jun. 29,* 2010, doi: 10.1371/journal.pone.0011367

- Dhanasekaran, Renumathy et al. "The MYC oncogene - the grand orchestrator of cancer growth and immune evasion." *Nature reviews. Clinical oncology* vol. 19,1 (2022): 23-36. doi:10.1038/s41571-021-00549-2

In addition to my research, I have served on the editorial boards of several leading cancer-related journals and as a scientific reviewer for over 20 top-tier journals, including *Nature*, *Science*, *Cell*, and *Nature Medicine*. I hold senior editorial roles with *Cancer Research* and *Oncogene*, where I review cancer-related studies. My publications have been cited over 25,000 times, and I have received numerous awards for my contributions to oncology, including the National Cancer Institute Outstanding Investigator Award.

I have been invited to present my research at numerous international cancer conferences and have delivered over 240 presentations on topics related to cancer causation, tumor biology, and the development of cancer treatments. I have also lectured extensively on cancer mechanisms and carcinogenesis, training the next generation of oncologists and cancer researchers.

My opinions in this declaration are held to a reasonable degree of medical and scientific certainty. They are based on my education, training, and experience, as well as my clinical and scientific research on cancer and cancer causation, knowledge of the literature, and my review of relevant materials and records.

Attached as exhibits are my CV, a list of publications from the past 10 years, a list of cases in which I testified in the past 4 years, and my fee schedule for this case.

II. METHODS

I describe my methodology in further detail below, which includes considering my experience as a scientist and a physician. I reviewed the medical and scientific literature and used a weight-of-evidence approach to evaluate causation in this case. I integrated my analysis of mechanistic, preclinical, and epidemiological studies and considered the Bradford Hill considerations, using methods that any scientist and doctor with my training would utilize and that are commonly utilized by other scientists, governmental agencies, and public organizations such as the EPA, NTP, ATSDR, and IARC, in their analysis of similar considerations. I have also reviewed, and to varying degrees, relied upon general causation reports from Morris Maslia, Kelly Reynolds, Kate Gilbert, and my own general causation report in this case.

I performed a differential etiology using the same methods that are would be generally accepted and commonly used in usual practice of physicians and scientists with expertise in determining etiology and are generally considered standard for considering the contributions of risk factors to a disease process. In my daily practice as a physician and scientist, I conduct analysis to determine the contributing risk factors and/or causes of disease processes. Such differential etiology is often multi-faceted and involves consideration of a multitude of mechanisms that can occur over a period of time and can be influenced by other factors that are related to both the individual and environment. In these analyses I performed, I consider both general factors that are applicable often to a general population as well as factors that are specific to an individual. My opinions consider whether there is at least as likely as not a causal relationship between the chemical carcinogens described in this report and hematopoietic cancers, and in particular, non-hodgkins lymphoma (NHL) and chronic lymphocytic leukemia (CLL) also called small lymphocytic lymphoma (SLL). I understand "at least as likely as not" to be the causation standard under the Camp Lejeune Justice Act. I define "at least as likely as not" as meaning that there is at least an equal or greater than equal chance (50% or greater chance) that the exposure described below was sufficient to have a causal relationship. In defining "at least as likely as not," I have also reviewed the *2017 ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases* and its definition of "equipoise and above," which I have found to be reliable based on my years of education, research, and clinical practice.

Also, I considered other risk factors associated with the development of hematopoietic cancers including NHL as part of my differential etiology, and I reviewed the materials relating to Mr. Hill to determine which, if any, of them apply to his case. Included in that analysis was an evaluation of the trichloroethylene (TCE), vinyl chloride (VC), and tetrachloroethylene (perchloroethylene, PCE) and benzene concentrations to which Mr. Hill was exposed during his time at Camp Lejeune. I then performed a differential etiology as described above to determine whether Mr. Hill's exposure to chemicals in the water at Camp Lejeune is at least as likely as not the cause of his NHL.

To perform a differential etiology generally, I first compiled a list of demographic and risk factors for NHL based both on my experience with hematopoietic cancers and on information from the same scientific sources referenced herein. For completeness, I include demographic and risk factors, even if I do not conclude there is sufficient evidence to consider them as risk factors for NHL, if there is any scientific literature suggesting an association with NHL.

Second, I evaluated, for each individual, here for Mr. Hill, whether any of the risk factors apply to the particular plaintiff based on all available evidence, including medical records, deposition testimony, military records, and any other relevant evidence about the individual plaintiff's history.

Third, for each risk factor that could apply, I applied the available scientific knowledge and literature to determine whether a specific risk factor was at least as likely as not a substantial contributing factor and whether it was a substantial contributing cause in the development of the plaintiff's NHL.

As a scientist and a physician, I review the literature based upon my already-existing knowledge of the medical literature, reviewing the science cited in such publications, and reviewing the science that cites these documents. I do not rely on any single specific search term or combination of terms alone, but my searches did include using multiple search engines, including PubMed, Google, Google Scholar, and/or DuckDuckGo. I included in my search terms: Camp Lejeune, and each specifically named carcinogen (as described further below): trichloroethylene (TCE), benzene, vinyl chloride (VC), and tetrachloroethylene (perchloroethylene, PCE). I also reviewed related documents pertaining to Camp Lejeune and to each carcinogen from the EPA, ATSDR, IARC, as well as other reports and associated literature as references and/or described in these government reports or in my documents considered list.

In my materials considered, I include the primary documents reviewed. This includes published scientific literature and government documents from ATSDR, IARC, and other organizations. When I cite these government reports, I note that I have also independently reviewed the data and literature contained therein. I note that not all of the documents that underlie my opinions are contained in these documents.

I reserve the right to continue to review medical and scientific literature and other documents made available to me that may result in new opinions. Further, when I am deposed, I cannot anticipate what questions I will be asked or what reports, documents, or publications I will be shown by defense counsel, which may result in my having new opinions. In addition, when the defense experts' reports and testimony are made available to me, I will likely have additional scientific and medical opinions.

III. SUMMARY OF OPINIONS

I conclude with a reasonable degree of medical certainly that it is more likely than not that exposure to volatile organic compounds (VOCs) that include benzene, trichlorethylene (TCE), VC and tetrachlorethylene (PCE) can generally be a cause of hematopoietic cancers including chronic lymphocytic leukemia (CLL) and other non-hodgkins lymphomas (NHL).

I conclude as to Mr. Bruce Hill specifically, with a reasonable degree of medical certainty that his exposure to Camp Lejeune water containing a combination of benzene, VC, TCE and PCE was, more likely than not, a substantial contributing cause of his CLL.

IV. HEMATOPOIETIC CANCERS AND NON-HODGKIN'S LYMPHOMA

Hematopoietic (blood) cancers are cancers often derived from hematopoietic cells and stem cells (Bryder 2006, Filipek-Garzala 2024). There are many types and subtypes of hematopoietic stems cells that when they acquired sufficient genetic changes such as mutations, deletions and chromosomal translocations can become cancerous (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger p). This includes erythroid, myeloid, and lymphocytes amongst other lineages. Most types of hematopoietic cancers initially arise in immature stem, progenitor cells and/or less differentiated cells that can in some circumstances further matured to become more or fully differentiated (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).

There are a multitude of associated hematopoietic (blood) diseases, including hematopoietic cancers commonly deriving from immature cells as well as less or more mature cells (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024). For example, diseases causing a disruption of the bone marrow function can be a myeloproliferative disorder such as chronic myelogenous leukemia (CML) or essential thrombocytopenia (ET) or a disease called myelodysplasia. Alternatively, when the disease involves solid immune organs such as lymph nodes or the spleen, then the disease is often described as a lymphoma. Additionally, hematopoietic cancers are usually considered leukemias when they are associated mostly with abnormal increased numbers of leukocytes that have become abnormal cancerous cells seen in excess in the blood. Hematopoietic cancers are usually considered lymphomas when they are associated with abnormal increased normal of lymphocytes in hematopoietic or lymphoid organs such as bone marrow, lymph nodes and/or spleen. However, all types of hematopoietic cancers, whether designated by a name of leukemia or lymphoma, generally are both in the blood and the hematopoietic organs. Hence, hematopoietic cancers can be called a leukemia or lymphoma but indeed be the same disease such as chronic lymphocytic leukemia and small lymphocytic lymphomas which are the same disease. While each of these types and subtypes have different naming conventions, they are all hematopoietic cancers due to the types of cells from which they arise, and many of these cancers begin and/or are initiated in the same common hematopoietic stem cells even if the final disease at diagnosis for a patient corresponds to a more specific hematopoietic lineage and differentiative state, or subtype of that lineage or state, as has been generally accepted for decades (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).

All hematopoietic cancers including those that are derived from erythroid, myeloid cells, B-cell or T-cell lymphocytes arise when an otherwise normal cell acquires a compliment of genetic events that activate oncogenes and inactivate tumor suppressor genes (Weissman, Blood, 2008). These genetic events can occur spontaneously but occur more frequently when bone marrow cells, myeloid cells or lymphocytes are exposed to environmental agents, such as carcinogens that are known to initiate, promote, accelerate cancer formation and can reduce the sensitivity of cancer cells to therapy. (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).

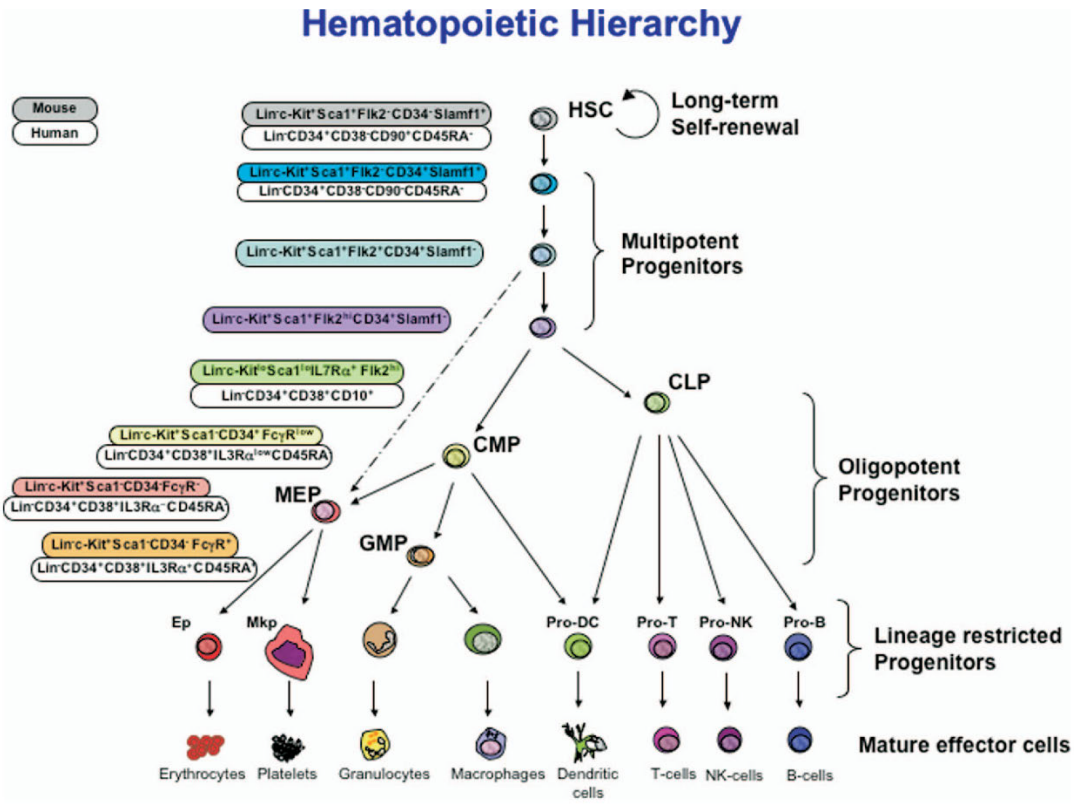

Figure 1. Schematic of hematopoietic development indicating intermediates in the hierarchy of hematopoietic differentiation. Surface markers used for isolation are indicated at left for human (top) and mouse (bottom) for each stem and progenitor cell. HSC indicates long-term reconstituting, self-renewing; MPP, multipotent progenitors with limited self-renewal leading to transient but multilineage reconstitution; CMP, common myeloid progenitor; CLP, common lymphoid progenitor; BLP, B lymphocyte progenitor; ProT, T-cell progenitor; GMP, granulocyte/macrophage progenitor; MEP, megakaryocyte/erythroid progenitor; MKP, megakaryocyte progenitor; EP, erythroid progenitor. This figure and legend is from Figure 1 in Bryder D, Rossi DJ, Weissman IL. Hematopoietic stem cells: The paradigmatic tissue specific stem cell. Am J Pathol. 2006; 169:338-346, with permission from the American Society for Investigative Pathology.

Hematopoietic cancers include Lympho-Hematopoietic cancers that are derived from lymphocytes that include: acute lymphocytic or lymphoblastic leukemia (ALL), multiple myeloma (MM), non-hodgkins lymphoma (NHL) and hodgkins lymphoma (HL). Patients with these types of hematopoietic cancers have cancerous lymphocytes that can be found in the blood and bone marrow and other locations. Lymphocytes can be either B-cells that are the antibody producing cells or T-cells that are the cells that have a T-cell receptor. B-cells are the type of the cell that when they become mature are normally in the human body helps fight infections through the production of antibodies. T-cells are the type of cell that when they become mature are normally in the body to help fight infections and also help prevent cancer through their T-cell receptor that can result that help the function other immune cells including B-cells. ALL is a type of cancer that arising from lymphocytes, more commonly B-cells but also T-cells and usually in less mature pre-B or pre-T-cells or lymphocytes that have not completed the formation of their antibodies or T-cell receptor respectively. MM is a type of cancer arising in B-cell lymphocytes that are

antibody producing. B-cell lymphocytes make antibodies that are secreted, and they are called plasma cells.

NHL is a group of cancers that occur more commonly in B cells but also occurs in T cells and rarely in other types of lymphocytes, that occur in different stages of their maturation (Rohit 2020, Leval 2020, Pasqualucci 2024). Most commonly NHL occurs in mature B-cells in humans. NHL can be further divided clinically into the high grade fast growing lymphomas and low grade more slow growing lymphomas and then further subdivided into many other subtypes mainly based upon their phenotypic features as observed by a pathologist under the microscopy through histology as well as through characterization of cell surface markers (Rohit 2020, Leval 2020, Pasqualucci 2024) The World Health Organization (WHO) consensus classification of hematologic malignancies uses an updated version of the Revised European-American Lymphoma (REAL) classification for lymphoid neoplasms (de Leval, Cancer Journal, 2020). In the Working Formulation. NHL can be subdivided into many specific subtypes that can be differentiated based upon their morphologic features, surface antigen phenotype, biological behavior, immunoglobulin rearrangement status, oncogenic activation as well as other parameters (Rohit 2020, Leval 2020, Pasqualucci 2024).

Mr. Hill has Chronic Lymphocytic leukemia (CLL), also known as Small lymphocytic lymphoma (SLL), which is a type of hematopoietic malignancy, lympho-hematopoietic malignancy and NHL (Shadman, 2023, Kumar 2023, Montague 2023). CLL can affect adults as young as 30; however, it is more common in adults at an average age of 70 years. CLL is extremely rare in children. The incidence is known to rapidly increase with increasing age. CLL has a slightly higher incidence in men. CLL is most commonly seen in adults of the Western population and amongst the Caucasian population compared to the Asian Pacific Islanders or the African-American population.

CLL is diagnosed based upon over 5 × 10^9/L monoclonal B cells in the blood (Shadman, 2023, Kumar 2023, Montague 2023). CLL is associated with an immunocompromised state and an increased complications from infections. It is estimated that up to 80% of CLL patients will experience a severe infectious event during the course of the disease, and that mortality due to infections can be as high as 50% (Murru 2023). The majority of CLL are asymptomatic at the time of diagnosis. Approximately one-third of CLL patients do not require treatment. There are multiple prognostic models to estimate the time to first treatment and the overall survival. For patients who are asymptomatic clinical observation is the standard of care. Patients who need treatment include those with symptomatic disease who have bulky or progressive lymphadenopathy or hepatosplenomegaly and/or low blood counts and/or symptoms of fever, drenching night sweats, and weight loss (also known as B symptoms). Treatment includes Bruton tyrosine kinase (BTK) inhibitor (acalabrutinib, Zanubrutinib, or ibrutinib), a BCL2 inhibitor (venetoclax). There is no evidence that starting either type of therapy improves outcomes. In patients with multiple relapses, chimeric antigen receptor T-cell (CAR-T) therapy is recommended.

In general, there are certain demographics and many risk factors generally associated with lympho-hematopoietic cancers and/or NHL (Koff 2015).

Age and gender: In general, increasing age is associated with increased risk. In some circumstances, gender influences risk, with males slightly more at risk than females.

Family History: There is some suggestion that a family history of a hematological malignancy can be associated with increased risk (Cerhan, Blood, 2015).

Familial predisposition syndromes: Familial predisposition syndromes are associated with increased lymphoma such as Ataxia Telangiectasia, Klinefelter's Syndrome, Wiskott Aldrich Syndrome, Chediak-Higashi Syndrome amongst many others often associated with defects in DNA repair, aging mechanisms and/or immune deficiencies (Szymd 2021).

Autoimmune Disorders: Many autoimmune disorders are associated with a risk of lymphoma, including rheumatoid arthritis, systemic lupus erythematosus, Sjogren's syndrome and Crohn's disease (Smedby 2008).

Immune Suppression: Congenital, infectious or iatrogenic immune suppression including patients with HIV, transplant patients in immune suppression (Gruhlich 2007). Some infectious agents can be associated with increased risk such as EBV, HTLVIII, Herpes 8, Heliobacter pylori, hepatitis C and tuberculosis (Engels 2007).

Exposure to Radiation and Chemotherapy: Prior treatment with chemotherapy or radiation therapy and radiation exposure can increase the risk of NHL (Harbon 2020).

Exposure to chemical carcinogens: The exposure to chemical carcinogens, such as benzene (Smith 2007, Vlaanderen 2010, Steinmaus 2015,Rana 2021, Ge 2024, Bassig 2024) trichloroethylene (also for benzene and TCE see as described in detail further elsewhere in this report) (see for example NTP 2015), formaldehyde (see for example Catalina 2019), glyphosate (see for example Zhang 2021, Davoren 2018) and ethylene oxide (see for example: IRIS 2016), as well as many other chemical agents, such as pesticides (Cavalier 2021), or other environmental exposures can cause NHL (Francisco 2023).

Other risk factors include the use of some types of ***breast implants*** (Kricheldorff 2018); obesity (Lichtman 2010, Skibola 2007, Willet 2008); ***diabetes***, (for example: Xu 2019); and ***NHL survival*** (Han 2023). The exposure to **tobacco smoke** may be associated with NHL, but evidence is conflicting. (for example: Diver 2012, Taborelli 2017, Schollkopf 2005).

Hematopoietic cancers and lymphohematopoietic cancers are associated with genetic events that contribute to their etiology. CLL is almost always a disease of mature B cells preceded by monoclonal B cell lymphocytosis (MBL) leading to the clonal expansion of CD5 positive mature B cells in the peripheral blood, bone marrow, and lymph nodes. Like other hematopoietic and lympho-hematopoietic cancers, CLL is often driven by impaired cell apoptosis and increased cell proliferation secondary to genetic abnormalities. Around 80% of CLL tumors have at least one of four common chromosomal abnormalities. These cytogenetic abnormalities include del(13q14), Trisomy 12, del(11q22-23), and del(17p12).

CLL is associated with chromosomal abnormalities and mutations (Shadman, 2023, Kumar 2023, Montague 2023). The most common chromosomal abnormality in CLL is del(13q14) that is seen in 50 to 60% of CLL tumors and contains two important microRNAs: miR15A and miR16A. These microRNA molecules regulate apoptosis and the normal cell cycle. Additionally, miR16A and miR15A can upregulate BCL2. The second most common chromosomal abnormality, Trisomy 12, is found in a10 to 20% of CLL tumors and activates RUNX3. Del(11q22-23) has been found in 10 to 20% of CLL tumors that contains the ataxia-telangiectasia (ATM) gene that is important for DNA repair. Other gene mutations contribute to CLL including DNA damage (TP53, ATM, POT1), chromatin modifiers (ASXL1, SETD2, HIST1H1B, HIST1HIE, BAZ2A, ZMYM3, SYNE1, CHD2, ARID1, KMT2D), RNA splicing and metabolism (XPO1, MED12, SF3B1,

CNOT3, U1, FUBP1, DDX3X, RPS15, ZNF292). Genes involving microenvironment-dependent signaling include the MAPK-ERK pathway (PTPN11, MAP2K1, KRAS, BRAF, NRAS), Notch signaling (FBXW7, NOTCH1), NF-kB signaling (EGR2, TRAF2, TRAFR3, NFKB2, NRKBIE, BIRC3, NKAP), and B cell receptor (BCR) and Toll-like receptor signaling (IRF4, BCOR, TLR2, KLHL6, IRAK1, PAX5, MYD88).

Notably, CLL specifically has been associated with both familial (hereditary) and environmental risk factors (Shadman, 2023, Kumar 2023, Montague 2023). Occupational exposure to certain chemicals, radiation exposure, and exposure to tobacco has been associated with CLL, although the evidence on smoking is conflicting. Farming, rubber manufacturing industries and workers exposed to benzene and other chemical solvents increase the risk of CLL/SLL. Uranium miners exposed to ionizing and non-ionizing radiation have increased CLL incidence. Tobacco users and cigarette smokers have an elevated risk of CLL. The VA recognizes exposure to Agent Orange and other herbicide exposure during military service as a risk factor for CLL, despite the fact that to date epidemiology studies do not necessarily support a causal relationship. Like other cancers, CLL can have more than one significant contributing cause. Like other cancers, CLL requires many steps that occurs over a period of time and involves initiation, progression and therapeutic resistance.

In general, for hematopoietic and lympho-hematopoietic cancers such as NHL there are risk factors that are generally relevant even when there are some risk factors are specific to subtypes (for discussion see both Wang 2010; Wild 2020). Indeed, it is generally accepted often for purposes of generally evaluating potential risks, that for lympho-hematopoietic and NHL cancers, that these cancers can be grouped together, as has been commonly accepted scientific methodology for epidemiological studies, as well as for analyses performed and reported by scientists at the EPA, IARC and other scientific and government organizations that assess carcinogenesis and carcinogenic risk.

V. SCIENTIFIC EVIDENCE UNDERLYING OPINON

I incorporate by reference my general causation report, including all of the opinions contained therein and the materials considered list, submitted in this case.

I have performed an integrative analysis to examine and conclude based upon weight of evidence and examination of epidemiological, preclinical and mechanistic data that the exposure to benzene, TCE, VC and PCE can be a cause of hematopoietic cancers including leukemia, lymphoma, lympho-hematopoietic cancers and NHL (see Felsher, General Causation Report, Leukemia and NHL).

VI. BRUCE HILL MEDICAL DIAGNOSIS AND CAUSATION

A. Medical Summary

1. Medical History

Chaplain Bruce Hill is a 72-year-old African American male who is a veteran of the United States Navy and was based on Camp Lejeune for 24 months (.31.1983 through 6.01.1985) and was exposed to VOCs from July 1983 through March 1965. While serving in the Mavy at Camp Lejeune, Chaplain Hill was exposed to several toxic chemicals including trichloroethylene (TCE), tetrachloroethylene (PCE), benzene and Vinyl Chloride (VC) which unfortunately led to a diagnosis via flow cytometry of B-Cell chronic lymphocytic leukemia (CLL) in 2004 at the age of 52. He has received multiple rounds of chemotherapy,

radiation, and IVIG from 2004 through 2023 for treatment of CLL. Chaplain Hill has been in an immunocompromised state requiring repeated Emergency Department visits and hospitalizations for infections sustained due to his compromised immune system.

Chaplain Hill started having leukocytosis in September 1998 and was formally diagnosed in August 2004 with B-Cell chronic lymphocytic leukemia (CLL). Flow cytometry was interpreted as B cell chronic lymphocytic leukemia involving 60% of leukocytes and no overt evidence of large cell transformation. His WBC slowly increased over 3 years (WBC/lymphs: 2001 -> ~12K/70%, 8/2003 -> ~19K/93%, 6/2004 -> ~30K/85%, 8/2004 ~40K/95%). A CT of the pelvis on August 16, 2004, showed thoracic, axillary, abdominal, and pelvic lymphadenopathy consistent with the patient's history/diagnosis of CLL. As there was no evidence of visceral metastatic disease, he was regarded as being Stage 1. At that time, his plan of care was observation.

Over the ensuing months, Chaplain Hill's disease progressed and on May 31, 2005, the decision was made to initiate chemotherapy. He was administered four cycles of Fludarabine and Rituximab through September 2005 with a positive response. Of note, there were delays in his treatment due to myelosuppression. By October 2005, his white blood cell count was 3800 with 43% lymphocytes. He remained in remission for approximately two years, but by 2008, his white blood cell count began to climb once again. Because he remained at a low stage, no additional therapy was initiated until January 2009, at which time his second round of chemotherapy was initiated. He received three cycles of Pentostatin, Cyclophosphamide and Rituxan to April 2009 and then given 18 rounds of radiation to the axillae of 36 Gy. However, by January 2010, increased lymphadenopathy was again noted. A CT scan dated January 4, 2010, showed increased splenomegaly at 17 cm as well as an increase in the size and number of subdiaphragmatic nodal chains. A bone marrow biopsy in February 2010 showed a rapid recurrence of CLL with a predominance of lymphocytes. There were single-cell cytogenetic abnormalities but no recurrent cytogenetic abnormalities. In February 2010, because of bulky adenopathy and scrotal and leg edema, he was treated with a second salvage therapy (regimen #3) consisting of Bendamustine and Rituximab. He tolerated this less well but did notice after the first cycle a reduction in the scrotal and leg edema. He received a second cycle of Bendamustine and Rituximab in April 2010. He again noted increasing adenopathy in December 2010. On December 21, 2010, the white blood cell count had increased to 36,000 with an increase to 86% lymphocytes. His platelet count was reduced to 50,000 and hematocrit to 36.9. A CT scan showed diffuse bulky adenopathy and marked splenomegaly. In January of 2011, flow cytometry showed a lambda clonal B-cell population co-expressing CD5 and CD23 with dim CD20. CD38 was positive, while Zap-70 was negative. Conventional cytogenetics were largely normal with one cell showing non-clonal abnormalities. Prolymphocytes were found to comprise about 20-25% of tumor lymphocytes. Chaplain Hill then went on to complete 4 cycles of Rituxan, Mitoxantrone, and Bendamustine from January 2011 through April 2011. Chaplain Hill also received ~30gm IVIG over those same months for low immunoglobulin.

In April 2011, imaging studies showed that he had an excellent response with marked shrinkage of lymphadenopathy with no evidence of bulky adenopathy. Chaplain Hill's VA oncologist felt that he had reached a state of excellent partial remission and requested that he be considered for a haploidentical hematopoietic stem cell transplant (haplo-alloTx). In June of 2011, an extensive search of both family members and the National Marrow Donor Program for adult volunteer donors and cord blood donors was conducted. Unfortunately, no suitable match was identified. However, his son was found to be a half match, and it was felt the haplo transplant could proceed. However, Chaplain Hill decided it was not the

time for a transplant as his wife had been diagnosed with progressive breast cancer and he needed to be her caregiver.

Chaplain Hill decided in June 2011 not to pursue further chemotherapy as he encountered increasing pain and fatigue and longer lasting neutropenia from chemotherapy. At a follow up visit with his oncologist in August of 2011, Chaplain Hill reported feeling well, but his WBC was found to be low at 1.98 with an ANC of 120. He was again sent for a bone marrow biopsy which revealed no evidence of recurrent CLL. Rituxan was suspected as the cause. His neutropenia then recovered spontaneously without intervention. Chaplain Hill had a Bone Marrow Aspiration and Biopsy on October 24, 2011, which showed normocellular bone marrow, (35-40%) with minimal involvement by chronic lymphocytic leukemia, (5-10% of total marrow cellularity). His count seemed to be recovering on its own.

In January of 2012, Chaplain Hill's platelets dropped from >200k to 94k. Due to complaints of vague discomfort in his left upper abdominal quadrant with early satiety, labs were repeated and a CT scan ordered, which was concerning for splenomegaly and mild LAD in the cervical/axillary region. During this time, Chaplain Hill had a colonoscopy with polyp biopsy which reported a submucosal lymphoid aggregate with features consistent with CLL. He delayed chemotherapy due to travel. Chaplain Hill refused a bone marrow biopsy at that time, as he was grieving his wife's death from breast cancer. He agreed to chemotherapy and completed 2 cycles of Rituxan, Cytoxan, & Fludarabine between June 2012 and July 2012. Cycle 3 was held due to neutropenia (ANC 430).

In August 2012, Chaplain Hill developed severe body aches and bone pain along with a fever (WBC 1.77 and ANC 550). A CT showed improvement in the size of all lymph nodes and spleen, particularly in the mediastinum, when compared to the prior examination. Prolonged neutropenia was felt to be due to prolonged bone marrow recovery after previously being treated with Fludarabine and Pentastatin. As his bone marrow needed time to recover, Cycle 3 was held while he continued under observation.

In March 2013, Chaplain Hill was admitted to the hospital for infection. A CT and PET scan revealed multiple enlarged lymph nodes located both above and below the diaphragm. He was discharged home with a plan to follow-up with Oncology on an outpatient basis. He received seven cycles of Rituximab & Lenalidomide from May 2013 through December 2013 and 35mg of IVIG over the course of several months. During this period, he was admitted for severe back pain. A CT showed increased size of lymph nodes in the back, but no evidence these were causing his pain. It was felt his back pain was most likely due to over exertion.

Chaplain Hill reported to the Emergency Department on January 14, 2014, with complaints of pruritus, which was felt to be caused by a drug reaction. Lenalidomide was stopped at that time. In April 2014, Chaplain Hill complained of abdominal swelling and leg swelling. A CT scan showed bulky conglomerate abdominal adenopathy in the upper abdomen, retroperitoneal, mesenteric, and pelvic areas. His spleen had increased in size to 11.2 x 19.4 cm while his liver had increased to 19.5 cm. Mr. Hill then consented to Ibrutinib (420mg PD OD) with continued IVIG treatments. He continued the Ibrutinib regime from May 13, 2014, through July 17, 2019. IVIG (35-40 gm x51 cycles) treatments continued through January 13, 2023.

Mr. Hill suffered from mild ALP elevation in 2019 which was found to be secondary from the Ibrutinib treatment (07/05/2019: AST/ALT 62/97, 07/15/2019: AST/ALT 51/83). Chaplain Hill preferred to hold Ibrutinib due to transaminitis for one month and recheck labs. He was then monitored by his oncologist

and primary care physician. In 2019, Chaplain Hill maintained his IVIG treatments every 2-3 months (1.2019 IgG-736, 3.2019 IgG-736, 7.2019 IgG-619, 8.2019 IgG-<480, 11.2019 IgG-497). In August 2019 labs revealed elevated liver enzymes. A hepatitis screening was negative. An abdominal ultrasound was interpreted as showing persistent hepatomegaly, gallbladder polyp, and a normal spleen. An abdominal ultrasound in December 2019 showed unchanged results from the prior study with no signs of cholecystitis. His CLL continued to be asymptomatic and the transaminitis resolved.

On January 6, 2020, Chaplain Hill reported to the Emergency Department with complaints of abdominal pain. A CT scan showed no acute abnormalities, and he was ultimately diagnosed with gastroenteritis. Ibrutinib continued to be held and IVIG treatments (2.2020 IgG-909, 8.2020 IgG-516) were completed and his CLL remained asymptomatic. In October 2020 he presented to the Emergency Department complaining of vertigo, nausea and vomiting. He was treated with medication and discharged home. He reported to the Emergency Department on October 3, 2020, and again on December 21, 2020, complaining of positional vertigo. He was treated in the ED with medication and discharged home. On December 29, 2020, he visited the Emergency Department complaining of periumbilical pain. He was diagnosed with multiple non-bleeding duodenal ulcers via ERCP, direct hyperbilirubinemia and cholecystitis. He was treated with medications and discharged home.

In 2021, his IVIG cycles continued (2.2021 IgG-584, 8.2021 IgG-739). The Ibrutinib treatments were held as his CLL continued to be asymptomatic. In February 2021, he presented to the Emergency Department with abdominal pain and was diagnosed with possible congestive hepatopathy and cholelithiasis. He followed up with general surgery in March and discussed the possibility of choledocholithiasis vs. biliary colic being the cause of his symptoms. He was offered a cholecystectomy, but Chaplain Hill decided on definitive management of cholelithiasis. He underwent an echocardiogram in May 2021, which showed an estimated LVEF of 55-59%.

Chaplain Hill's CLL continued to be asymptomatic in early 2022, but did unfortunately acquire COVID. His IgG was 625 and it was determined at that time no further treatment was required. He presented to the Emergency Department in August and October 2022 for nasal and upper respiratory congestion, postnasal drip, and sinus pressure. He was treated with medication and discharged home in both instances. In November 2022, he had a colonoscopy with removal of one polyp. The colonoscopy further revealed diverticulitis in the sigmoid colon. He was also seen by a urologist in November 2022 for follow up of his BPH diagnosis at which time his medications were adjusted.

CT scans obtained in March 2023 and June 2023 showed significant progression of lymphadenopathy and splenomegaly. He was started on Zanubrutinib 160 mg BID in July 2023. On July 11, 2023, Chaplain Hill underwent a cystoscopy and urethral calibration. Chaplain Hill presented to the Emergency Department on September 7, 2023, and again on September 8, 2023, with complaints of a runny nose and sore throat. He was tested for COVID with negative results and discharged home in stable condition with a diagnosis of sinusitis and prescriptions for Medrol Dosepak, Augmentin, and Sudafed.

On January 17, 2024, a CT scan of the chest/abdomen/pelvis, was interpreted as showing an interval treatment response with a marked decrease in the size of the thoracic, abdominal, and pelvic adenopathy with a decrease in the size of the spleen. No new adenopathy was identified. A couple of tiny lung nodules were identified with attention to follow-up recommended.

On May 10, 2024, Chaplain Hill was seen by Urologist Dr. Frederick Leach, for follow-up of urethral stricture status post direct vision internal urethrotomy (DVIU) procedure and occlusive middle lobe of the prostate. Dr. Leach advised Chaplain Hill that he needed to be off chemotherapy for at least three months prior to the recommended Photo selective Vaporization of the Prostate (PVP) procedure.

A CT scan of the abdomen and pelvis performed June 4, 2024, was interpreted as showing continued improvement of the lymphadenopathy in the abdomen and pelvis. Similar small nodes in the mediastinum were present, but none enlarged by size criteria. When compared with the previous January 17, 2024, CT scan, interval development of an elongated area of ground glass opacity was seen in the right lower lobe, thought to likely be due to an infectious or inflammatory process. A small 4 mm nodule in the right lower lobe was unchanged from the previous exam. A CT scan obtained on June 4, 2024, was interpreted as showing continued improvement of lymphadenopathy in the abdomen and pelvis. Similar small lymph nodes were noted in the mediastinum, but none were deemed enlarged by size criteria. Other findings remained unchanged from the previous CT scan.

Chaplain Hill was hospitalized between June 30, 2024, to July 6, 2024, for salmonella colitis and bacteremia-salmonella. An echocardiogram obtained July 3, 2024, was reported as showing normal systolic function and wall motion with normal diastolic dysfunction. No obvious signs of valvular abnormality or vegetation were present. A repeat CT showed some colitis and lymphadenopathy from his CLL. A CTA performed July 3, 2024, was interpreted as showing mild hazy opacities in the medial right lower lobe suspected to reflect atelectasis or scarring. A 3 mm nodule was noted in the right lower lobe. Clinical correlation and short-term follow-up was recommended to assess for changes. The CTS also revealed abnormal wall thickening within the descending colon, sigmoid colon, and rectum suggestive of a nonspecific colitis. Zanubrutinib was held while inpatient, but IVIG infusions continued. Chaplain Hill was discharged home with instructions to follow-up with Oncology.

At a follow-up visit with Oncology on July 10, 2024, Chaplain Hill reported significant weakness and requested a break in his chemotherapy treatment. His ECOG Performance status was rated at ECOG 1- (Restricted in physically strenuous activity but is ambulatory and able to carry out work of low activity or sedentary nature). A white blood cell count obtained July 4, 2024, included in the Oncology note shows a white blood cell count of 4.10 with a lymph percentage of 19.8 (L). Oncology opted to hold Zanubrutinib for 6 months and repeat imaging at that time. Zanubrutinib was voluntarily discontinued July 2024 after dramatic response to treatment due to patient preference and infection. IVIG treatments continued every 6 weeks consisting of 40 gm IVIG (Gammunex). Frequency was increased from every 8 weeks to every 6 weeks, as his IgG had been running a little low.

On November 14, 2024, Chaplain Hill presented to the Emergency Department with complaints of severe upper abdominal pain. Imaging was obtained and a heart catheterization performed, which reportedly showed a mass in the left atrium attached to the interatrial area of the fossa, measuring 16 x 14 mm, felt to be a cardiac myxoma. In light of these findings, previously planned prostate surgery was postponed, pending further assessment and treatment of the cardiac mass. Chaplain Hill is currently scheduled to see a cardiac surgeon about the cardiac mass in February 2025.

Chaplain Hill continues to suffer from recurrent and chronic sinus issues, reporting a new bout of sinusitis with onset around Christmas 2024 and continuing to the present (January 2025), for which he is currently prescribed Amoxicillin and Tussin cough syrup. Currently, he remains off chemotherapy and awaits follow-up for the cardiac mass.

Chaplain Hill's medical history with respect to the course of his CLL reveals that he has had periods of remission, followed by refractory disease with numerous complications related to both his underlying CLL as well as the treatments for his disease. As an example, Chaplain Hill's CLL and treatment has resulted in hypogammaglobulinemia as well as a profoundly immunocompromised state, leading to recurrent infections. In addition, he has sexual dysfunction that is related to CLL and its treatment.

2. Past Medical History

His past medical history includes chronic tonsilitis (1977-present), benign prostatic hypertrophy (2004-present), erectile disfunction (2008-present), genital herpes (2014-present), anemia (hyperbilirubinemia, neutropenia (1998-present)), arthralgias (chronic lower back pain (2002-present)), and hypothyroidism (2003-present).

3. Family History

He has no history of hematopoietic cancers within his family. Chaplain Hill does have a family history of cancer: mother- pancreatic/stomach cancer and brother- stomach/colon cancer.

Parents

- **Freddie Hill** (mother)
  - Died in 1997 from pancreatic cancer *(Hill Depo Tran p 100 erroneously states cervical cancer; RN Interview 1/11/2025 confirmed mother with pancreatic cancer)*
  - Occupation: "domestic worker" *(Hill Depo Tran p 117)*
- **Curtis Hill** (father)
  - Died in 1977; CHF *(Hill Depo Tran p 100)*
  - Occupation: sharecropper *(Hill Depo Tran p 117)*

Grandparents

- Bruce is unaware of the medical history of his grandparents *(Hill Depo Tran p 102)*

Siblings

- Bruce Hill is the "baby of 14" – *(Hill Depo Tran p 106)*
  - **James Lee Hill** – (oldest brother); deceased *(Hill Depo Tran p 107)*
    - Died from hepatitis? *(Hill Depo Tran p 109)*
  - **Joe Lee Hill** – (brother); deceased *(Hill Depo Tran p 107)*
    - Died in 1991 *(Hill Depo Tran p 108)*

- Prostate cancer? *(Hill Depo Tran p 109)*

- **Hayward Franklin** (brother); deceased *(Hill Depo Tran p 109)*
  - Died of liver cirrhosis; died in his late 60s *(Hill Depo Tran p 110)*
- **Curtis Hill Jr.** (brother); deceased *(Hill Depo Tran p 110)*
  - Died early 2000 of Heart Failure *(Hill Depo Tran p 110)*
- **Ola May Atkins** (sister); deceased *(Hill Depo Tran p 110)*
  - Died of "Heart Attack" *(Hill Depo Tran p 111)*
- **Nathaniel Hill** (brother) – alive
  - Lives in Logansport, Louisiana *(Hill Depo Tran p 111)*
  - Health Conditions known to Bruce: Asthma (*Hill Depo Tran p 112)*
- **Robert Hill (brother) – deceased**
  - **Died** in truck accident *(Hill Depo Tran p 112)*
- **Jessie Hill** (brother) – deceased
  - Died of COVID *(Hill Depo Tran p 113)*
- **Ernestine Hill – deceased**
  - Died of "Heart Attack" *(Hill Depo Tran p 113)*
- **Bazemore Hill** – deceased
  - Died of heart attack *(Hill Depo Tran p 114)*
- **Annie Hill** (sister) – alive (79) *(Hill Depo Tran p 114)*
  - Lives in Shreveport, Louisiana
  - Health Conditions: Acid Reflux *(Hill Depo Tran p 114)*
- **Juanita Lloyd** (sister) ; deceased *(Hill Depo Tran p 115)*
  - Cause of death unknown *(Hill Depo Tran p 115)*
- **Dorothy Waines** (sister); alive
  - Lives in Keachi, Louisiana *(Hill Depo Tran p 115)*

Spouse & Children

- Mr. Hill's wife – **Bernice Elizabeth Hall**; **deceased**
  - Married in 1978 - *(Hill Depo Tran p 91)*
  - Bruce's Children with Bernice Elizabeth Hall *(Hill Depo Tran p 92)*
  - **Kristie Renice Hill (daughter); alive**
    - Kristie Lives in Gainesville, Florida *(Hill Depo Tran p 93)*
    - DOB [redacted]/1979 *(Hill Depo Tran p 93)*
    - Chron's Disease *(Hill Depo Tran p 97)*
    - Kristie has a daughter – Jenesis Dye *(Hill Depo Tran p 98)*
    - Kristie's daughter has eczema *(Hill Depo Tran p 99)*
    - Born in 2008 *(Hill Depo Tran p 100)*
    - Jenesis diagnosed with anxiety *(Hill Depo Tran p 100)*
  - **Nathan Earl Hill (son); deceased**
    - Born/died in 1982 *(Hill Depo Tran p 94)*
    - Premature birth = cause of death *(Hill Depo Tran p 94, 99)*
  - **Anthony Levar Hill (son); alive**
    - Anthony lives in Gainesville, Florida *(Hill Depo Tran p 94)*
    - DOB [redacted]/1983 *(Hill Depo Tran p 94)*
    - Anthony is married with children *(Hill Depo Tran p 95)*
    - Anthony's wife is Jazmine Hill *(Hill Depo Tran p 95)*
    - Anthony's Children *(Hill Depo Tran p 95-96)*
      - Malachi Hill
      - Micaiah Hill
      - Zila Hil
  - Mr. Hill has a son with Mary Helen Peterson *(Hill Depo Tran p 96)*

- Mr. Hill and Ms. Peterson's son is **Marcus Dwayne Peterson** *(Hill Depo Tran p 96)*
  - Marcus was born in [redacted] 1970 *(Hill Depo Tran p 97)*
  - He lives in Arizona and has 2 children *(Hill Depo Tran p 96)*
- No children have leukemia *(Hill Depo Tran p 98)*
- No grandchildren (6) /great grandchildren (3) have medical conditions *(Hill Depo Tran p 98)*
- Autoimmune disease history in family
  - Kristie (daughter) has Chron's Disease *(Hill Depo Tran p 98)*
- Niece with cancer; breast cancer
  - Thelma Mims *(Hill Depo Tran p 98)*
- Niece with cancer; breast cancer
  - Gwendolyn Grave *(Hill Depo Tran p 104-105)*
- Nephew died from cancer
  - Clifford Hill (son of brother Curtis Hill) *(Hill Depo Tran p 100)*
- Neice died of COVID *(Hill Depo Tran p 105)*
  - Linda Faye Atkins *(Hill Depo Tran p 106)*
- Nephew died of cirrhosis of the liver *(Hill Depo Tran p 106)*
  - Douglas Hill *(Hill Depo Tran p 106)*
- No family history of leukemia *(Hill Depo Tran p 97)*

4. Social History

Chaplain Hill is a non-smoker, does not consume alcohol, and is not a substance abuser.

- Socioeconomic Information – Retired from Navy; Retired from Florida State Correctional Facility; Supports self independently
- Education Level – Master of Divinity, 1983
- Occupation – Chaplain, Pastor
- Employment Status – Retired from Florida Department of Correction 2012; Retired from US Navy Reserve 2010; Retired from US Navy Active Duty 1992

- Marital Status – Married; first marriage ended in divorce *(Hill Depo Tran p 128)*;
- Parental Status –
    - Son – June 1970
    - Miscarriage – 1978
    - Daughter – November 1979
    - Son (premature; died after birth) – 1982
    - Son – September 1983
    - Mobility – Independent in activities of daily living; drives daily to town 12 miles away and medical appointments *(Hill Depo Tran p 11)*

5. Prognosis and Future Care

- Mr. Hill has an incurable neoplastic disease. After Chaplain Hill's CLL diagnosis, he has struggled with recurrent sinus and ear infections. He has shown progression of his CLL that is being further evaluated that requires ongoing medical evaluation and treatment. Mr. Hill suffers from a number of chronic conditions including: Steatosis; Chronic Kidney Disease; Hyperlipidemia; Benign Prostatic Hypertrophy; Osteoarthritis; Degenerative Joint Disease; Gastroesophageal Reflux Disease; Hypothyroidism; Hearing Loss; Herpes; Erectile Disorder; + Heliobacter Pylori; Duodenal Ulcers; Pre-Diabetes with impaired fasting glucose; Benign Paroxysmal Positional Vertigo; Allergic Rhinitis; Hypogammaglobulinemia; Ocular Hypertension; Cataracts; Neutropenia; Hemorrhoids; Low Back Pain; Diverticulitis; and Cardiac Myxoma. *(See VA Medical Records. His CLL diagnosis and his treatments over the years is* more likely than not, causative of his decreased energy, decreased functional abilities, depressed immune status and sexual dysfunction.
- Mr. Hill will require long-term treatment and management. Mr. Hill was treated with chemotherapy before receiving first generation BTK therapy, and as a result, he would be at a higher risk in the future for the development of secondary, treatment-related cancers. Due to his severely immunosuppressed condition, Mr. Hill is not a candidate for future stem cell transplantation. He will have an increased risk of premature death. He will succumb eventually to his disease, most likely from an overwhelming infection.

6. Risk Factors for CLL as Relates to Mr. Hill

In my performance of a differential etiology, I considered the medical and scientific literature as well as reviewed the medical records and deposition testimony as well as other medical records to determine whether Mr. Hill had risk factors relating to his CLL and, if so, which risk factor(s), if any, were at least as likely than not, causative of his CLL.

Mr. Hill is a male. Being male in and of itself is a risk factor but not a cause of CLL. Advanced Age is a risk factor for NHL and CLL but is not independent of any other risk factors that are not considered to be

causative. Regardless, Mr. Hill was young for a diagnosis of CLL that is consistent with his exposure environmental risk factor.

Mr. Hill also has a history of obesity. Obesity does increase the risk of many diseases including cancer, so his obesity might have increased his risk for cancer. Obesity is a risk factor for cancer and NHL, by mechanisms including causing inflammation and impeding the immune system that can make one more susceptible to cancer. However, standing on its own, or in combination with age and sex, obesity is not causal of Mr. Hill's CLL.

According to his medical records, Mr. Hill was neither a smoker nor a drinker. While the epidemiology on the relationship between smoking and CLL is conflicting, alcohol is not considered as a risk factor for CLL. In this case, smoking is not a risk factor for Mr. Hill's cancer.

Mr. Hill was exposed to radiation as part of his past medical/dental care. Routine x-rays and a CT scan would not be a likely risk factor for the development of CLL.

Mr. Hill has a family history that includes cancer, but he has no known familial susceptibility syndrome. susceptibility syndromes are not causes of cancer per se but increase the risk that exposures to environmental carcinogens would be a cause of his cancer.

Mr. Hill's occupational history included fueling aircraft for a period of time while serving on an aircraft carrier. He did wear personal protective equipment during these endeavors. While exposure to jet fuel is thought to be associated with certain cancers, CLL is not one of them. In addition, Mr. Hill's use of a mask and gloves and protective gear would have minimized any inhalation of the vapors. In my opinion, any exposure that Mr. Hill may have had to jet fuel would not be a risk factor for his CLL.

Finally, and of most significance, Mr. Keller was exposed to benzene, TCE, VC and PCE at Camp Lejeune. The combination exposure to these immunotoxic and genotoxic chemicals was more likely that not a significant contributing cause of his CLL.

<u>Radiological Procedures Prior to CLL Diagnosis:</u>

- May 25, 1994 – Maxillofacial CT scan of Paranasal Sinuses *(00028_HILL_VHA_0000000242)*
- Annual dental x-rays while in US Navy *(RN Interview 1/11/2025)*

<u>Body Mass Index (BMI) / Weight History:</u>

- BMI List *(Representative Sample)*

Date	BMI	Date	BMI
3/19/2001	31	7/29/2016	33
8/18/2003	31	11/29/2016	33
6/29/2004	31	2/28/2017	33
5/24/2005	31	6/2/2017	32
10/12/2005	33	2/26/2018	31
4/12/2006	32	7/2/2018	32

2/17/2009	30	7/8/2019	32
8/18/2009	31	2/25/2020	30
12/31/2009	31	6/30/2020	30
2/22/2010	31	10/20/2020	31
6/6/2013	33	1/19/2021	30
7/9/2013	31	2/22/2021	31
1/16/2014	31	8/11/2021	32
12/9/2014	33	4/10/2024	32
8/5/2015	32	6/30/2024	32
7/6/2016	31	7/17/2024	32

Genetic Testing:

- Chromosome Analysis 03/04/2010 *(See 00028_HILL_0000004754)*
  - Single cell nonclonal abnormalities: 46, XY, t (4;12) (q25; q12)[1] 46, Y, add(X) (q26), add (4) (q31), del (11) (q23q25), add12 (q13), add (16) (q12) [1]
  - Male karyotype with nonclonal abnormalities
  - INTERPRETATION: Within the limits of the technology utilized, no chromosome abnormalities were evident in 20 of the 22 cells examined. One metaphase cell was found to have a translocation between chromosomes 4 and 12. One other metaphase cell was found to have rearrangements of chromosomes X, 4, 12 and 16, and a deletion of chromosome 11. Twenty additional cells above the normal 20-cell analysis were partially analyzed looking specifically for evidence of a small clonal population but no additional aberrant cells were found. The significance of the nonclonal abnormal cells observed in this analysis is unclear. They may represent undetected clonal cell populations, technical artifact, or random events. Their presence has been noted for future studies.

Occupational History:

Time Period	Occupation
Teenager	Worked in the hay field, pulp wood, fixing fences, raking leaves *(Hill Depo Tran p 117);* worked 2 months as a painter's helper following high school graduation painting outside of homes *(RN Interview 1/11/2025)*
1972 – 1974	US Navy - Aviation Boatswain's Mate/Airman; Division Yeoman/Administrative Secretary *(Hill Depo Tran p 121-122)*
1974 – approx. 1976	Weiner's Corporation – Assistant Manager of a shoe store *(Hill Depo Tran p 127)*

July 1976 - August 1976	US Navy – Reorientation and Reenlistment *(Hill Depo Tran p 127-128)*
September 1976 – November 1978	US Navy, USS Guam - Aviation Boatswain's Mate; Damage Control Petty Officer; Division Training PO *(Hill Depo Tran p 128-129) (00028_HILL_0000011790)*
1978 – May 1983	Full Time Student *(Hill Depo Tran p 131)* US Navy Reserves *(Hill Depo Tran p 130-132)*
May 1983 - 1992	US NavyC – Chaplain *(Hill Depo Tran p 132 – 153)*
1992	Counselor at a Boy's Home *(Hill Depo Tran p 166)*
1992 – 2010	US Navy Reserve – Chaplain *(Hill Depo Tran p 154)*; Retired 2010
1993 – 2000	Church Pastor *(Hill Depo Tran p 166-167)*
March 2000 – 2012	Florida Department of Corrections – Chaplain, Lake Butler Florida – Reception and Medical Center *(Hill Depo Tran p 59, 163-164)*; 2004-2012 reported frequent absences from work due to CLL treatment *(Hill Depo Tran p 52)*; Retired 2012 *(Hill Depo Tran p 51)*
2012 – Present	Retired *(Hill Depo Tran p 51)*

Other environmental history of potential relevance:

- Fueled aircrafts while stationed on the USS Independence; aircraft fuel
  - PPE worn while fueling aircrafts – helmet, life vest, gloves, and mask *(Hill Depo Tran p 124)*
- Fueled aircrafts while stationed on the USS Guam *(Hill Depo Tran p 128)*
  - Duration 1976 – November 1978
- Mr. Hill has no recollection of exposure to chemicals during his lifetime through employment or otherwise *(Hill Depo Tran p 149)*
- "**Q.** Other than Camp Lejeune, do you recall any exposure to chemicals in your lifetime through employment or otherwise? **A.** I can't recall, you know, but I don't know what all they had at the -- when I used to do my pastor visitation at the motor pools and things." *(Hill Depo Tran p 149)*

7. Damages

Mr. Hill has both short-term and long-term consequences and risks related to his CLL and treatment. First, CLL is not a curable disease. As previously mentioned, Mr. Hill has had refractory episodes of CLL despite aggressive and appropriate treatment. Second, all CLL patients are at further risk of additional cancers. Given Mr. Hill's treatment of CLL with chemotherapy, the risk of future cancer is increased. Third, CLL patients have a high risk of infection that are the most common cause of death. Mr. Hill's medical history is replete with examples of infections that are related to his immuno-compromised state, including a recent salmonella infection in 2024 which required hospitalization. Fourth, CLL patients have suppression of bone marrow function and have lethargy and reduced energy. As previously noted, Mr. Hill was diagnosed with hypogammaglobulinemia, and fatigue, diarrhea, sinus infections and ear infections are all associated with this condition. Fifth, Mr. Hill has sexual dysfunction that is related to his diagnosis and treatment of CLL.

B. Exposure History

1. Time at Camp Lejeune

Mr. Hill testified in his April 9, 2024, deposition that he first arrived at Camp Lejeune in May 1983, and remained there until his departure in June 1985; a total of approximately 24 months. In addition to this time, Mr. Hill further reported he was at Camp Lejeune at least twice more for Marine Corps training, outside of the 24 months previously referenced. While living in Officer's Housing in Paradise Point on Camp Lejeune between December 1983 to June 1985, Mr. Hill reportedly was exposed to the water in his activities of daily living.

During this 24-month period, he was assigned as Battalion Chaplain and worked out of the Battalion Headquarters and Chapel in the French Creek area of Camp Lejeune where his Chaplain duties required him to visit locations all over the base. He ate lunch at the Officer's Club once per week but visited the Club 2-3 times per week due to professional development training, and other activities. Other places he frequented included restaurants on the base, the PX (Exchange), the Commissary, the Naval hospital, and the gym, which he visited 2-3 times per week to exercise. Mr. Hill also reports occasional use of the swimming pool at Camp Lejeune both for recreational and training purposes. As a Chaplain, Mr. Hill went out into the field with the Marines in his Battalion for Marine Corps indoctrination training and visited the troops weekly in the barracks at French Creek.

Residential History:

Birth – Age 18	Born in Shreveport, Louisiana; Raised in Longstreet, Louisiana; moved to Shreveport, Louisiana prior to enlisting in Navy *(Hill Depo Tran p 118)*
1971-1974	US Navy – Active Duty; Enlisted
October 13, 1971 – approx. January 1972	Orlando Florida, US Navy Basic Training; approximately 8 weeks *(Hill Depo Tran p 118-119)*
**	Millington Tennessee, Fleet Prep School; approximately 3 weeks *(Hill Depo Tran p 118)*

**	Norfolk, Virginia; approximately 32-34 months lived aboard aircraft carrier, USS Independence; includes a 7-month deployment to Europe *(Hill Depo Tran p 121-122)*
1974-1976	Shreveport, Louisiana *(Hill Depo Tran p 126)*
1976-1978	US Navy – Active Duty; Enlisted
July 1976	Approximately one month in Chicago, Illinois; US Navy Re-Orientation *(Hill Depo Tran p 127)*
August 1976 – November 1978	Norfolk Virginia; lived aboard the USS Guam; one 6-7 month deployment to the Mediterranean; two deployments to North Atlantic *(Hill Depo Tran p 127-129)*
November 1978 – May 1980	Petersburg, Virginia *(Hill Depo Tran p 130)*
May 1980 – May 1983	Richmond, Virginia *(Hill Depo Tran p 131)* Summer spent in Newport, Rhode Island, July – August 1984 *(Hill Depo Tran p 132)*
May 1983 – 1992	US Navy; Active Duty – Officer
May 1983 - January/February 1984	Apartment (while awaiting base housing) Jacksonville, North Carolina *(Hill Depo Tran p 133-134)*
January/February 1984 – June 1985	Officer's Housing Camp Lejeune, North Carolina *(Hill Depo Tran p 131, 134)*
June 1985 – Fall 1987	Naval Air Station, Meridian, Mississippi *(approx. 27 months); (Hill Depo Tran p 151)*
Fall 1987 – December 1989	Norfolk Virginia, lived onboard USS Savannah *(approx. 24-27 months);* one cruise to the Mediterranean, one cruise to the North Atlantic *(Hill Depo Tran p 152-153)*
January 1990 – November 1991	Okinawa Japan *(Hill Depo Tran p 152-153)*
1992	Naval Air Station, New Orleans, Louisiana *(Hill Depo Tran p 53)*
1992 – 2010	US Navy; Reserve Duty – Officer

1993-2000	Augusta, Georgia *(Hill Depo Tran p 166-167)*
	- *1993:*
	- *1994: 1922 Driftwood Drive, Augusta, GA 30909*
	  ○ *(00028_HILL_0000011801)*
	- *1995:*
	- *1996: 1922 Driftwood Drive, Augusta, GA 30900*
	  ○ *(00028_HILL_0000011797)*
	- *April – May 1998 for 3 weeks – Bethesda, Maryland (RN Interview 1/11/2025)*

2000 to 2009	Gainesville, Florida *(Hill Depo Tran p 28, 167)* - ***2000**: 2056 NW 55th Blvd. Apt. C3, Gainesville, FL 32653* ○ *00028_HILL_0000011793* - *2001:*

	- *2002:*
	- ***2003:*** *2056 NW 55th Blvd. Apt. C3, Gainesville, FL 32653*
	  ○ *(00028_HILL_0000011805)*
	- *2004:*
	- ***2005:*** *2056 NW 55th Blvd. Apt. C3, Gainesville, FL 32653*
	  ○ *(*00028_HILL_0000011803)
	- *2006:*
	- *2007:*
	- *2008:*
	- *2009:*

2009 to Present	485 SW Donovan Glen Lake City, Florida *(Hill Depo Tran p 28)*

Camp Lejeune Water Contamination

I have reviewed the Expert Report by Morris L. Maslia dated October 25, 2024. According to pages 91-92 of the report:

> **For the Hadnot Point water treatment plant (HPWTP) service area:**
>
> The reconstructed contamination of finished water exceeding the current maximum contaminant level (MCL) for TCE was 374 months (August 1953–January 1985) (Table 7.14). With the onset of pumping at well HP-651 during July 1972, the concentration of TCE in well HP-651 affected the resulting finished-water concentrations of TCE at the HPWTP, which exceeded 750 µg/L during November 1983 (Table 7.16). Measured TCE concentrations in finished water at the HPWTP during the period May 1982 through February 1985 ranged from 1.2 µg/L to 1,400 µg/L (Table 7.15).
>
> The reconstructed contamination of finished water exceeding the current MCL for PCE was 114 months (August 1974–January 1985) (Table 7.16), also a consequence of the onset of pumping of well HP-651. The maximum reconstructed finished-water concentration of PCE was about 40 µg/L during November 1983 (Table 7.14). Measured PCE concentrations at the HPWTP ranged from below detection limits (1–10 µg/L) to 100 µg/L during the period May 1982–February 1985 (Table 7.16).
>
> The reconstructed duration of contamination of finished water exceeding the current MCL for benzene was 63 months (January 1979–November 1984) (Table 7.16); the maximum reconstructed finished water concentration of benzene was about 12 µg/L during April 1984 (Table 7.16). Measured benzene concentrations at the HPWTP ranged from below detection limits (10 µg/L) to 38 µg/L during the period December 1984– December 1985. An unexplained value of 2,500 µg/L of benzene was measured on November 11, 1985 (Table 7.16).

Additionally, I have examined Appendix J of the same report, which contains the ATSDR water modeling tables. These tables provide detailed monthly mean contaminant concentrations for Hadnot Point, expressed in micrograms per liter in finished water.

Hadnot Point				
Month and year	Tetrachloroethylene (PCE) Concentration [µg/L]	Trichloroethylene (TCE) Concentration [µg/L]	Vinyl Chloride (VC) Concentration [µg/L]	Benzene Concentration [µg/L]
May 1983	22	449	36	8
June 1983	27	546	45	7
July 1983	30	618	51	7
August 1983	32	659	54	9
September 1983	26	543	45	9
October 1983	5	134	9	10
November 1983	39	783	67	10
December 1983	34	688	59	9
January 1984	21	427	36	11
February 1984	27	560	47	8
March 1984	28	587	50	7
April 1984	18	400	33	12
May 1984	23	491	42	10
June 1984	22	471	41	7
July 1984	24	507	45	7
August 1984	26	539	48	8
September 1984	21	443	39	8
October 1984	3	94	6	8
November 1984	31	639	59	8
December 1984	2	43	4	2
January 1985	16	324	31	4
February 1985	0	0	0	3
March 1985	0	0	0	3
April 1985	0	0	0	4
May 1985	0	0	0	3
June 1985	0	0	0	3

In Dr. Kelly Reynolds' report, she estimated the ingestion exposure of Mr. Hill. The contaminants at Camp Lejeune, as stated above, are volatile organic compounds and as a result Mr. Hill experienced significant exposure through inhalation and dermal routes. To a reasonable degree of medical and scientific certainty that exposure increased both the total exposure as well as the cancer risk.

VII. CONCLUSION

I conclude that, to reasonable degree of medical certainly, after review of Mr. Hill's medical records, deposition testimony, other expert reports and documents and performing a differential etiology, using well established methods that are generally accepted, and after consideration and review of the medical and scientific literature, that Mr. Hill's exposure to the VOC's in the water at Camp Lejeune, including benzene, trichlorethylene (TCE) , VC and PCE from contaminated drinking water at Camp Lejeune, was at least as likely as not, the cause of his CLL.

I performed a differential etiology to determine whether Mr. Hill had risk factors potentially related to his CLL and if so, what were the specific risk factors. He is a male and being male in itself is a risk factor but is not known to be a cause of CLL and most importantly it is not an independent risk factor to environmental exposures.

He was diagnosed at age 51, which is young for a diagnosis of CLL, and is consistent with his cancer having as a substantial contributing cause an environmental exposure. He is African-American , and individuals who are African-American have a lower risk for CLL. He has some family history of cancer, but no family history of hematopoietic cancers so there is no evidence for a familial susceptibility. He has as an environmental exposure that he was involved his being exposed to VOCs (benzene, TCE, PDE and VC) in the contaminated drinking while based in Camp Lejeune that, more likely than not can be a cause of hematopoietic, lympho-hematopoietic cancers including NHL and the CLL subtypes. He does not have a known history of exposure to smoking, alcohol or radiation exposure. He is considered over-weight and this may generally increase his susceptibility to cancer and would have increased his risk upon a carcinogenic exposure. However, obesity is not likely a cause of his cancer. His cancer does have evidence for multiple genetic abnormalities that can be caused by exposure to carcinogens, including the VOCs in the contaminated drinking water at Camp Lejeune.

Chaplain Hill has an incurable type of cancer. He has suffered from knowledge of the diagnosis, from effects of the cancer on his body and from the therapy he has received. This includes that he has reduced energy and lethargy related to his cancer and has associated bone marrow suppression. He has had numerous life threating infections caused by his cancer and associated immune system dysfunction. Based on my knowledge, skill, training, experience and education, Mr. Hill will have further progression of his cancer, that will be very likely associated with life-threatening infections and further bone marrow and immune suppression associated ~~also~~ with increasing lethargy and further reduced energy.

Dean W. Felsher, M.D., Ph.D.

VIII. REFERENCES

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IX. ADDITIONAL MATERIALS CONSIDERED

Deposition Transcripts

- Deposition Transcript – Bruce Hill - April 9, 2024, and exhibits
- Deposition Transcript – Bradley Fletcher, MD/PHD (Treating Physician) – June 13, 2024, and exhibits
- Deposition Transcript – Jessica Schmit, MD (Treating Physician) – July 15, 2024, and exhibits
- Deposition Transcript – Maxim Norkin, MD (Treating Physician) – June 6, 2024, and exhibits
- Deposition Transcript – Stephen McCready, PA-C (Treating Medical Professional) – May 8, 2024
- Deposition Transcript – Kristie Hill (Fact Witness)– June 14, 2024, and exhibits

Medical Records

- VAMC Gainesville Florida
  [00028_HILL_VHA_0000000260 to 00028_HILL_VHA_0000000343]
- VAMC Gainesville Florida

  [00028_HILL_VHA_0000000345 to 00028_HILL_VHA_0000000860]
- VAMC Gainesville Florida
  [00028_HILL_0000000005 to 00028_HILL_000000001]
- James A. Haley VA Hospital
  [00028_HILL_VHA_0000000124 to 0028_HILL_VHA_0000000142]
- Overton Brooks VA Medical Center
  [00028_HILL_VHA_0000000143 to 00028_HILL_VHA_0000000183]
- North Florida/South Georgia VA
  [00028_HILL_0000000016 to 00028_HILL_000000201]

- Overton Brooks VA Medical Center
  [00028_HILL_VHA_0000000184 to 00028_HILL_VHA_0000000224]

- North Florida/South Georgia VA
  [00028_HILL_0000002016 to 00028_HILL_0000004015]

- Charlie Norwood VAMC – Augusts
  [00028_HILL_VHA_0000000225 to 00028_HILL_VHA_0000000240]

- North Florida/South Georgia VA
  [00028_HILL_0000004016 to 00028_HILL_0000004808]

- North Florida/South Georgia VA
  [00028_HILL_0000004809 to 00028_HILL_0000004813]

- UF Health Shands Hospital
  [00028_HILL_0000004865 to 00028_HILL_0000005388]

Profile Form & Short Form Complaint

- Bruce Hill Discovery Pool Profile Form, 00028_HILL_DPPF_000000001 - 00028_HILL_DPPF_0000000023

- Bruce Hill Short Form Complaint, Filed November 20, 2023.

Expert Reports

- Expert Report of Morris L. Maslia, P.E., D.WRE, DEE, Fellow EWRI - October 25, 2024

- General Causation Expert Report of Dean W. Felsher, MD, PHD. [Leukemia, and Non-Hodgkin's Lymphoma] - December 9, 2024

- General Causation Expert Report of Howard Hu, MD, MPH, SC. D. - December 9, 2024

- General Causation Expert Report of Kathleen M. Gilbert, PHD. [TCE, Non-Hodgkin Lymphoma, and Leukemia] - December 9, 2024

- General Causation Expert Report of Lukasz Gondek, MD, PhD. [Leukemia] - December 9, 2024

- General Causation Expert Report of Steven B. Bird, MD. [Hematopoietic Cancers: Leukemia & Non-Hodgkin's Lymphoma] - December 9, 2024

- Specific Causation Expert Report of Kelly Reynolds, MSPH, PhD – February 7, 2025

In addition to the materials listed here, I have considered all of the materials listed in the materials considered list that accompanied my General Causation report dated December 9, 2024, as well as all materials referenced within this report and in my December 9, 2024, General Causation report.

All facts and data listed herein are either identified by bates number or are publicly available to and accessible by Defendant United States of America.

Dr. Felsher reserves the right to review and consider additional facts, data and publications;

Dr. Felsher reserves the right to consider the report of any other witness in this action; and

Dr. Felsher reserves the right to supplement this list of reliance files.

Exhibit 1

Biographical and Bibliographic Information

Identifying Information:

Name:	Dean W. Felsher MD PhD
Citizenship:	United States of America

Academic History:

Colleges and University

9/81-7/85	University of Chicago, B.A.
7/85-7/92	University of California, Los Angeles, M.D., PhD.
7/92-6/94	Hospital of the University of Pennsylvania, Resident, Internal Medicine
7/94-6/99	University of California, San Francisco, Fellow, Hematology-Oncology

Scholarships and Honors

1985	Special Honors, Chemistry, University of Chicago
1992	Emil Bogen Research Award for Excellence in Science
1985-1992	Medical Scientist Training Program

Residency and Post-Doctoral Training

7/92-6/94	Resident, Hospital of the University of Pennsylvania, Internal Medicine
7/94-6/99	Fellow, University of California, San Francisco, Hematology-Oncology
7/95-6/99	Fellow, University of California, San Francisco, J. Michael Bishop's Laboratory

Board Certification

1996	Internal Medicine
1998	Medical Oncology

Employment History:

12/97-7/98	Clinical Instructor, Department of Medicine, UCSF
7/98-9/99	Assistant Adjunct Professor, Step I, Department of Medicine, UCSF
9/1/99-12/1/99	Acting Assistant Professor, Division of Oncology, Department of Medicine, Stanford University
12/1/99-	Assistant Professor, Division of Oncology, Department of Medicine, Stanford University
11/1/01-	Assistant Professor, Division of Oncology, Departments of Medicine and Pathology, Stanford University
2/1/07-	Associate Professor, Division of Oncology, Departments of Medicine and Pathology, Stanford University
8/01/12-	Professor, Division of Oncology, Departments of Medicine and Pathology, Stanford University.

Public and Professional Service:

Departmental Affiliations and Leadership
Associate Chief, Division of Oncology, Department of Medicine, Stanford University
Department of Pathology, Stanford University
Founding Director of Translational Research and Applied Medicine (TRAM)
Director of Oncology Research, Division of Oncology
Director of Admissions, Medical Scientist Training Program (MSTP)
Director of Advanced Residency Training Program (ARTS)
Director of Team Science, Department of Medicine
Co-Director Cancer Nanotechnology Training (C-TNT)
Co-Director KL2 Mentored Training Program
Member Stanford Comprehensive Cancer Institute
Member Molecular Imaging Program
Member Tumor Biology Training Program
Member Immunology Training Program
Member BioX Selection Committee
Member Canary Institute
Member ChEM-H

Graduate Programs

2000-	Cancer Biology, Stanford University
2001-	Immunology, Stanford University

Research and Professional Experience

7/85-7/92	Medical Scientist Training Program, UCLA
7/87-7/91	Graduate Student, MBI, UCLA, advisor: Dr. Jonathan Braun
7/92-6/94	Resident, Hospital of the University of Pennsylvania
7/94-6/97	Fellow, Division of Hematology-Oncology, UCSF
7/95-6/99	Fellow, Hooper Foundation, advisor: Dr. J. Michael Bishop
7/98-9/99	Assistant Adjunct Professor, Department of Medicine, UCSF
9/99-	Assistant Professor, Department of Medicine, Stanford University
11/01-	Assistant Professor, Departments of Medicine and Pathology, Stanford University
02/01/07-	Associate Professor, Division of Oncology, Departments of Medicine and Pathology, Molecular Imaging, Stanford University
09/01/12-	Professor, Division of Oncology, Departments of Medicine and Pathology, Molecular Imaging, Stanford Imaging
10/01/16-	Director of Research, Division of Oncology, Stanford University
07/02/18-	Director of Advanced Residency Training (ARTS)
07/01/20-	Co-Director, CTSA KL2 Mentored Training Program
07/01/20-	Associate Chief, Division of Oncology, Stanford University
07/01/20-	Director of Team Science, Department of Medicine

Clinical Experience

6/94-7/96	General Oncology, UCSF-Mt. Zion
8/96-1/98	AIDS Oncology, San Francisco General Hospital
2/99-6/15	General Oncology and Lymphoma, Stanford University

University Services

2001-2006	Internal Medicine Housestaff Selection Committee, Department of Medicine,
2001-	Center for Clinical Immunology, Steering Committee Member
2001-	Medical Scientist Training Program Admission Committee
2002-2005	Immunology Graduate Program Admission Committee
2002-	Organizer, Division of Oncology Annual Retreat
2002-	Member, Digestive Diseases Consortium, Stanford University
2002-2005	Cancer Biology Graduate Program, Executive Steering Committee
2005-	Tumor Biology Training Program, Executive Steering Committee
2005-2009	Dean's Committee on Animal Research
2005-	Member, Stanford Comprehensive Cancer Center
2005-	Faculty Co-Leader, Stanford Comprehensive Cancer Center Transgenic Core Facility
2006-	Review Panel Bio-X Interdisciplinary Research Initiative
2006-2010	Chair, Grants Committee, Stanford's Center for Children's Brain Tumors
2007-	Member, Advanced Residency Training at Stanford Program
2007-2011	Leader, Molecular Therapeutics Program, Stanford Cancer Center The development of a new program including programmatic development, an annual symposium, 3 invited speakers per year and support for joint grant applications.
2008-	Faculty Member, Molecular Imaging Program
2011-	Founding Director, Translational and Applied Medicine Program (TRAM), Department of Medicine: An integrated translational research program that I am the founding Director includes: pilot grants (15-20 funded projects per year), MED121/221 year-long training course, an TRAM Annual Symposia, 18 invited speaker , 3 educational talks, 3 workshops in bioinformatics, industry-academic interactions stem cell biology and infectious diseases, and a dedicated translational research core facility run by two senior scientists, 4 faculty advisory and 3 external advisors.
2014-	SPECTRUM Council of Mentors
2016	Co-Director and Co-PI Cancer Nanotechnology Training Program, Radiology: A mentored research training program funded by a NIH T32 to support integrated research in cancer and nanotechnology involving molecular imaging, diagnostics and therapeutics.
2017-2020	Director of Oncology Research, Division of Oncology: I coordinate funding, semi-annual research retreats, annual Oncology division retreat, pilot funding and NIH T32 Oncology training grant.
2017-	Associate Director and Director of Admissions Medical Scientist Training Program: I am responsible for review of all applications and selecting interview candidates and admission committee for the Stanford MSTP program.
2018-	Director of Advanced Residency Training (ARTS) Program, a PhD granting program for medical doctors during their clinical training that supports up to 10 candidates.
2019-	Co-Director of KL2 Program: I am responsible for providing training, and mentorship for junior medical faculty in the School of Medicine.

2020-	Associate Chief of Oncology: I am responsible for scientific affairs in the division including mentorship and support and training of junior research faculty and support for our medical oncology research programs.

Clinical Teaching

Medical Oncology Attending, Med X, Stanford Hospital
Med X Lecture Series: Oncogenes as Targets for Therapy of Human Neoplasia
Medical Oncology Journal Club
Cancer Education Seminar
Translational Medicine MED121/221
MSTP
ARTS Program
KL2 Mentored Training
Cancer Nanotechnology
ReCap

Community Service

Highlands Elementary School, Science Fair Judge, 2003
Highlands Elementary School, Science Fair Judge, 2004
Baywood Elementary School Science Fair Judge, 2007
American Cancer Society, Lecture, Spring 2004
NIH Step-up Program/UCSF High School Program, Lecture, 2004
Leukemia and Lymphoma Society MWOY Campaign 2010
Medical School Outreach 2017-
SUMMA 2017-

Teaching Activities /Courses

Fall 2000	Discussion Leader, Cell Signaling and Cancer Mol Pharm 210/Cancer Bio 242
2001-2002	Discussion Leader, Cancer Biology Graduate Program Journal Club
Winter 2001	Faculty Speaker, Cancer Biology, 241
Winter 2002	Faculty Speaker, Cancer Biology 241, Study and Treatment of Cancer
Spring 2002	Faculty Speaker, Cancer Biology 243, Tumor Suppressor Genes
Spring 2002	Faculty Speaker, Advanced Immunology II
Spring 2003	Faculty Speaker, Pathology 243, Lecture: Carcinogenesis
Spring 2003	Faculty Speaker, Biology 205, DNA Repair
Fall 2004	Faculty Speaker, Cancer Biology
Spring 2004	Faculty Speaker, Advanced Immunology II
Fall 2004	Faculty Speaker, Pathology 243, Lecture: Carcinogenesis
Winter 2004	Faculty Speaker, Pathology 243, Lecture: Carcinogenesis
Fall 2005	Faculty Speaker, Pathology, 243, Lecture: Carcinogenesis
Winter 2005	Faculty Speaker, Pathology, 243, Lecture: Carcinogenesis
Winter 2006	Faculty Speaker, Health and Human Disease, Lecture: Carcinogenesis
Winter 2007	Faculty Speaker, Health and Human Disease, Lecture: Carcinogenesis/Immunity
Spring 2008	Faculty Speaker, Health and Human Disease, Lecture: Carcinogenesis
Winter 2008	Faculty Speaker, BIOE22B
Spring 2008	Faculty Speaker, CCRTP Course
Spring 2009	Faculty Speaker, Neoplasia, Carcinogenesis and Immune Surveillance
Spring 2009	Faculty Speaker, CCRTP Course
Spring 2010	Faculty Speaker, Advanced Immunology II
Spring 2010	Faculty Speaker, Cancer Biology, 222C

Spring 2010	Faculty Speaker, CCRTP Course
Spring 2011	Faculty Speaker, Health and Human Disease, Lecture: Cancer Biology
Spring 2011	Faculty Speaker, Advanced Immunology II
Spring 2011	Faculty Speaker, CCRTP Course
Spring 2012	Faculty Speaker, Neoplasia, Carcinogenesis and Immune Surveillance
Winter 2013	Faculty Speaker, Cancer Biology 241, Tumor Immunology
Spring 2013	Faculty Speaker, Advanced Immunology
Spring 2013	Faculty Speaker, Lung Block, Human Health & Disease Course
Fall 2013	Faculty Speaker, CCRTP Course, Mouse Models
Winter 2014	Faculty Speaker, Cancer Biology 241
Winter 2015	Faculty Speaker, Pathology 290
S, W, F	Faculty Director and Speaker, MED121/221
S, W, F. 2016	Faculty Director and Speaker MED121/221
Spring 2016	Faculty Speaker, HHD 221 Lecture
Spring 2016	Faculty Speaker, Immunology 209, Immune Checkpoints
S, W, F 2017-2018	Faculty Director and Speaker MED121/221
Spring 2017	Faculty Speaker, HHD Human Cancer Biology Lecture
Spring 2017	Faculty Speaker, Oncology Lecture, Grantsmanship and Funding
Spring 2017	Faculty Speaker, MSTP Lecture, Oncogene Addiction
S, W, F 2018-2019	Faculty Director Speaker MED121/221
Spring 2019	Faculty Speaker, KL2
S, W, F. 2019-2020	Faculty Director and Speaker MED121/221
S, W, F 2020-2021	Faculty Director and Speaker MED121/221
S, W, F. 2021-2022	Faculty Director and Speaker MED121/221
Spring 2021	Faculty Speaker, Immunology 258, Ethics, Science, and Society
S, W, F 2021-2022	Faculty Speaker, ReCAP
Summer 2023	Faculty Director and Speaker MED221
Winter 2024	Faculty Speaker, INDE 217 Physician Scientist Hour (PhySH)

Trainees

High School Students

2003	Michael Lin, UCLA MD, resident Stanford University
2004	Talia Lincoln, Medford College
2004-2005	Julian Burns, UCSD Medical Scholars Program, CA
2006	Charles Liu, Harvard University
2010	Julia Arzeno, UCLA Medical School, CA
2011	Nnola Amuzie, Stanford University, Stanford, CA
2019	Iwanshi Ahuja, Cupertino High School, CA
2022	Tony Zhang, Brookline High School, Brookline, MA, Carnegie Mellon University

College Students

2000-2001	Shelly Beer, UCLA, Stanford PhD, Merck
2000-2001	Sui Sui Song, Cornell University, Stanford Medical Student
2000-2001	Sandy Jung, Stanford University, Resident Harbor-General UCLA
2001-	Charles Feng, Stanford University, Honors, UCLA Medical School
2002-2003	Jared Miller, Stanford University, Washington University, Med Student
2003-2007	Maria Chang, Stanford University, NIH Scholar Program
2004-2008	Michael Lin, Stanford University, UCLA Medical Student

2004-2006 Cynthia Zamora, Stanford University, UCSF Medical School
2004-2009 Kim Komatsubara, Stanford University, UCLA Medical School
2004-2006 Talia Lincoln, Medford College
2004-2006 Julian Burns, currently in the UCSD Medical Scholars Program
2005 Troy McEachron, Stanford University, NYU Graduate Program
2005-2006 Ogechi Amarachukwu Okolo, Stanford University
2006-2008 Ada Yee, Stanford University, Stanford PhD, currently Editor, Nature
2006-2008 Jessie Tao, Stanford University, Harvard Medical School, Johns Hopkins
2006-2008 Stephen Hinshaw, Stanford University, currently RA Harvard U.
2006-2008 Joy Chen, Stanford University, Case Western Med Student, Stanford Surgery
2007-2008 Peter James Bellisle, Stanford University
2007-2010 Ramya Parameswaran, Stanford University, MSTP U. Chicago
2008-2010 Evan Chen, Stanford University, currently Stanford Medical Student
2009 Michael Sanchez, Stanford University
2009-2011 Sashendra Ravinath Aponso, Stanford University, Duke Singapore Program
2008 Erin Young, Utah State University
2009-2012 Vanessa Chang, Stanford University, U. Penn MSTP
2011-2014 Christine Yost, Stanford University, Baylor Medical School
2012-2016 Rachel Do, Stanford University, Vanderbilt Medical School
2012-2013 Julia Arzeno, UCLA, currently UCLA Medical School
2012-2015 Alia Yaghi, Stanford University, U. Texas, San Antonio Medical School
2014-2016 Georgia Toal, Stanford University, currently Stanford University Medical School
2015-2018 Theodore Hu, Stanford University, currently Masters Program, Cambridge
2017-2020 Maya Krishnan, Stanford University, currently MSTP Student
2018-2020 Natalie Wu, UC Davis, currently medical student
2019- Fidelia Alvina, U. Wisconsin Medical School,
2019-2021 Baokun Gu (Jack), Stanford University
2019-2020 Bryce Rossellini, Santa Clara University
2019-2020 Richard Barros, SFSU
2021- Nikhiya Shamsher, Stanford University
2021- Jessica Layne, Stanford University
2021 Chloe Zhao, Johns Hopkins University
2022- Connor Gonzales, Stanford University Bio-X Undergraduate Summer Program
2022 Zoe Gould, Smith College, MA
2022 Kevin Yang, Duke College, NC
2022 Eway Cai, Carleton College, MN, UC Berkley
2022 Majd Nasra, Stanford University

Graduate Students/Medical Students

2001-2003 Asa Karlsson, Division of Oncology, Stanford University and University of Goteberg
2001-2007 Constadina Arvanitis, Biological Sciences, Stanford University
2001-2007 Shelly Beer, Cancer Biology, Stanford University
2002-2004 Andrew Kopelman, Stanford School of Medicine, Stanford Med Scholar/HHMI
2004-2008 Pavan Bachireddy, Stanford School of Medicine, Stanford Med Scholar/HHMI
2004-2008 Pavan Bendapudi, Stanford School of Medicine, Stanford Med Scholar/HHMI
2005-2012 Peter Choi, Immunology Program, Stanford University
2005-2012 Alper Yetil, Biological Sciences Program, Stanford University
2006-2007 Melissa Horoschak, Stanford School of Medicine, Stanford Med Scholar
2006-2012 Kavya Rakhra, Immunology Program, Stanford University

2007-2009 Mathias Orbin, Medical Student, Munich, German
2014-2016 Rebecca Gao, Stanford Medical Student, Med Scholars
2016-2019 Nia Tope Adeniji, Stanford Medical Student, Med Scholars, UCSF Residency
2016-2017 Michael Richardson, Stanford Medical Student, Med Scholars
2017-2018 Line Heftdal, Aarhus University Medical Student, Danish Society
2021-2023 Josiah Yarbrough, Stanford University, Department of Chemical Engineering
2022-2023 Amanda Li, UC Berkley, Columbia University
2022-2023 Chris Aboujudom, Stanford University, TRAM Graduate Student
2022- M. Gohazrua K. Butler, Stanford University, TRAM Graduate Student

Post-Doctoral Fellows

2000-2002 Flora Tang, MD,
Current Position: PKPD Analyst, Genentech
2000-2001 Meenakshi Jain, MD
Current Position: Staff Physician, Santa Clara Valley Medical Center
2001-2005 Debabrita Deb, PhD, Fellow of Tumor Biology Training Grant
Current Position: Leadership Team, Inscopix
2001-2005 Sylvie Giuriato, PhD, Fellow of Lymphoma Foundation
Current Position: Research Scientist, Tolouse, France
2001-2006 Catherine Shachaf, PhD, Fellow FAMRI award
Current Position: President, Stelo Technologies
2002-2005 Karen Rabin, MD, Fellow of the Berry Foundation
Current Position: Associate Professor, Pediatrics, Baylor University
2002-2005 Suma Ray, PhD, Fellow of Stanford Dean's Scholar Award
Current Position: Vice President, Intas Pharmaceuticals
2002-2007 Alice Fan, MD, Fellow of the Leukemia and Lymphoma Society
Current Position: Assistant Professor Division of Oncology, Stanford
2002-2007 Chi-hwa Wu, PhD, Fellow of Immunology Training Program
Current Position: Scientist, Complete Genomics
2003-2007 Asa Karlsson, PhD, Fellow of Cancer Biology Training Grant
Current Position: Scientist Karolinska
2005-2012 Jan van Riggelen, PhD, Fellow of the Lymphoma Research Foundation
Current Position: Assistant Professor, Georgia Institute of Technology
2006-2009 Phuoc Tran, MD PhD, Fellow in Radiation Oncology
Current Position: Associate Professor, Johns Hopkins University
2006-2007 Ling Liu, PhD, Post-Doctoral Fellow
Current Position: Fellow, Dr. Tom Rando, Stanford
2006-2008 George Horng, Stanford University, Fellow Pulmonary Program
Current Position: Pulmonologist Palo Alto Clinic
2007-2012 David Bellovin, PhD, Post-Doctoral Fellow, NIH NRSA Award
Current Position: VP Discovery and Translational Biology, Attovia Therapeutics
2007-2012 Aleksey Yevtodiyenko, PhD, Post-Doctoral Fellow, Immunology Training Program
Current Position: Scientist, Life Sciences and Technology
2007-2012 Stacey Adam, PhD, Post-Doctoral Fellow, ACS Fellowship Award
Current Position: Director, Cancer in Research Partnerships Foundation
2007-2009 Zhongwei Cao, PhD, Post-Doctoral Fellow
Current Position: Assistant Professor, NYU
2007-2014 Yulin Li, PhD, Post-Doctoral Fellow, USC-NIH PSOC
Current Position: Assistant Professor, Houston Methodist Hospital

2009-2015 Emelyn Shroff, PhD, Post-Doctoral Fellow, American Lung Fellowship
Current Position: Senior Research Officer, Public Health Ministry, Seychelles
2009-2013 Bikul Das, PhD, Post-Doctoral Fellow, Canadian Cancer Fellowship
Current Position: Assistant Professor, Forsythe Institute, Boston, MA
2010-2013 Tahera Zabuawala, PhD, Post-Doctoral Fellow
Current Position: Project Manager, Personalis
2011-2016 Ling Tong, PhD, Fellow, BioX-Sanofi
Current Position: Senior Research Scientist, Stanford University
2012-2018 Stephaney Casey, PhD, Post-Doctoral Fellow, NIH NRSA, CRI, K22
Current Position: Amgen Scientist
2012-2017 Meital Ryan (Gabay), PhD, Post-Doctoral Fellow, SIP Award
Current Position: Head of Operations, Medical Devices, Verily
2013-2018 Dan Koch (now Liefwalker), PhD, Fellow, Burroughs Wellcome Fund, K22
Current Position: Assistant Professor, Oregon State Universtity
2014-2020 Anja Deutzmann, PhD, Post-Doctoral Fellow, Lymphoma Research Foundation Fellow
Current Position: Senior Research Scientist, Stanford University
2014-2020 Arvin Gouw, PhD, NIH T32 Fellowship
Current Position: Founding CEO, Bacchus Therapeutics
2015-2018 Srividya Swaminathan, PhD, Post-Doctoral Fellow. LLS Special Fellow
Current Position: Assistant Professor, City of Hope
2016-2021 Renu Dhanasekaran, MD, Instructor, Gastroenterology, TRAM, AGA, K08, ARTS
Current Position: Assistant Professor, Stanford University
2017-2022 Wadie Fernandez, PhD, TRAM
Current Position: Scientist, Sutro Biopharma
2017-2019 Sibu Kuruvilla, PhD, NIH T32 Fellow
Current Position: Manager, Genentech
2017-2019 Minsoon Kim, PhD
2018-2021 Christina Kim, PhD, NIH T32
2019-2021 Aida Hansen, PhD, Denmark Fellowship
Current Position: Assistant Professor, University of Southern Denmark
2021- Danielle Atibalentja, MD PhD, Heme Fellow, ASH Scholar
2021- Alessia Felici, PhD
2021- Xinyu Chen, PhD
2021-2023 Petronela Bulga, PhD2022- Selene Zhou PhD

Graduate Student Committees

Orals Committees

2002 Rebecca Begley, Dr. Mochly-Rosen Laboratory, Molecular Pharmacology
2002 Joshua T. Jones, Dr. Meyer Laboratory, Molecular Pharmacology
2003 Jacob Chudnovksy, Dr. Kharvari Laboratory, Cancer Biology
2003 Ryan B. Corcoran, Dr. Scott Laboratory, Cancer Biology
2004 Shelly Beer, Cancer Biology
2004 Constandina Arvanitis, Molecular Pharmacology
2004 Tom Johnson, Dr. Attardi Laboratory, Cancer Biology
2004 William Wong, Dr. Cleary Laboratory, Cancer Biology
2005 John Garcia, Dr. Khavari Laboratory, Cancer Biology
2006 Lauren Woodward, Cancer Biology
2007 Alper Yetil, Cancer Biology

2007 Kavya Rakhra, Immunology
2007 Peter Choi, Immunology
2011 Magdalena Franco, Microbiology and Immunology
2012 Joanna Kavalski, Cancer Biology
2016 Kayvon Pedram, Chemistry
2017 Benjie Smith, MSTP
2017 Stan Shor, MSTP
2020 Bastian Krenz, ChEM-H
2021 Andrea Garofalo, MSTP

Dissertation Committees
2002 Joon Whan Rhee, Dr. Cleary Laboratory, Immunology (Chair)
2003 Ryan Corcoran, Dr. Scott Laboratory, Cancer Biology
2003 Rebecca Begley, Dr. Mochly-Rosen Laboratory, Molecular Pharmacology
2003 Joshua T. Jones, Dr. Meyer Laboratory, Molecular Pharmacology
2006 Ryan Corcoran, Dr. Scott Laboratory, Cancer Biology
2007 Yakov Chudnovsky, Dr. Khavari Laboratory, Cancer Biology
2007 Thomas Johnson, Dr. Scott Laboratory, Cancer Biology
2007 Shelly Beer, Dr. Felsher Laboratory, Cancer Biology
2007 Lauren Woodward, Dr. Shapiro Laboratory, Cancer Biology
2007 Constadina Arvanitis, Felsher Laboratory, Cancer Biology
2008 Zhuang Liu, Dr. Dai Laboratory, Chemistry
2008 Meaghan Wall, Melbourne School of Graduate Research
2011 Sarah Sherlock, Dr. Dai Laboratory, Chemistry
2011 Kavya Rakhra, Dr. Felsher Laboratory, Immunology
2011 Alper Yetil, Dr. Felsher Laboratory, Cancer Biology
2011 Peter Choi, Dr. Felsher Laboratory, Immunology
2014 Magdalena Franco, Boothroyd Laboratory, Microbiology and Immunology
2021 Andrea Garofalo, Ash Alizadeh Laboratory, Cancer Biology
2021 Benjamin Smith, Carolyn Bertozzi Laboratory, Chemistry
2022 Dana Lee Cortade, Defense Chair, Shan X Wang Group, Materials Science & Engineering

Editorial Board
2008- Cancer Biology and Therapy
2009- Journal of Clinical Investigation
2009- Chinese Journal of Cancer
2010- Cancer Research
2010- Hematology Oncology
2010- OncoTarget
2010 Cancer Research, Associate Editor of Breaking Advances
2010- International Journal of Oncology
2012- OncoImmunology – Journal of the European Academy of Tumor Immunology
2012- Oncogene, Nature Publishing Group, Senior Editor
2013- Cancer Immunology Research – AACR Journal
2013- Cancer Hallmarks
2018- Cancer Research, Senior Editor

Invited Journal Reviews
American Journal of Pathology

American Journal of Pharmacogenomics
Blood
Breast Cancer Research
Cancer Research
Cancer Cell
Cancer Discovery
Cell
Cell Metabolism
Cell Systems
Cell Stem Cell
Clinical Cancer Research
Current Immunology
eLife
EMBO
Experimental Cell Research
Gastroenterology
Genes and Development
Journal of Clinical Investigation
Journal of National Cancer Institute
Lancet
Leukemia
Molecular Cancer Research
Molecular and Cellular Biology
Molecular Cell
Nature
Nature Biotechnology
Nature Cancer
Nature Chemistry
Nature Communications
Nature Genetics
Nature Medicine
Nature Reviews of Cancer
Oncogene
PLoS Genetics
PLoS One
Proceedings of the National Academy of Sciences
Science
Science Translational Medicine
Trends in Genetics
Trends in Molecular Medicine

NIH Study Sections

2000	NIH Ad Hoc, Review K08s
2004	NIH Site Visit, Hospital University of Pennsylvania
2005	NIH Experimental Therapeutics B Cluster
2006	NIH Clinical and Molecular Oncology Cluster
2006	NIH Clinical and Molecular Oncology Cluster
2007	NIH Molecular Carcinogenesis Study Section
2008	NIH Molecular Carcinogenesis Study Section

2010 NIH Molecular Oncology Study Section
2010 NIH Nanomedicine Development Center
2017 NIH Integrative Cancer Biology Program Special Study Section
2020 NIH NCI SPORE Review
2020 NIH SBIR Review, Co-Chair
2021 NIH NCI Program Projects
2021 NIH NCI Mechanisms of Cancer Therapeutics
2021 NIH 10 MCT2 Mechanisms of Cancer Therapeutics
2022 NIH NCI R35 Outstanding Investigator Award
2024 NIH NCI R35 Outstanding Investigator Award

NIH Intramural Review
2011 NIH Laboratory of Pathology
2011 NIH Laboratory of Pathology Core Facilities
2016 NIH Laboratory of Pathology

National Service
2005 Organizational Committee American Association for Cancer Research
2006 Organizational Committee, American Society for Clinical Oncology
2006 Organizational Committee. European Society of Hematology
2007 Organizational Committee, American Society for Hematology
2007 Organizational Committee, American Association for Cancer Research
2007 Organizational Committee, American Society for Clinical Oncology
2008 Sub-Committee Chair, American Association of Cancer Research
2011 Sub-Committee Chair, American Association of Cancer Research
2013- AACR Clinical and Translational Cancer Research Grants Scientific Review
2014 Organizational Committee, RECOMB Meeting
2015 Co-Chair, American Associate of Cancer Research, Conference of MYC oncogene
2016 Organizational Committee, RECOMB Meeting
2016 Organizational committee, Chair, Mini-Symposia, AACR
2019 Organizational committee, Chair, Mini-Symposia, AACR
2021-2022 AACR Basic Cancer Research Grants Scientific Review Committee
2022-2023 AACR Basic Cancer Research Grants Scientific Review Committee
2023- AACR Basic Cancer Research Grants Scientific Review Committee
2023-2024 AACR Basic Cancer Research Grants Scientific Review, Chair

Program Reviews
2009 Review Panel: UCSF BMS Graduate Program

Scientific Advisory Boards
2007-2010 Cell Biosciences, Palo Alto, California
2013- American Gene Therapeutics, Rockville, Maryland
2016-2020 Tragara Therapeutics, Carlsbad, California
2017- Molecular Decisions, California
2017- Apostle, California
2018- J Michael Bishop Institute, Chengdu, China
2019- Bacchus

Search Committees

2009	Chief of Infectious Disease, Department of Medicine
2010	Canary Early Detection Institute/Molecular Imaging Program
2010-2013	Medical Oncology, Lymphoma Program
2013	Medical Oncology, Melanoma Program
2013	Canary Center
2014	Medical Oncology, Head and Neck Program
2015	Canary Center
2016-	Canary Center
2018-	Medical Oncology, UTL Search

Honors, Awards and Memberships:

Honors

1985	Honors, Chemistry, University of Chicago
1992	Emil Bogen Research Award for Excellence in Science
2002	Charles Carrington Prize in Molecular Mechanisms of Disease

Awards

1985-1992	Medical Scientist Training Program, UCLA
1996-1998	Pfizer Medical Post-Doctoral Fellowship
1996-1998	Lymphoma Research Foundation Fellowship
1997-1999	Howard Hughes Medical Institute, Medical Post-Doctoral Fellowship
1998-2003	NIH Physician Scientist Award (K08 CA75967)
1999-2001	Pilot Feasibility Grant, UCSF Liver center
2000-2001	ASCO Young Investigator Award
2000-2001	Office of Technology Licensing Research Incentive Fund
2000-2002	V Foundation Scholar Award
2000-2003	Esther Ehrman Lazard Faculty Scholar Fund
2000-2001	Stanford Cancer Council Award
2001-	National Cancer Institute (R01 CA89305)
2001-2002	Leukemia Research Foundation Fellowship Award
2001-2002	Lymphoma Research Foundation Junior Faculty Award
2002-2003	Elsa U. Pardee Foundation
2002-2003	Pilot Feasibility Grant, Digestive Disease Consortium at Stanford University
2003-2004	Sarcoma Foundation of America
2003-2008	Damon Runyon-Lilly Clinical Investigator Award
2003-2006	Emerald Foundation Research Award
2003-2006	The Leukemia & Lymphoma Society Translational Research Award
2003-2008	National Cancer Institute (R01 CA105102)
2004-2007	National Cancer Institute (P20 CA112973)
2005-	National Cancer Institute (ICMIC P50 CA114747)
2005-2011	Burroughs Wellcome Fund Translational Investigator Award
2005-2011	National Cancer Institute (U54 CA119367)
2005-	Elected to American Society of Clinical Investigation
2006-2011	National Cancer Institute (P01 CA034233)
2006-2008	The Leukemia & Lymphoma Society
2006-2008	Bio-X Interdisciplinary Initiatives Award
2009-2012	Department of Defense Award

2011 Elected to the Association of American Physicians
2012-2016 NIH R01 Provocative Question Award
2014-2019 NIH U01 (CA188383)
2014-2019 NIH R01 (CA184384)
2015-2020 NIH T32 Training Grant, Department of Radiology
2017-2022 NIH RO1 Provocative Question Award
2021-2027 NIH R35 Outstanding Investigator Award

Memberships

1994- American College of Physicians
1995- American Medical Association
1996- American Society for Clinical Oncology
1998- American Society for Cell Biology
2000- American Society of Hematology
2000- American Association of Cancer Research
2001- American Society of Gene Therapy
2005- American Society of Clinical Investigation
2009- American Gastroenterological Association
2011- Association of American Physicians
2011- European Academy for Tumor Immunology (EATI)

Major Invited Addresses

1. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Charles Carrington Award Lecture. Stanford University, September 2003.

2. Felsher, D. W. Cancer Revoked: Oncogenes as therapeutic targets. Grand Rounds, Stanford University, Department of Medicine, Stanford, California, November 20, 2003.

3. Felsher, D. W. Reversing oncogene induced tumorigenesis. XV Zentrum Molecular Biology Heidelberg FORUM, Heidelberg, Germany, May 7-9, 2004.

4. Felsher, D. W. Co-Chair: Major Symposium: The malignant phenotype: Stability and reversibility. American Association of Cancer Research Annual Meeting, Orlando, Florida, March 27, 2004.

5. Felsher, D. W. Chair of Major Symposia: Oncogenes and tumor suppressor genes: Tumor biology in the clinic. American Society of Clinical Oncology Annual Meeting, Orlando Florida, May 13-17, 2005.

6. Felsher, D. W. Reversing Tumorigenesis. 100th Birthday Korea University Symposium, Seoul, Korea, November 3, 2005.

7. Felsher, D. W. Pushing cancer to the brink of normalcy through oncogene inactivation. Joint Graduate Symposium, Cell Fate Decisions in Health and Disease, University of Wuerzburg, Germany, November 8, 2005.

8. Felsher, D. W. Modeling Oncogene Addiction, Nobel Symposia, Karolinska Institutet, Stockholm, Sweden, 2012

Research Support:

Ongoing

Revolution Medicines 07/01/17-12/31/24
"Therapeutics in the mTor Pathway"

The goal is to identify a novel Tor pathway drug for the treatment of cancer.

NIH 1KL2TR003143, Felsher (Mentor) 07/15/19-06/30/24
"Institutional Career Development Core (KL2)

Goal is to function as a senior faculty mentor for the training of junior faculty.

NIH R35 Felsher (PI) 09/08/20-8/31/27
"Targeting the MYC Pathway for the Treatment of Cancer"

The goal is to develop a translational research program to study the MYC pathway.

Earli, Inc., Felsher (PI) 03/18/21-03/14/25
"Early Detection of Cancer"

The goal of the Earli grant is to develop a PET imaging prove for the early detection of cancer.

Pepper Bio, Felsher (PI) 10/01/21-09/30/24
"Phosphoproteomic Examination of Oncogene Pathways"

The goal of this project is to use novel computational biological approaches to identify phoshphoproteomic signatures of cancer.

Initial Therapeutics, Inc. (PI) 04/20/22-04/19/25
"Targeting Oncogene Protein Expression"

The goal is to study a novel small molecule for the treatment of myc driven cancers.

NIH UL1TR003142, Felsher (Co-I) 07/15/19-12/31/24
"Stanford Center for Clinical & Translational Education and Research (Spectrum)"

The goal mentorship and training of junior faculty engaged in translational medical research.

NIH 1R21EB034967-01 Felsher (Co-I) 07/01/23–06/30/25
"PET Tracer for Imaging Senescence"

Major Goals: This R21 project proposes to develop novel PET radiotracers for PET imaging of senecscent cells in vivo.

1U01CA288433-01, Felsher 09/19/23-08/31/27
“Molecular Mechanisms by which Statins Prevent and Reverse Hepatocellular Carcinoma”

The goal of this grant is to perform collaborative preclinical and basic science medical studies on the mechanisms by which statins can be used as an agent to reduce Hepatocellular carcinoma.

MEI Pharma, Inc. 03/18/24-12/31/24
“Voruciclib Efficacy in Solid Tumors”

The goal is to investigate if a specific cdk9 Inhibitor has activity against specific cancers that are Myc driven.

Completed

ASCO Young Investigator Award Felsher (PI) 07/01/00-06/30/01
“Defining When MYC Inactivation Results in the Regression of Hepatoma”

The goal of this study was to investigate if MYC inactivation induces the regression of hepatoma.

Lymphoma Research Foundation of America, Inc. Felsher (PI) 07/01/01-06/30/02
“MYC’s Role in Human Lymphomagenesis”

The major goal of this project was to determine if MYC induces reversible tumorigenesis in human lymphocytes.

Leukemia Research Foundation Felsher (PI) 07/01/01-06/30/02
“Targeting MYC Inactivation for the Treatment of Lymphoma”

The major goal of this project was to define how MYC inactivation causes the regression of hematopoietic tumors.

The V Foundation Felsher (PI) 08/01/00-07/31/02
“The Role of the MYC Proto-Oncogene in The Initiation and Maintenance of Tumorigenesis”

The major goal of this project was to examine how MYC activation cooperates with other oncogenes to induce neoplasia.

Elsa U. Pardee Foundation Felsher (PI) 11/01/01-02/28/03
“Defining when MYC will be an Effective Target for the Therapy of Cancer”

The major goal of this project was to investigate MYC’s role in the induction and maintenance of a neoplastic phenotype in human lymphomas.

Digestive Disease Center Felsher (PI) 03/01/02-02/28/03
“MYC’s Role in the Induction of Hepatocellular Carcinoma”

The focus of this project was to study the role of the MYC oncogene in the induction of hepatocellular carcinoma.

NIH/NCI 5K08 CA75967-02 Felsher (PI) 09/01/98–08/31/03
"C-MYC Induced Tumorigenesis and Genomic Instability"

The major goal of this project was to investigate how MYC induces genomic destabilization.

Sarcoma Foundation of American Felsher (PI) 04/01/03-03/31/04
"Targeting the Inactivation of the MYC Oncogene to Treat Osteogenic Sarcoma"

The goal of this project was to develop a new treatment for osteosarcoma.

3R01 CA89305-03S1 NOT-CA-03-017 Felsher (PI) 06/01/03-05/31/04
NIH/NCI (Supplemental)
"MYC's Role in the Initiation and Maintenance of Cancer"

The goal of this project was to define the role of immune-mediated mechanisms in the suppression of MYC-induced tumorigenesis.

Emerald Foundation Felsher (PI) 07/01/03-06/30/06
"Determining when Brief MYC Inactivation will Reverse Tumorigenesis"

The major goal of this proposal was to evaluate the duration of MYC oncogene inactivation required to result in sustained regression of hematopoietic tumors.

The Leukemia & Lymphoma Society Felsher (PI) 10/01/03-9/30/06
"Inactivating MYC for the Treatment of Lymphoma"

The goal of this project was to pre-clinically evaluate a new anti-sense drug that targets MYC in our transgenic animal model of lymphoma.

Ludwig Translational Program Cancer Research Felsher (PI) 11/01/04-10/31/06
"Phosphoprotein Signatures that Define the Therapeutic Efficacy of Atorvastatin for the Treatment of Lymphoma"

The major goal was to study phosphoprotein signatures in tumors treated with statins.

The Leukemia & Lymphoma Society Felsher (PI) 10/01/06-9/30/08
"A Phase 1 Study of Atorvastatin in Patients with Low Grade or Refractory Non-Hodgkin's Lymphoma"

The goal of this project is to pre-clinically evaluate atorvastatin for the treatment of lymphoma.

Bio-X Interdisciplinary Initiatives Award Felsher (PI) 10/01/06-09/30/08
"Carbon Nanotube Mediated Therapy of Lymphoma"

The goal of this project is to develop novel therapies for the treatment of lymphoma.

Damon Runyon Cancer Research Foundation Felsher (PI) 07/01/03-12/31/08
"Targeting MYC for the Treatment of Lymphoma"

The goal of this project is to perform a phase I/II trial to evaluate a new anti-sense drug that targets MYC for the treatment of lymphoma.

NIH/NCI 1R01 CA105102 Felsher (PI) 02/01/04-01/31/09
"Differentiation of Osteogenic Sarcoma By MYC Inactivation"

The goal of this project is to study how MYC inactivation induces the differentiation of osteogenic sarcoma in a transgenic mouse model.

NIH/NCI U56 CA112973 Plevritis (PI) 03/01/10-08/31/10
"Computational Modeling of Cancer Biology"

The goal of this project is to develop a multi-disciplinary research program in the systems biology of cancer. Dr. Felsher is a co-investigator receiving 5% effort and some laboratory support.

NIH/NCI U54 CA119367 Gambhir (PI) 05/12/06-04/30/11
Co-Leader Project 4 and 6
"Centers of Cancer Nanotechnology Excellence on Therapy Response"

The goal s of these projects are to apply nanotubes towards the development of novel therapies for cancers. Dr. Felsher is a co-investigator on two of the projects to pre-clinically evaluate nanotechnology in animal models.

Burroughs Wellcome Fund Felsher (PI) 07/01/05-06/30/11
Clinical Translational Award
"Pre-Clinical Validation of G-Quadruplex Drugs that Target MYC to Treat Cancer"

The major goal of this project is to perform a preclinical validation in transgenic mouse models of the role of G-Quadruplex drugs for the inactivation of the MYC oncogene for the treatment of cancer.

NIH R01 CA105102-05A1 Felsher (PI) 07/17/09-07/16/11
"Molecular and Cellular Basis of Oncogene Addiction"

The goal of this project is to define the mechanism by which oncogene inactivation elicits the phenomena of oncogene addiction.

NIH/NCI 2R01CA89305 Felsher (PI) 05/01/07-02/29/12

"MYC's role in the Initiation and Maintenance of Cancer"

The objective of the project is to define how MYC contributes to tumorigenesis by identifying and then interrogating how the repair of specific genetic events, such as p53 mutation restores the ability of MYC inactivation to induce sustained tumor regression through influences on proliferation, apoptosis and angiogenesis.

NIH/NCI P01 CA034233 (NCX) Levy (PI) 07/17/06-03/31/12
"Clinical and Laboratory Studies of Malignant Lymphoma"
Project Leader Project 3 "Immune Status and Tumor Regression Upon Oncogene Inactivation"

The goal of this project is to examine the contribution of the immune system and specific immune effector pathways in tumor regression upon MYC inactivation.

DOD CDMRP Felsher (PI) 04/15/09-04/14/12
"Nanoscale Proteomic Analysis of Oncoproteins in Hematopoietic Cancers"

The goal of this project is to develop novel methods to examine the oncogenic proteomic signaling pathways in hematopoietic cancers in response to therapy.

NCI 2P30CA124435-04 Mitchell (PI) 09/15/10-05/31/15
Stanford University Cancer Center

The major goal of this project is to build on institutional strengths in both technology development and translational research to foster interdisciplinary collaborations.

Onyx Pharmaceutical Corporation 108030 Felsher (PI) 06/17/12-12/16/12
"Defining and Predicting Carfilzomib activity using Novel Nanoscale Proteomic Methods in Preclinical Transgenic models of Lymphoma and Lung Cancer"

The goal of this project is to interrogate mechanism of carfilzomib using mouse models.

Onconova Therapeutics, Inc. Felsher (PI) 05/01/12-04/30/13
"Biomarker Analysis of MDS"

The goal of this project is to identify phosphoproteins that predict therapeutic response to a novel therapy for hematopoietic malignancies.

Laurel Foundation Felsher (PI) 12/01/10-05/31/13
"Identification of a rare population of human embryonic stem cells having potential tumorigenic activity following exposure to hypoxia oxidative stress"

The goal of this project is to characterize the role of oncogenes in the regulation of stem cell programs.

LLS Specialized Center of Research Grant Mitchell (PI) 10/01/08-09/30/13
"Characterization of Hematopoietic Stem Cells in Myelodysplastic Syndromes"
"Molecular and Cellular Characterization of Myelodysplastic Syndromes" Core D: (D. Felsher)

The goal of this project is to perform genomic/proteomic analysis of MDS/Leukemia specimens.

Geron Corporation Felsher (PI) 07/01/10-12/31/13
"Evaluation of Inhibitors or Regulators of c-MYC for the Treatment of Malignancies"

The Goal of this project is to develop a novel therapeutic agent.

NIH/USC U54 CA143907 Agus (PI) 08/01/12-07/31/14

"Multiscale Complex Systems Transdisciplinary Analysis of Response to Therapy (MCSTART)"

The goal of this project is to model and predict the therapeutic response of lymphoma to a chemotherapeutic agent.

Massachusetts Institute of Technology Felsher (PI) 08/01/12-07/31/14
(NIH PRIME) NIH/NCI U54 CA143874
"Defining and Predicting Response to Targeted Therapy Using Dry Density Measurement"

The goal is to utilize a novel nanofluidic to predict consequences of oncogene inactivation.

Onconova Therapeutics, Inc. #106824 Felsher (PI) 05/01/12-10/31/14
"Biomarker Analysis of MDS"
The goal of this project is to identify phosphoproteins that predict therapeutic response to a novel therapy for hematopoietic malignancies.

Regulus Therapeutics, Inc. Felsher (PI) 01/28/13-05/31/15
"Identification and Evaluation of Myc Regulated MicroRNAs as Potential Therapeutic Targets"
The purpose of this study is to examine the role of microRNA in the pathogenesis of MYC associated tumorigenesis.

NIH/NCI R21 CA169964 Felsher (PI) 08/01/12-07/31/15
"Nanoscale Proteomic Profiles of Hypoxia Pathways to Develop Biomarkers of Renal Cell Carcinoma"

This proposal is to develop prognostic and predictive proteomic biomarkers for primary and metastatic renal cell carcinoma using NIA technology to profile hypoxia pathways.

Onconova Therapeutics, Inc. #114321 Felsher (PI) 01/01/14-07/31/15
"Phase I Study of Platinum-based Chemoradiotherapy (CRT) with Oral Rigosertib in Patients with Intermediate or High-risk Head and Neck Squamous Cell Carcinoma"

Onconova Therapeutics, Inc. #110214 Felsher (PI) 03/01/13-08/31/15
NIA correlative studies of Oral Rigosertib in SCC

NIH/NCI ICMIC P50 CA114747 Gambhir (PI) 08/01/05-08/31/15
"In Vivo Cellular and Molecular Imaging Center Grant"
Project 3 Leader: "Multi-Modality Imaging of Oncogene-Induced tumorigenesis"

The objective is to utilize PET imaging to investigate the mechanism by which oncogene inactivation induces the regression of hematopoietic tumor.

Sanofi-Aventis, US, Inc./BioStar Felsher (PI) 12/10/12-12/09/15
"Prediction of Therapeutic Efficacy of Targeted Oncogene Inactivation via PET Imaging Using a Novel Smart Apoptosis Probe ([18F] CAIP)"

The goal of this project is to develop a novel approach for predicting the consequences of oncogene inactivation.

NIH/NCI ICBP CCSB U54 CA149145 Plevritis (PI) 05/01/10-02/29/16
Modeling the Role of Differentiation in T-ALL, Murine and Human
Project Leader Project 4: "Modeling the Role of Differentiation in Cancer Progression"

The goal of the Stanford Center for Systems Biology of Cancer (CCSB) is to discover molecular mechanisms underlying cancer progression.

NIH/NCI CCNE-T U54 CA151459 Gambhir (PI) 08/26/10-07/31/16
"Magneto-Nano Diagnostic and Analytical Devices for Cancer"
Project 2-(Wang/Felsher) Proteomic Validation of Micro-Chip Assay

The major goal of this project is to apply novel nanoscale diagnostic devices for the detection and monitoring of cancer.

Cancer Research Institute CLIP grant Felsher (PI) 07/01/14-06/30/17
"Oncogene addiction and immune activation"

The goal is to examine the mechanistic role of CD4+ T-cells in Oncogene Addiction.

Onkaido Therapeutics #119779 Felsher (PI) 03/25/15-06/30/17
"C-MYC Collaboration"

The Goal is to evaluate a novel therapy for liver cancer.

American Gene Technologies International Inc. Felsher (PI) 05/01/15-06/30/17
"HCC Lentiviral Therapeutic"

The goal is to develop a new therapeutic delivery approach for treatment of HCC.

NIH/NCI CCNE-T U54 CA151459 Gambhir (PI) 08/26/10-07/31/17
"Magneto-Nano Diagnostic and Analytical Devices for Cancer"
Project 2-(Wang/Felsher) Proteomic Validation of Micro-Chip Assay

The goal of this project is to apply novel nanoscale diagnostic devices for the detection and monitoring of cancer.

NIH/NCI R01 CA170378 PQ22 Felsher (PI) 08/01/12-07/31/17
"Mechanisms by Which Oncogene Inactivation Elicits Tumor Cell Death"

The goal of this study is to identify the mechanistic basis of cell death upon oncogene inactivation.

Tragara Pharmaceuticals, Inc., Felsher (PI) 07/01/16-06/30/17
"K9 Inhibitor Collaboration 2016"

This project investigators a novel CD inhibitor for cancer.

Apostle, Inc. 10/01/17-07/31/18

"Capturing Genetic Signature of Hepatocellular Carcinoma Through Liquid Biopsy with a Novel MiniMax Technology: a Pilot Study"

The goal is to identify a unique prognostic gene signature for liver cancer.

Roche TCRC, Inc. Felsher (PI) 09/01/16-02/28/19
"Investigation of Therapeutic Activity of RG6416"

The goal of this project is to study the mechanism of action of novel therapeutics.

Emerson Collective Cancer Research Fund, Felsher (PI) 04/01/17-03/31/19
"Identifying Small Molecules That Can Restore a Global Immune Response Against Cancer"

The goal is to identify new therapeutics to restore the immune response against cancers.

NIH R01 CA184384 Felsher/Zare (PI) 04/04/14-08/31/19
"Prognostic metabolic signatures of cancers through mass spectrometry imaging"

The goal of this project is to utilize DESI MS Imaging to determine the mechanistic role of MYC mediated regulation of lipid metabolism in tumorigenesis.

NIH U01 CA188383 Felsher/Gambhir (PI) 09/16/14-08/31/19
"Modeling and Predicting Therapeutic Resistance of Cancer"

The goal of this project is mathematically model how the immune system is involved in therapeutic resistance in T-cell acute lymphoblastic lymphoma.

Alligator Bioscience Felsher (PI) 09/03/14-09/02/19
"Development of Bispecific Immune Modulating Antibodies"

The goal of this project is to predict efficacy of novel immune therapeutics.

Sanofi US Services, Inc., Felsher (PI) 12/24/19-12/23/21

"Lipogenesis inhibition in cancer"

Goals: The goal of this study is to identify novel targets in the lipogenesis pathway to treat cancer.

NIH 1T32CA196585-01 Rao/Felsher (co-PI) 08/01/15-07/31/22
"Cancer-Translational Nanotechnology Training Program"

The Goal of this program is to train cancer biologist in nanotechnology.

Bio-X, Felsher (PI) 10/01/18-09/30/22
"Imaging changes in immune surveillance by natural killer (NK) cells during the progression of MYC oncogene-driven lymphomas"

Goals: The goal is study mechanisms of NK immune surveillance.

Patents:

Gouw A, Felsher DW, Jin F, Zare RN, Margulis K, Schow SR, Greenhouse RJ, Loughhead D, Richards S, inventors; Leland Stanford Junior University, assignee. Inhibitors of phospholipid synthesis and methods of use. United States patent US 11,702,394 B2. 2023 Jul 18.

Swaminathan S, Felsher DW, Mecker HT, inventors; Leland Stanford Junior University, assignee. Profiling and treatment of MYC-associated cancers with NK cells and type 1 interferon. United States patent US 11,648,275 B2. 2023 May 16.

Deutzmann A, Felsher DW, Li Y, inventors; Leland Stanford Junior University, assignee. Target genes in MYC-driven neoplasia. United States patent US 11,576,912 B2. 2023 Feb 14.

Felsher DW, Gabay M, Tibshirani R, inventors; Leland Stanford Junior University, assignee. Method of determining the prognosis of hepatocellular carcinomas using a multigene signature associated with metastasis. United States patent US 10,894,988 B2. 2021 Jan 19.

Felsher DW, Fan A, inventors; Leland Stanford Junior University, assignee. Discovery and validation of cancer biomarkers using a protein analysis methodology to analyze specimens. United States patent US 10,145,851 B2. 2018 Dec 04.

Publications:

Chapters (total of 3)

1. Arvanitis, C., Bendapudi, P. K., Bachireddy, P., and Felsher, D. W. Identifying critical signaling molecules for the treatment of cancer. Recent Results in Cancer Research, Vol. 172, Springer-Verlag Berlin Heidelberg 2007.

2. Bellovin, D.I., Das, B., and Felsher D.W. Tumor Dormancy, Oncogene Addiction, Cellular Senescence, and Self-Renewal programs. Systems Biology of Tumor Dormancy, pp 91-107, Part of the Advances in Experimental Medicine and Biology book series (AEMB, Vol. 734), Springer Link 2012.

3. Felsher, D.W., Arvanitis, C., Bendapudi, P., and Bachireddy, P. Oncogenes and the initiation and maintenance of tumorigenesis. Northwestern University | Northwestern Scholars, The Molecular Basis of Human Cancer, pp 143-157, Springer New York 2016.

Peer-reviewed articles (total of 134)

1. Welches, W., Felsher, D. W., Landshultz, W., and Maraganore, J. M. A rapid method for the purification of monomeric and/or dimeric phospholipases in crotalid snake venoms. Toxicon, 23(5): 747, 1985.

2. Felsher, D. W., Denis, K., Weiss, D., Ando, D. T., and Braun, J. A murine model of B-cell lymphomagenesis in immunocompromised hosts: C-MYC rearranged B-cell lines with a premalignant phenotype. Cancer Research, 50(21): 7042, 1990.

3. Felsher, D. W., Rhim, S., and Braun, J. A murine model for B-cell lymphomagenesis in immunocompromised hosts: Natural killer cells are an important component of host resistance to premalignant B-cell lines. Cancer Research, 50(21): 7050, 1990.

4. Felsher, D. W., Ando, D., and Braun, J. Independent rearrangement of Ig lambda genes in tissue culture-derived murine B-cell lines. International Immunology, 3(7): 711, 1991.

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7. Felsher, D. W., and Bishop, J. M. Reversible tumorigenesis by MYC in hematopoietic lineages. Molecular Cell, 4(2): 199, 1999.

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42. Deb-Basu, D., Fan, A., Voehringer, D., Felsher, D. W. Measurement of oncoproteins in primary hematopoietic malignancies pre-and post therapy using a nano-immunoassay system. AACR, San Diego, CA, April 12-16, 2008.

43. Shachaf, C. M., Gentles, A., Elchuri, S., Sahoo, D., Chang, M., Sharpe, O., Nolan, G., Plevritis, S., Felsher, D. W. Genomic and proteomic analysis reveals a threshold level of MYC required for tumor maintenance. AACR, San Diego, CA, April 12-16, 2008.

44. Riggelen, J. V., Felsher, D. W. The epigenetic context determines myc's oncogenic potential in a conditional mouse model for osteosarcoma. AACR, San Diego, CA, April 12-16, 2008.

45. Wu, C. H., Sahoo, D., Arvanitis, C., Bradon, N., Felsher, D. W. Comparative analysis of murine and human microarrys reveals a gene signature associated with the ability of myc to maintain tumorigenesis. AACR, San Diego, CA, April 12-16, 2008.

46. Horng, G. S., Tran, P. T., Chen, J., Bendapudi, P. K., Lin J., and Felsher, D. W. S-transfarnesylthiosalicylic acid (FTS) inhibits growth of k-ras4bG12D and myc induced primary lung adenocarcinoma in conditional mouse models of malignancy. American Thoracic Society International Conference, Toronto, Ontario, Canada, May 16-21, 2008.

47. Lin, H. J., Tran, P. T., Bendapudi, P. K., Chen, J., Horng, G., Felsher, D. W., Paik, D. S. A predictive model of oncogene-addiction. World Molecular Imaging Congress, September 2008.

48. Lin, H. J., Tran, P. T., Bendapudi, P. K., Chen, J., Horng, G., Felsher, D. W., Paik, D. S. A mathematical model of the escape mechanism that differentiates the behavior of oncogene-

and non-oncogene addicted tumor cells. World Molecular Imaging Congress, September 2008.

49. Fan, A. C., Deb-Basu, D., Gotlib, J. R., Orban, M. P., Voehringer, D., Felsher, D. W. Quantification of changes in protein phosphorylation during targeted therapy of primary hematopoietic malignancies using a nano-immunoassay system. ASCO-NCI-EORTC Annual Meeting on Molecular Markers in Cancer, Hollywood, Florida, October 30-November 1, 2008.

50. Fan, A. C., Orban, M. W., Shirer, A. E., Rajwanshi, R., Kong, C., Natkunam, Y., Lee, H. E., Coutre, S., Felsher, D. W. Nanoscale analysis of changes in signaling proteins in patients treated with single agent atorvastatin for low grade or refractory NHL. American Society of Clinical Oncology 2009 Annual Meeting, Orlando, Florida, May 29-June 2, 2009.

51. McClellan, S., To, C., Sikic, B. I., Brown, J. M., Fan, A., Felsher, D. W. Rib lesion in an oncology patient: Cancer or an uncommon presentation of an infectious disease? ACP Northern Chapter Conference.

52. Fan, A. C., Dermody, J., Kong, C., Zhang, N., Colevas, A. D., and Felsher, D. W. Nanoimmunoassay profiling of ERK and MEK isoforms in fine needle aspirates of solid tumors. ASCO Annual 2010 Meeting, Chicago, Illinois, June 4-6, 2010.

53. Fan, A. C., Dermody, J. L., Kong, C., Zhang, N., Xu, L., Renschler, J. P., Orban, M. W., Varasteh, B., Sridhar, K., Natkunam, Y., Coutre, S. E., Greenberg, P. and Felsher, D. W. Nanoscale approaches to define biologic signatures and measure proteomic response to targeted therapies in hematologic and solid tumors. AACR Fourth International Conference on Molecular Diagnostics in Cancer Therapeutic Development: Challenges and New Horizons. Denver CO, September 27-30, 2010.

54. Fan, A. C., Xu, L., Sridhar, K., Tran, M., Banerjee, P., Renschler, J. P., Tripuraneni, R., Wilhelm, F., Greenberg, P., and Felsher, D. W. A Novel Nano-immunoassay (NIA) Reveals Inhibition of PI3K and MAPK Pathways in CD34+ Bone Marrow Cells of Patients with Myelodysplastic Syndrome (MDS) Treated with the Multi-Kinase Inhibitor ON 01910.Na (Rigosertib). 53rd ASH Annual Meeting and Exposition, San Diego, CA, December 10-13, 2011.

55. Fan, A., Banerjee, P. and Felsher, D. W. A novel automated microfluidic size-based proteomic assay rapidly generates quantitative profiles of MAPK and PI3K proteins in clinical specimens. AACR Annual Meeting 2012, Chicago, Ill, March 31-April 4, 2012.

56. Ismail, A., Perry, R., Shroff, E., Zabuawala, T., Bellovin, D., Felsher, D. W., Zare, R. Desorption Electrospray Ionization Imaging Mass Spectrometry Identifies Lipid Species Regulated by the c-MYC Oncogene. ASMS Conference. Vancouver, BC, May 19-20, 2012.

57. Fan, A. C., Banerjee, P., Leppert, J., Harshman, L. C., Sabatti, C., Brooks, J. D., and Felsher, D. W. Nano-immuno assay generates rapid, quantitative nano-scale proteomic profiling of the hypoxia pathway in renal cell carcinoma clinical specimens. ASCO 2012 Annual Meeting, Chicago, Ill, June 1-5, 2012.

58. Nwabugwu, C., Felsher, D. W., and Paik, D. Mathematical modeling of the sequence of and interactions between cellular programs in response to oncogene inactivation measured by bioluminescence imaging. 2012 World Molecular Imaging Congress, Dublin Ireland, September 5-8, 2012.

59. Eberlin, L. S., Shroff, E. H., Zhang, J., Bellovin, D. I., Tibshirani, R., Felsher, D. W., and Zare, R. N. DESI-MS imaging of lipids and metabolites in cancers activated by the MYC and RAS oncogenes. ASMS 2013 Annual Conference, Minneapolis, MN, June 9-13, 2013.

Invited Presentations: (total of 288)

1. Felsher, D. W. Ando, D. T., and Braun, J., Independent Rearrangement of Lambda Light Chain in CD5+ B-cells. Western Conference of Molecular Biology, Berkeley, CA, 1989.

2. Felsher, D. W. and Braun, J. Pathophysiology of CD5+ B-cells. UCLA Symposia: B-cell Development. Taos, NM, 1990.

3. Felsher, D. W. and Braun, J. A Murine Model of CD5+ B-cell Lymphomagenesis. Western Conference of Immunology. Asilomar, CA, 1990.

4. Felsher, D. W. and Braun, J. A Murine Model for the Pathophysiology of CD5+ B-cells. Annual MSTP Conference, Aspen, CO, 1990.

5. Felsher, D. W. and Braun, J. CD5+ B-cells. Western Conference of Pathology. Los Angeles, CA, 1991.

6. Felsher, D. W. MYC Induces Genomic Destabilization. Stanford-UCSF Grand Rounds, San Francisco, CA, 1996.

7. Felsher, D. W. Transient MYC Overexpression Induces Tumorigenesis and Genomic Destabilization. UCSF, Mission Center, San Francisco, CA, 1998.

8. Felsher, D. W. The Mechanism of MYC Induced Tumorigenesis. UCSF, Division of Hematology-Oncology Grand Rounds, San Francisco, CA, 1998.

9. Felsher, D. W. Is MYC Induced Tumorigenesis Reversible? Grand Rounds, Gladstone Institute, San Francisco General Hospital, San Francisco, CA, 1998.

10. Felsher, D. W. MYC Induced Tumorigenesis, Invited Speaker. HHMI Physician Scientist Meeting, 1998.

11. Felsher, D. W. MYC Induced Genomic Destabilization and Tumorigenesis. UCSF Cancer Center, Hematopoietic Malignancies Group, San Francisco, CA, 1998.

12. Felsher, D. W. New Insights Into the Mechanism of MYC Induced Tumorigenesis. UCSF Cancer Center Discussion Group, San Francisco, CA, 1998.

13. Felsher, D. W. Oncogenes as Targets for the Therapy of Lymphoma. Lymphoma Research Foundation Conference, 1998.

14. Felsher, D. W. Reversible Tumorigenesis by MYC, Microbiology Seminar Series. UCSF, San Francisco, CA, May 1999.

15. Felsher, D. W. Reversible Tumorigenesis by MYC Using a Conditional Transgenic Model. Invited speaker, Oncogenes and Growth Control Meeting, Salk Institute, August 1999.

16. Felsher, D. W. Reversible Tumorigenesis by MYC Using a Conditional Transgenic Model. Invited speaker, Hematology Seminar, Stanford University, Stanford, CA, October 1999.

17. Felsher, D. W. Reversible Tumorigenesis by the MYC Proto-Oncogene Using a Conditional Transgenic Model System. Department of Medicine Rounds, Stanford University, Stanford, CA, January 3, 2000.

18. Felsher, D. W. MYC Signaling in Normal and Pathological Processes. Stanford University, Stanford, CA, March 2, 2000.

19. Felsher, D. W. Reversible Tumorigenesis by MYC. Invited Speaker, UCSF Cancer Center, San Francisco, CA, May 5, 2000.

20. Felsher, D. W. Reversible Hepatocellular Carcinoma by MYC Using a Conditional Transgenic Model. Invited Speaker, 16th Annual meeting on Oncogenes and Tumor Suppressors, Salk Institute, La Jolla, CA, June 22-25, 2000.

21. Felsher, D. W. MYC Inactivation in Hematopoietic Tumors that have Lost P53 Still Regress, but Subsequently Relapse. The 42nd ASH Annual Meeting, San Francisco, CA December 2000.

22. Felsher, D. W. Reversible MYC-induced Tumorigenesis. Stanford University, Stanford, CA, October 9, 2000.

23. Felsher, D. W. Reversible Tumorigenesis by MYC Using a Conditional Transgenic Model System. University of Louisville, Louisville, Kentucky, November 6, 2000.

24. Felsher, D. W. Oncogene-induced Tumorigenesis is Reversible. AXYS Pharmaceuticals Seminar, San Francisco, CA, December 2000.

25. Felsher, D. W. MYC's Role in Signaling, Invited seminar. Stanford University, Stanford, CA, February 22, 2001.

26. Felsher, D. W. Reversing MYC-induced Tumorigenesis in a Transgenic Model. Invited seminar, DNAX, Palo Alto, CA, March 6th, 2001.

27. Felsher, D. W. Conditional Oncogene Expression in Transgenic Mice. Invited talk, The 2nd Gordon Research Conference, New London, NH, July 4, 2001.

28. Felsher, D. W. Defining When MYC Inactivation Induces Reversible Tumorigenesis. Salk/EMBL Oncogenes and Growth Control, La Jolla, CA, August 20, 2001.

29. Felsher, D. W. Reversing MYC-induced Tumorigenesis. Sunnybrook and Women's College Health Sciences Center, Toronto, Ontario Canada, March 27, 2001.

30. Felsher, D. W. Defining when Oncogenes will be Effective Therapeutic Targets for the Treatment of Cancer. Sunnybrook and Women's College Health Sciences Center, Toronto, Ontario Canada, March 27, 2001.

31. Felsher, D. W. The MYC Oncogene's Role in the Induction and Maintenance of Hepatocellular Carcinoma. Digestive Diseases Consortium Seminar, Stanford University, Stanford, CA, June 13, 2002.

32. Felsher, D. W. Permanent Loss of a Neoplastic Phenotype by Brief MYC Inactivation. SALK Oncogene meeting. San Diego, CA, June 22, 2002.

33. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. Chiron Corporation, Emeryville, CA, September 13, 2002.

34. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. Karolinska Hospital, Sweden, October 2, 2002.

35. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. UCLA Department of Pathology, Grand Rounds, Los Angeles, CA, October 23, 2002.

36. Felsher, D. W. Reversing Cancer through Oncogene Inactivation. Stanford University, Stanford, CA, October 31, 2002.

37. Felsher, D. W. MYC's Role in the Induction and Maintenance of Tumorigenesis. Epithelial Biology Seminar. Stanford University, Stanford, CA, November 22, 2002.

38. Felsher, D. W., Deb-Basu, D., and Karlsson, A. Restoration of p27 Function Prevents MYC from Inducing Genomic Instability and Apoptosis. ASCB, San Francisco, CA, December 2002.

39. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. SALK, La Jolla, CA, December 19, 2002.

40. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. Cyternex, Inc., San Diego, CA, February 6, 2003.

41. Felsher, D. W. Oncogenes as Therapeutic Targets. Scheduling Program in Epithelial Biology Seminar Series, Stanford University, Stanford, CA, March 12, 2003.

42. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Tularik, Inc., San Francisco, CA, April 23, 2003.

43. Felsher, D. W. Reversing MYC-Induced Lymphomagenesis. FASEB, Saxtons River, Vermont, July 26-31, 2003.

44. Felsher, D. W. Reversing MYC-Induced Tumorigenesis. AVI BioPharma, Portland, OR, August 5, 2003.

45. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Charles Carrington Award Lecture. Stanford University, Stanford, CA, September 2003.

46. Felsher, D. W. Reversibility of Lymphomas. Swiss-German Hematology Meeting Marburg University, October 4-8, 2003.

47. Felsher, D. W. Reversibility of Lymphomas. Swiss German Hematology, Basel, Switzerland, October 7, 2003.

48. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. University of Pennsylvania, Philadelphia, Pennsylvania, October 16, 2003.

49. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Grand Rounds, Stanford University, Department of Medicine, Stanford, CA, November 20, 2003.

50. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Signal Transduction 2004, Luxembourg, January 27, 2004.

51. Felsher, D. W. Cancer Revoked: Targeting Oncogenes to Treat Cancer. Nuclear Medicine Grand Rounds, Stanford University, Stanford, CA, March 16, 2004.

52. Felsher, D. W. Co-chair: Major symposium: The Malignant Phenotype: Stability and Reversibility. AACR, Orlando, Florida, March 27, 2004.

53. Felsher, D. W. Reversing Oncogene Induced Tumorigenesis. XV ZMBH FORUM, Heidelberg, Germany, May 7-9, 2004.

54. Felsher, D. W. Cancer Revoked: Oncogenes as Therapeutic Targets. Genentech Molecular Oncology, South San Francisco, CA, June 10, 2004.

55. Felsher, D. W. Reversing Oncogene Induced Tumorigenesis. King's College, London, England, August 11, 2004.

56. Felsher, D. W. Revoking Cancer Through Targeted Oncogene Inactivation. American Cancer Society, Los Gatos, CA, September 1, 2004.

57. Felsher, D. W. Lymphoma Revoked: Through Oncogene Inactivation. 3rd Mouse Models of Hematopoietic Malignancies Workshop. Memorial Sloan-Kettering Cancer Center, New York, NY, October 11-13, 2004.

58. Felsher, D. W. Reversing Oncogene-Induced Tumorigenesis. University of California San Francisco Cancer Center, San Francisco, CA, November 12, 2004.

59. Felsher, D. W. EMBO Molecular Medicine Meeting, Germany, November 28 – December 1, 2004.

60. Felsher, D. W. MYC Inactivation Uncovers Stem Cell Properties and Tumor Dormancy in Liver Cancer. Cell and Developmental Biology Faculty Talks. Stanford University, Stanford, CA, January 10, 2005.

61. Felsher, D. W. Conditional Mouse Models of Oncogene Induced Cancer. ICBP Meeting, Stanford University, Stanford, CA, January 11, 2005.

62. Felsher, D. W. Reversing MYC Induced Tumorigenesis. Keystone Symposia: Cancer and Development, Banf Canada, February 5-10, 2005.

63. Felsher, D. W. Cancer: A Genetic Paradigm in an Epigenetic Context. Stanford University, Department of Dermatology, Epithelial Biology Seminar, Stanford, CA, March 11, 2005.
64. Felsher, D. W. U.S. Japan Workshop, Animal Models for Hematologic Malignancies And Hematopoiesis. Maui Hawaii, March 22-26, 2005.

65. Felsher. D. W. Reversing Oncogene Induced Tumorigenesis. Organnon. Oss, Netherlands, April 11, 2005.

66. Felsher, D. W. Invited Talk: ASCI/AAP 2005 Joint Meeting, Chicago, Illinois, April 15-17, 2005.

67. Felsher, D. W. Methods Workshop: Conditional Oncogene Induced Tumorigenesis. AACR 96th Annual Meeting, Anaheim, CA, April 16-20, 2005.

68. Felsher, D. W. Targeting MYC to Reverse Lymphomagenesis. Damon Runyon Foundation, New York, May 1, 2005.

69. Felsher, D. W. Chair of Major Symposia: Oncogenes and Tumor Suppressor Genes: Tumor biology in the clinic. ASCO, Orlando Florida, May 13-17, 2005.

70. Felsher, D. W. ICBP Meeting, Integrative Cancer Biology Program NCI, Berkeley, CA, May 15-18, 2005.

71. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Microbiology and Tumor Biology Center. Karolinska Institutet, Stockholm, Sweden, June 1, 2005.

72. Felsher, D. W. Tumor Dormancy: Cancer Genetics Put into an Epigenetic Context, June 3rd and Myc repair and genomic instability, June 4th, 10th. Congress of the European Hematology Association, Stockholm, Sweden, June 2005.

73. Felsher, D. W. Targeting MYC for the Treatment of Lymphoma. Lilly Research Laboratories, Indianapolis, Indiana, June 10, 2005.

74. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addition. Dana Farber Cancer Institute, Harvard Medical School, Boston, MA, June 28, 2005.

75. Felsher, D. W. Reversing Hematopoietic Tumorigenesis. Gordon Research Conference, Rhode Island, July 2005.

76. Felsher, D. W. Reversing Oncogene Induced Tumorigenesis. SALK/EMBL Oncogene and Growth Control Meeting, Salk Institute, San Diego, CA, August 12-16, 2005.

77. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. University of Cincinnati, Cincinnati, OH, September 23, 2005.

78. Felsher, D. W. Imaging the Reversal of Tumorigenesis upon Oncogene Inactivation. Cancer and stem cells, Imaging 2020. Jackson Lodge, Wyoming, September 29, 2005.

79. Felsher, D. W. Digestive Disease Consortium, Stanford University, Stanford, CA, October 1, 2005.

80. Felsher, D. W. MYC Function and Liver Cancer Stem Cells. International Titisee Conference, Black Forest, Germany October 2005.

81. Felsher, D. W. Reversing Tumorigenesis. 100th Birthday Korea University Symposium, Seoul, Korea, November 3, 2005.

82. Felsher, D. W. Pushing Cancer to the Brink of Normalcy Through Oncogene Inactivation. 1st Joint Graduate Symposium, Cell Fate Decisions in Health and Disease, University of Wuerzburg, Germany, November 8, 2005.

83. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Fred Hutchinson Cancer Center, Seattle WA, November 29, 2005.

84. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Massachusetts General Hospital, Boston, MA, January 11, 2006.

85. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Epithelial Biology Seminar Series, Stanford University, Stanford, CA, 2006.

86. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. PCCM Division, Stanford University, Stanford, CA, March 24, 2006.

87. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Van Andel Institute, Grand Rapids, Michigan, April 12, 2006.

88. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Dartmouth, Hanover, New Hampshire, May 10, 2006.

89. Felsher, D. W. Tumor Intrinsic and Host-Dependent Mechanisms of Oncogene Addiction. NCI Mouse Models of Human Consortium Meeting, Seattle, Washington, June 28, 2006.

90. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. IFOM-IEO, Campus, European Institute of Oncology, Milan, Italy, September 27, 2006.

91. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. ISREC, Switzerland, October 2, 2006.

92. Felsher, D. W. Oncogenes on Target to Treat Cancer. Molecular Pharmacology and Quantitative Chemical Biology Seminar, Stanford University, Stanford, CA, October 10, 2006.

93. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Lymphoma Meeting, Palermo, Italy, October 2006.

94. Felsher, D. W. Mechanisms of Oncogene Addiction. Seminars in Oncology, Dana-Farber Cancer Institute and the Dana-Farber/Harvard Cancer Center, Boston, Massachusetts, October 17, 2006.

95. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. AACR Mouse Model Meeting, Cambridge Massachusetts, October 25, 2006.

96. Felsher, D. W. Liver Cancer Stem Cells. German, Austria and Swiss Society of Hematology and Oncology, Leipzig, Germany, November 4, 2006.

97. Felsher, D. W. Imaging Death and Resurrection of Cancer. Small Animal Imaging Symposium, Stanford University, Stanford, CA, November 15-18, 2006.

98. Felsher, D. W. Reversing Oncogene-Induced Tumorigenesis. Applied Biosystems, Foster City, CA, November 30, 2006.

99. Felsher, D. W. Molecular Basis of Oncogene Addiction. Oregon Health Sciences. Portland, Oregon, January 2007.

100. Felsher, D. W. Imaging the Death And Resurrection of Cancer. MIPS Seminar, Stanford University, Depart of Radiology/Nuclear Medicine, Stanford, CA, February 5, 2007.

101. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Stanford University, Developmental Biology, Stanford, CA, March 5, 2007.

102. Felsher, D. W. Plenary Session on Mouse Models. AACR Annual meeting, Los Angeles, CA, April 2007.

103. Felsher, D. W. Educational Session: Validation of Targets/Models of Human Cancer. Molecular and cellular basis of oncogene addiction. AACR Annual Meeting, Los Angeles, CA, April 2007.

104. Felsher, D. W. Morning Session: Mouse Models of Cancer. AACR Annual Meeting, Los Angeles, CA, April 2007.

105. Felsher, D. W. The Role of Oncogenes in the Pathogenesis of Neoplasia. Tromso, Norway, April 2007.

106. Felsher, D. W. The Cellular and Molecular Basis of Oncogene Addiction. Karolinska Institute, Stockholm Sweden, April 2007.

107. Felsher, D. W. Reversing Tumorigenesis. Centro Nacional de Investigaciones Oncologicas, Madrid, June 2007.

108. Felsher, D. W. Imaging Tumor Regression upon Oncogene Inactivation. COBRA Meeting, August 24, 2007.

109. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Pharmacology and Cancer Biology Lecture Series, Duke University, Durham, NC, September 2007.

110. Felsher, D. W. Modeling Oncogene Addiction and Oncogene Escape. ICBP Steering Committee Meeting, Washington DC, November 13-14, 2007.

111. Felsher, D. W. Reversing tumorigenesis. Translational Oncology Symposium, UCSD Cancer Center, La Jolla, CA November 16, 2007.

112. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. University of Manchester, England, November 28, 2007.

113. Felsher, D. W. Molecular and Cellular Basis of Oncogene addiction. Lankenau Institute of Medical Research, Philadelphia, Pennsylvania, December 13, 2007.

114. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Abramson Family Cancer Research Institute, University of Pennsylvania, December 14, 2007.

115. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. University of California San Francisco, San Francisco, CA, January 25, 2008.

116. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Ohio State, Columbus, Ohio, February 5, 2008.

117. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. UCSD Director's Seminar Series, La Jolla, CA, February 13, 2008.

118. Felsher, D. W. Molecular and Cellular Basis of Oncogene Addiction. Celgene Corporation, San Diego, CA, February 28, 2008.

119. Felsher, D. W. ICBP Meeting, Columbus, OH, May 13-14, 2008.

120. Felsher, D. W. Mechanisms of Oncogene Addiction. Marburg, Germany, June 3, 2008.

121. Felsher, D. W. Gordon Conference, Rhode Island, July 28-August 1, 2008.

122. Felsher, D. W. Oncogene Addiction and a Dr Jekyll and Mr Hyde Model of Cancer. Dana Farber Cancer Institute, Boston MA, August 4, 2008.

123. Felsher, D. W. Drug Discovery and Innovative Therapeutics, Boston MA, August 6, 2008.

124. Felsher, D.W. Cancer Genetics & Epigenetics. Cold Spring Harbor Symposium, Cold Spring Harbor NY, August 13-17, 2008.

125. Felsher, D. W. Oncogenes and Cancer. Stanford Cancer Research Training Program, Stanford University, CA September 14, 2008.

126. Felsher, D. W. Nanoscale Proteomic Analysis of Clinical Cancer Specimens. Biomarker Discovery Summit 2008, Sixth Annual Protein Biomarker, Philadelphia PA, September 29-October 1, 2008.

127. Felsher, D. W. Mechanisms of Oncogene Addiction: A Dr Jeckyll and My Hyde model of tumorigenesis. Cell and Developmental Biology Faculty Lunch Series, Stanford University, Stanford, CA, November 3, 2008.

128. Felsher, D. W. Modeling Oncogene Addiction. Seminar IUH, Salle de Cours Batiment Inserm, Paris, France, December 12, 2008.

129. Felsher, D. W. Charite – Universitatsmedizin, Berlin, December 17, 2008.

130. Felsher, D. W. Non-Hodgkin Lymphoma (low Grade/indolent) & Waldenstrom's. Emerging Therapies for Blood Cancer Patients. Leukemia and Lymphoma Society, San Francisco, CA, January 31, 2009.

131. Felsher, D. W. Models and Modeling of Oncogene Addiction. Penn State Hershey Cancer Institute, Hershey, PA, March 9-11, 2009.

132. Felsher, D. W. Targeted Cancer Therapies. Keystone Symposia on Molecular and Cellular Biology, Whistler, British Columbia, Canada, March 27- April 4, 2009.

133. Felsher, D. W. Mouse Models of Liver Cancer. National Institute of Health, Bethesda, Maryland, April 9, 2009.

134. Felsher, D. W. Tumor Dormancy and Oncogene Addiction. AACR, Annual Meeting, Denver, Colorado, April 18-22, 2009.

135. Felsher, D. W. Reversing Cancer through Targeted Oncogene Inactivation. 2009 Annual Conference of the Chinese-American Bio/Pharmaceutical Society (CABS), San Francisco, CA, May 23, 2009.

136. Felsher, D. W. Mouse Models of Human Cancers. First Annual Center for Cancer Nanotechnology Excellence Symposium, Bechtel Conference Center, Stanford University, Stanford, CA, May 28-29, 2009.

137. Felsher, D. W. Proteomic Nanotechnology of Clinical Specimens Drug Discovery and Development. Keio Plaza Hotel, Japan, June 1, 2009.

138. Felsher, D. W. Modeling Oncogene Addiction. Molecular Therapeutics Research Association Meeting, Stanford, CA, July 19-22, 2009.

139. Felsher, D. W. The Expanding Role of Tet-Controlled Expression Models to Understand Oncogene Addiction and Malignant Progression. The EMBO Meeting, Amsterdam, August 29, 2009.

140. Felsher, D. W. MYC, Self-Renewal And Senescence. Gordon Research Conference: Stem Cells and Cancer, Switzerland, September 13-18, 2009.

141. Felsher, D. W. ADAPT Congress, Protein Biomarkers, The Grand Hyatt Washington, DC, September 22-25, 2009.

142. Felsher, D. W. Oncogene Addiction. Cell Regulation and Cancer. The Third Comprehensive Cancer Research Training Program at Stanford University (CCRTP-3), Menlo Park, CA, September 28- October 2, 2009.

143. Felsher, D. W. 2nd International Workshop on Cholangiocarcinoma and Hepatocellular Carcinoma, Washington, DC, October 6-7, 2009.

144. Felsher, D. W. Modeling Oncogene Addiction: Reversing Cancer from Inside And Out. Cancer Models and Mechanisms Symposium, Cancer Research UK, Cambridge, England, December 3-4, 2009.

145. Felsher, D. W. Molecular Modeling Oncogene Addiction. Lurie Cancer Center of Northwestern University, Chicago, IL, December 10, 2009.

146. Felsher, D. W. Bio-X/Novartis Meeting, James H. Clark Center, Stanford University, Stanford, CA, January 20, 2010.

147. Felsher, D. W. Modeling Oncogene Addiction for the Development of New Treatments for Cancer, Novartis, Emeryville CA, February 17, 2010.

148. Felsher, D. W. Molecularly Modeling and Predicting Oncogene Addiction in Lung Cancer, Bay Area Workshop on Lung Development, Physiology and Cancer, UCSF, San Francisco, CA, February 19, 2010.

149. Felsher, D. W. Targeting MYC Pathway for Cancer Treatment, SuperGen, Inc. Dublin, CA, March 22, 2010.

150. Felsher, D. W. c-Myc, as an Oncology Drug Discovery Target. Geron Corporation, Menlo Park, CA, March 24, 2010.

151. Felsher, D. W. Modeling and Predicting Oncogene Addiction. University of Toronto, Ontario Canada, April 9, 2010.

152. Felsher, D. W. Cancer Center's (ESAB) External Scientific Advisory Board Presentation, Stanford University, Stanford, CA, April 26, 2010.

153. Felsher, D. W. Modeling Oncogene Addiction. NIH/NCI Center for Cancer Research, Bethesda MD, May 3, 2010.

154. Felsher, D. W. Modeling Oncogene Addiction. ICBP Centers for Cancer Systems Biology Annual Meeting, Bethesda, MD, May 3-5, 2010.

155. Felsher, D. W. Modeling Oncogene Targeted Therapeutics. Agilent, Santa Clara, CA, June 21, 2010.

156. Felsher, D. W. Modeling of Oncogene Addiction in Transgenic Mouse Models. Cold Spring Harbor Laboratory Meeting, Mechanisms & Models of Cancer, Cold Spring Harbor, NY, August 17-21, 2010.

157. Felsher, D. W. Molecular Therapies that Target Oncogenes. Stanford Cancer Center CCRTP Course, Stanford, CA, September 14, 2010.

158. Felsher, D. W. Nanoscale Proteomics in Cancer. ADAPT Biomarker Meeting, Arlington, VA, September 15-16, 2010.

159. Felsher, D. W. Seminars in Oncology Lecture Series, Dana-Farber Cancer Institute and the Dana-Farber/Harvard Cancer Center, Boston, MA, September 21, 2010.

160. Felsher, D. W. AACR Molecular Diagnostics, Denver, CO, September 27-30, 2010.

161. Felsher, D. W. Advances in Oncology, Greece, October 7-9, 2010

162. Felsher, D. W. 2010 NanoPro User Meeting, Washington DC, October 13-15, 2010.

163. Felsher, D. W. Modeling Oncogene Addiction Inside Out. Columbia University, New York City, NY, November 8, 2010.

164. Felsher, D. W. Oncogene Addiction: Inside and out. Memorial Sloan Kettering Cancer Center, New York, NY, November 9, 2010

165. Felsher, D. W. Oncogene Addiction Inside Out. University of Arizona, Tucson, AZ, November 22, 2010.

166. Felsher, D. W. Targeting the MYC Pathway to Reverse Cancer. SuperGen, Inc., Salt Lake City, UT, January 19, 2011.

167. Felsher, D. W. Multi-Scale Modeling to Predict Therapeutic Response in Lung Cancer. Pulmonary Medicine and Biology Grand Rounds, Stanford University School of Medicine, Stanford, CA, February 11, 2011.

168. Felsher, D. W. Nanoscale Analysis of Oncogene Addiction. Genentech, San Francisco, CA, March 9, 2011.

169. Felsher, D. W. Modeling and Predicting Oncogene Addiction. 16th International AEK Cancer Congress, Duesseldorf, Germany, March 16-18, 2011.

170. Felsher, D. W. Modeling Oncogene Addiction. Amgen, Thousand Oaks, CA, March 21, 2011.

171. Felsher, D. W. Modeling Oncogene Addiction. Systems Biology Conference, Stanford University, Stanford, CA, May 2-3rd, 2011.

172. Felsher, D. W. Oncogene Addiction Inside And Out. Molecular Biology, Microbiology and Biochemistry Seminar Series, Southern Illinois University, Carbondale, IL, May 6, 2011.

173. Felsher, D. W. Modeling Tumor Dormancy, Dormancy Workshop, Boston MA, July 25-28, 2011.

174. Felsher, D. W. Cancer Therapy and Biomarkers. CCRTP Conference, Stanford, CA, September 14-16th, 2011.

175. Felsher, D. W. Reversing Tumorigenesis through Targeted Oncogene Inactivation. 16th World Congress on Advances in Oncology, Athens Greece, October 6-8, 2011.

176. Felsher, D. W. MYC as a Therapeutic Target. MYC and the Pathway to Cancer. Cold Spring Harbor, NY, November 6-9, 2011.

177. Felsher, D. W. Modeling Oncogene Addiction. Cancer Conference 2011. From Carcinogenesis to Cancer Therapy, Xcaret Mexico, November 9-13, 2011.

178. Felsher, D. W. International Society for Cellular Oncology 2012 Congress, Mallorca Spain, March 4-8, 2012.

179. Felsher, D. W. Modeling and Predicting Oncogene Addiction. Karolinska Institutet, Frontiers in Cancer Research and Therapy, Stockholm, Sweden, March 8-9, 2012.

180. Felsher, D. W. Targeting MYC for the Treatment of Cancer. Geron Corporation, Menlo Park, CA, March 21, 2012.

181. Felsher, D. W. Modeling and Predicting Oncogene Addiction. St. Jude Children's Research Hospital, Memphis, TN, March 28, 2012.

182. Felsher, D. W. Modeling Oncogene Addiction. MDC Systems Biology Meeting, Berlin, Germany, July 2012.

183. Felsher, D. W. Noncanonical Role the Immune Systems in Oncogene Addiction. MDC, Berlin, Germany, July 2012.

184. Felsher, D. W. Modeling and Measuring Oncogene Addiction. MD Anderson, Houston, TX, August 22, 2012.

185. Felsher, D. W. Funding Your Research, Stanford Translational and Applied Medicine Program, Stanford, CA, October 10, 2012.

186. Felsher, D. W., Oncogene Addiction and the Immune System, SITC Workshop, Bethesda, MD, October 24-25, 2012

187. Felsher, D. W. Modeling Oncogene Addiction, 5th Annual Beth Israel Deaconess Cancer Center Symposium, Boston, MA, 2012.

188. Felsher, D.W. IT2012: Therapeutic Manipulation of Inflammatory Microenvironment, Cuba, November 2012

189. Felsher D. W. Modeling and Predicting Oncogene Addiction, RECOMB Systems Biology Meeting, November 2012.

190. Felsher, D.W. Modeling and Predicting the Efficacy of Targeted Oncogene Inactivation, MD Anderson Cancer Medicine Grand Rounds, Houston, TX, January 2013

191. Felsher, D. W. Modeling and Predicting Oncogene Addiction, University of Freiberg, Germany, February 2013.

192. Felsher, D. W. Modeling Oncogene Addiction, University of Massachusetts, Worcester, MA, March 2013.

193. Felsher, D. W. Imaging the Immune System, AACR SNMI Molecular Imaging, San Diego, CA, February 27-March 2, 2013.

194. Felsher, D. W. Bone Marrow Mesenchymal Stem Cells as Possible Niche for Dormant Tuberculosis, ID Grand Rounds, Stanford University, March 14, 2013.

195. Felsher, D. W. Novel Biological Measurements to Detect, Predict and Prevent Human Disease, Johns Hopkins School of Public Health, Baltimore, MD, March 22, 2013.

196. Felsher, D. W. Modeling Oncogene Addiction, AFCR/Heme-Onc Seminar, University of Pennsylvania Cancer Center, Philadelphia, PA, March 26, 2013.

197. Felsher, D. W. Modeling and Predicting Oncogene Addiction. USC PSOC Seminar Series, Los Angeles, CA, April 26, 2013.

198. Felsher, D. W. Modeling Oncogene Addiction, Stanford Center for Cancer Systems Biology Annual Symposia, Stanford, CA, May 3, 2013.

199. Felsher, D. W. Modeling and Predicting Oncogene Addiction, Centre de Recherche en Cancerologie de Marseille, France, June 2013.

200. Felsher, D. W. Modeling and Predicting Oncogene Addiction, Royal Swedish Academy of Science, Stockholm, Sweden, September 1-3rd, 2013.

201. Felsher, D. W. Targeting MYC to Suppress Self-Renewal Programs in Cancer. Bone Marrow Failure Seminar, Stanford University, November 22, 2013.

202. Felsher, D.W. Modeling Oncogene Addiction. Cancercon2014, Chennai, India, January 30-February 2, 2014.

203. Felsher, D. W. Modeling Oncogene Addiction. Pediatric Oncology Research Conference, Stanford, CA, February 14, 2014.

204. Felsher, D. W. Modeling and Predicting Oncogene Addiction. Roswell Park Cancer Institute Distinguished Speaker, Buffalo, NY, March 12, 2014.

205. Felsher, D. W. Modeling Oncogene Addiction. 19th World Congress on Advances in Oncology and 17th International Symposium on Molecular Medicine, Metropolitan Hotel, Athens, Greece, October 9-11, 2014.

206. Felsher, D. W. Oncogene Addiction and the Immune System. CSHL Banbury Meeting, Cold Spring Harbor, NY, 2014.

207. Felsher, D. W. Modeling and Predicting Oncogene Addictions. Vanderbilt University Medical Center, Nashville, TN, January 22, 2015.

208. Felsher, D. W. Modeling and Predicting MYC Addiction. Roche Pharmaceuticals, Basel, Switzerland, February 13, 2015.

209. Felsher, D. W. Modeling Oncogene Addiction. UCSF Helen Diller Family Comprehensive Cancer Center Friday Seminar Series. UCSF, San Francisco, CA April 17, 2015.

210. Felsher, D. W. Childhood Liver Tumours Strategy Group, SIOPEL Meeting. Oslo, Norway, April 24-25, 2015.

211. Felsher, D. W. Modeling and Predicting Oncogene Addiction. Biozenrums Kolloquium Series, University of Wurzburg, Germany, May 20, 2015.

212. Felsher, D. W. Oncogene Addiction and Metabolism. AACR Special Conference: Metabolism and Cancer. Hyatt Regency Bellevue, Washington, June 7-10, 2015.

213. Felsher, D. W. Nanoscale Proteomics. Progenity, San Diego, CA. July 8, 2015.

214. Felsher, D. W. Modeling and Predicting Oncogene Addiction, University of Maryland Greenebaum Cancer Center, Baltimore, MD. November 18, 2015.

215. Felsher, D. W. Modeling and Predicting MYC Oncogene Addiction. MIT Koch Institute, Cambridge, MA. December 14, 2015.

216. Felsher D. W. Modeling and Predicting Oncogene Addiction, Harvard, Boston Children's Hospital, Boston, MA, December 15, 2015.

217. Felsher, D. W. The MYC Oncogene Regulator of Immune Checkpoints and Immune Surveillance. Weill Cornell Medical College Stem Cell Research and Regenerative Medicine, New York City, NY, April 11, 2016.

218. Felsher, D. W. Modeling and Predicting Oncogene Addiction, Hebron Institute, Barcelona, Spain, April 22, 2016.

219. Felsher, D. W. Predicting Metastasis, SIOPEL Meeting, Barcelona, Spain, April 22, 2016.

220. Felsher, D. W. Speaker: “Remodeling the Tumor Microenvironment through Oncogene Inactivation” AACR Annual Meeting, Chair of Symposia: Cancer Prevention through Modulation of the Tumor Microenvironment, New Orleans, LA, April 16-20, 2016.

221. Felsher, D. W. Oncogene Addiction, NIH CCR Eminent Lecture Series, Bethesda, MD, May 23, 2016.

222. Felsher, D. W. CSHL Course Seminar, Conditional Mouse Models, Cold Spring Harbor, NY, June 22, 2016.

223. Felsher, D. W. Oncogene Addiction and the Immune system, International Symposium in Molecular Medicine, Athens, Greece, October 6, 2016.

224. Felsher, D. W. Keynote Speaker, Oncology: Challenges and Opportunities, Sichuan Maternal and Child Health Hospital, Sichuan Sheng, China, November 11, 2016.

225. Felsher, D. W. Keynote Speaker, Oncology: Challenges and Opportunities, West China Medical School Sichuan University, Sichuan China, November 12, 2016.

226. Felsher, D. W. Keynote Speaker, Oncology: Challenges and Opportunities, Liuzhou Workers Hospital, Liuzhou China, November 15, 2016.

227. Felsher, D. W. The MYC Oncogene Globally Regulates the Immune Response, University of Miami Cancer Center, Miami, FL, February 9, 2017.

228. Felsher, D. W. Senescence & Aging Mini-Symposium, MYC Global Regulator Stemness versus Self-Renewal, Cancer Center & Cancer Research Institute Beth Israel Deaconess Medical Center, Boston, MA, March 7, 2017.

229. Felsher, D. W. Symposium on Tumor Motility, University of Freiberg, Germany, March 21-25, 2017.

230. Felsher, D. W. MYC Regulates the Immune Response, Major Symposium, AACR Annual Meeting, Washington DC, April 2, 2017.

231. Felsher, D. W. MYC Regulates the Immune Response, Keynote Speaker, University of Arizona Cancer Center Retreat, Tucson, AZ, April 21, 2017.

232. Felsher, D. W. Oncogene Addiction: A Paradigm for Translational Medicine, University of Maryland, College Park, MD, May 2, 2017.

233. Felsher, D. W. Oncology: Challenges and Opportunities, Speaker, Sichuan Cancer Hospital and Institute, China, May 9, 2017.

234. Felsher, D. W. Oncology: Challenges and Opportunities, Speaker, Beijing University of Chinese Medicine, China, May 10, 2017.

235. Felsher, D. W. Oncology: Challenges and Opportunities, Speaker, Chinese PLA General Hospital, China, May 10, 2017.

236. Felsher, D. W. Oncology: Challenges and Opportunities, Speaker, Taizhou Medical School, China, May 13, 2017.

237. Felsher, D. W. Characteristic Therapy Workshop for Traditional Chinese Medicine, Oncology: Challenges and Opportunities, Speaker/Chair, US Center for Chinese Medicine, Rockville MD, May 24, 2017.

238. Felsher, D. W. Liver Mini-Symposium, UCSF, San Francisco, CA, September 22, 2017.

239. Felsher, D. W. Roche Pharmaceuticals, San Francisco, CA, October 10, 2017.

240. Felsher, D. W. TRAM, Translational Research and Applied Medicine Program: Perspectives on Future of Translational Medicine, Stanford, CA, November 3, 2017.

241. Felsher, D.W. Societies of Biosciences of Argentina, Buenos Aires, Argentina, November 13th-19th, 2017.

242. Felsher, D. W. Modeling Metastasis in Hepatocellular Carcinoma, December 7-10th, Liver Meeting, 2017.

243. Felsher, D.W. Keynote Speaker, Cancercon, Chennai, India, Feb 1-2nd, 2018.

244. Felsher, D. W. Frontiers in Targeting MYC: Expression, Regulation, and Degradation. NIH campus, Bethesda, MD, April 9-10, 2018.

245. Felsher, D. W. The MYC Oncogene is a Global Regulator of the Immune Response, AACR Cancer Dormancy and Residual Disease, Montreal, QC, Canada, June 19-22, 2018.

246. Felsher, D. W. Invited Speaker, Conference Cancer and Environmental Mixtures. University of California Campus in Berkeley CA, August 21-22, 2018.

247. Felsher, D. W. Chinese Society of Clinical Oncology, Cancer Genomics Meets Immuno-Oncology: The Story of Myc. Xiamen China, September 2018.

248. Felsher, D. W. Modeling and Predicting Oncogene Addiction, MBICR Dedication, Chengdu China, October 8-15, 2018.

249. Felsher, D. W. Liver Cancer Symposium, Stanford University, Stanford, CA, October 17-18, 2018.

250. Felsher, D. W. Cancer Prevention and Therapy through Natural Products, Harvard Chinese Medicine Meeting, Harvard Medical School, Boston, MA, October 29-30, 2018.

251. Felsher, D. W. Keynote Speaker, GI Cancer Meeting, Guangzhou, November 7-12, 2018.

252. Felsher, D. W. MYC Master Regulator of the Immune System, Wurzburg, Germany, November 14, 2018.

253. Felsher, D. W. Invited Presentation, Milan, Italy, December 12-16, 2018.

254. Felsher, D. W. MYC is a Global Regulator of the Immune Response, Ludwig Cancer Center, Lausanne, Switzerland, January16, 2019.

255. Felsher, D. W. MYC is a Hallmark of Tumor Initiation and Maintenance, EPFL, Lausanne Switzerland, January 17, 2019.

256. Felsher, D. W. Invited Speaker, Conference Cancer and Environmental Mixtures. University of California Campus in Berkeley CA, February 6-7, 2019.

257. Felsher, D. W. Novel Therapeutics for Myc-Driven Cancer, SPARK, Stanford, CA, March 7, 2019

258. Felsher, D. W. The MYC Oncogene is a Global Regulator of the Immune Response to Cancer, Winship Cancer Institute of Emory University, Atlanta, Georgia, March 27, 2019.

259. Felsher, D. W. Trajectory of a Physician Scientist: The Usual and Unusual Suspects for Funding Opportunities, ReCAP Presentation, Stanford University, Stanford, CA, April 5, 2019.

260. Felsher, D. W. Targeting Specific Oncogenic Pathways to restore the Immune Response Against Cancers, World Vaccine Congress Washington 2019, Washington DC, April 14-17, 2019.

261. Felsher, D. W. Cancer Hallmarks: An Approach to Understanding the Biology of Tumorigenesis, Converging on Cancer Workshop, Washington D.C., April 29-30, 2019.

262. Felsher, D. W. The MYC Oncogene is a Global Regulator of the Immune Response, John Hart Lecture in Cancer Research, Northwestern University, Evanston, IL, May 23, 2019.

263. Felsher, D. W. MYC is a Global Regulator of the Immune Response, Amsterdam, European Hematology Association, June 13-16, 2019.

264. Felsher, D. W. MYC Regulates the Immune Response, Saint-Louis Hospital, Hematology Seminars, Paris, France, June 17, 2019.

265. Felsher, D. W. Invited speaker, FASEB, Lisbon, Portugal, July 21-26, 2019.

266. Felsher, D. W. Invited speaker, A Platform for Identifying Strategies for Reversing Cancer and Restoring the Immune Response, 2019 LakePharma Symposium on Next-Generation Therapeutics, San Francisco, CA, October 10, 2019.

267. Felsher, D. W. Invited speaker, Reversible Cancer by Targeting Oncogenes through Natural Products, BUCM Conference, Shenzhen China, December 12-17, 2019.

Felsher, D. W. Invited speaker, Universal Cancer Screening Summit, Mayo Clinic, Rochester, MN, February 3-4, 2020.

268. Felsher, D. W. Invited speaker, UCSD for Translational Medicine Day, San Diego, CA, March 11, 2020.

269. Felsher, D. W. Invited speaker, Stanford University TRAM Seminar MED121/221, Introduction to Translational Research and Applied Medicine: Pre-Clinical to Clinical Transition, Stanford, CA, September 30, 2020.

270. Felsher D. W. Targeting Cancer through the MYC Oncogene, Oppenheimer Biotech Emerging Science, virtual, Summit meeting, featuring Stanford University's SPARK Program, Friday, October 9, 2020.

271. Felsher, D. W. MYC and the Tumor Microenvironment. Prostate Cancer Foundation Annual Retreat, October 22, 2020

272. Felsher, D. W. Targeting MYC Oncogene Pathway: Global Gatekeeper of Tumor Growth and Immune Evasion. PBSS online Immuno-oncology Symposium. August 11-12, 2021.

273. Felsher, D. W. Oncogene Addiction, Frontiers in Clinical Translation Seminar Series, Stanford University, Stanford, CA, September 14, 2021.

274. Felsher, D. W. Introduction to TRAM: Translating Cancer Research, Translational Research and Applied Medicine (TRAM), Stanford University, Stanford, CA, September 29, 2021.

275. Felsher, D. W. Invited speaker, Translational Oncology: New Treatments for Cancer, Beijing China conference (zoom), December 11, 2021.

276. Felsher, D. W. Reversing Cancer: Targeting the MYC Oncogene. Eppley Institute for Research in Cancer and Allied Diseases, Eppley Seminar, University of Nebraska Medical Center, Omaha, Nebraska. April 28, 2022.

277. Felsher, D. W. American Society of Gene & Cell Therapy, AVV Vector Integrations in Human Hepatocytes in Liver-Targeted Gene Therapy, Annual Meeting (hybrid), Washington, DC, May 15, 2022.

278. Felsher, D. W. OHSU Pathology Grand Rounds, "Translational Oncology: Modeling, Predicting and Eliciting Oncogene Addiction", Portland Oregon, June 15, 2022.

279. Felsher, D. W. Stanford CVI 2022 Early Career Research Symposium: Session IV Translational Medicine. Stanford University, Stanford, CA, October 17, 2022.

280. Felsher, D. W. CIS2023 Cancer Immunotherapy Summit 2023, MYC Oncogene Global Regulator of the Immune Response, Hyatt Regency Boston MA, November 27-29, 2023.

281. Felsher, D. W. 18th International Conference on Genomics, Translational Research and Applied Medicine: Improving World health through Global Investment in Scientific Innovation, Singapore/Hangzhou, April 22-23, 2023.

282. Felsher, D. W. CIS2023 Cancer Immunotherapy Summit 2023, MYC Oncogene Global Regulator of the Immune Response, Hyatt Regency, Boston MA, November 27-29, 2023.

283. Felsher, D. W. Stanford INDE 217 Physician Scientist Hour (PhySH), "A Physicians Scientist Career in Reversing and Preventing Cancer: in the Laboratory and in the Courtroom", Stanford University, Stanford, CA, February 5, 2024.

284. Felsher, D. W. Invited speaker, "Oncogene Addiction: Exploiting a Vulnerability for the Treatment of Cancer". 19th International Conference on Genomics Thailand Part (ICG-19-THA), "Omics, Wellness and Longevity", Chulalongkorn Hospital, Bangkok, Thailand, May 18-19, 2024.

285. Felsher, D. W. CRC1479 Symposium 2024, International Symposium on Oncogene Driven Immune Escape, Freiburg, Germany, July 2024.

286. Felsher, D. W. Invited speaker, University of Chicago Cancer Center, "MYC Oncogene Pathway: the Achilles Heal of both Cancer Growth and Immune Evasion" Chicago, IL, July 25, 2024.

287. Felsher, D. W. Invited speaker, AbbVie Annual Internal Scientific Conference (Celebration of Science), South San Francisco, CA, September 23, 2024.

288. Felsher, D. W. Fred Hutchinson Cancer Center, Biology Seminar Series 2024-2025. Fred Hutchinson Cancer Center, Seattle, Washington, April 1, 2025.

Exhibit 2

From: Dean Felsher
To: Lori Merz; Dean Felsher
Subject: re: Camp Lejeune
Date: Thursday, August 17, 2023 6:22:50 PM

Dear Lori

Here is my CV.

My rates:

1000/hour review and preparation

1500/hour trial and depo, patient interview

Dean

Dean W. Felsher, MD PhD
Professor of Medicine-Oncology and Pathology
Associate Chief of Oncology
Director of Translational Research and Applied Medicine
Director of Admissions Medical Scientist Training Program
Co-Director Cancer Nanotechnology Training
Director Advanced Residency Training
Co-Director CTSA KL2 Training Program

Exhibit 3

Dean W. Felsher, MD Ph.D. Prior Testimony 2020-2025

Testimony Date	Court	Testimony	Case Name	Case Number
2022	Circuit Cout of Cook County, Illinois	Deposition & Trial	*Kamude, et al. v. Sterigenics US., LLC, et al.*	2018-L-010475
2022	Circuit Cout of Cook County, Illinois	Deposition & Trial	*Fornek v. Sterigenics US., LLC, et al.*	2018-L-010744
2022	Circuit Cout of Cook County, Illinois	Deposition	*Schumacher v. Sterigenics, US., LLC, et al.*	2018-L-018939
2021	Superior Court of California, Alameda County	Deposition & Trial	*Prudencio v. Johnson & Johnson*	RG20061303
2021	Superior Court of California, Alameda County	Deposition & Trial	*Van Klive v. Johnson & Johnson*	RG20062734
2022	Superior Court of California, Alameda County	Deposition	*Ta v. Kaiser Gypsum Co., Inc.*	RG21109884
2023	Superior Court of California, Alameda County	Deposition & Trial	*Valdez v. Johnson & Johnson*	22CV012759
2023	Superior Court of California, Santa Barbara County	Deposition & Trial	*Kevin Wright v. Union Oil*	21CV00925
2023	State Court of Gwinnett County, Georgia	Deposition	*Buczek v. Sterigenics, US., LLC, et al*	20-C-05918-S1
2024	State Court of Gwinnett County, Georgia	Deposition	*McLendon, et al. v. Becton, Dickinson and Company, et al.*	20-C-07123-S1
2024	State Court of Illinois, Cook County	Deposition	*Koch v. Medline Industries, et al.*	2023 L 000686
2024	U.S. District Court, N.D. Indiana, Fort Wayne Division	Deposition	*Asher v. RTX Corporation, et al.*	20CV000238

2024	Superior Court of Connecticut, Hartford Judicial District	Deposition	*Green et al. v. U.S. Steel Corp., et al.*	HHD-CV22-6158732
2024	District Court, Jefferson County, Colorado	Deposition	*Isaaks, et al. v Terumo BCT sterilization Services, INC., et al.*	22CV31124
2025	U.S. District Court, W.D. Missouri, Kansas City Division	Deposition	*Garavaglia v. GIB, et al*	4:25-cv-00014
2025	State Court of Illinois, Cook County	Deposition	*Cibelli Wagner v Sterigenics*	2023-L-005701